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Prevalence of autistic traits in functional neurological disorder and relationship to alexithymia and psychiatric comorbidity

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ABSTRACT

Introduction: In a cohort of adults with Functional Neurological Disorder (FND), we aim to:

1) Report the prevalence of autistic traits and alexithymia.
2) Report psychiatric comorbidity associated with autistic traits and alexithymia.
3) Explore whether alexithymia mediates the association between autistic traits and comorbidity.

Methods: 91 patients participating in a FND 5-week outpatient program completed baseline self-report questionnaires for total phobia, somatic symptom severity, attention deficit hyperactivity disorder (ADHD) and dyslexia. Patients were grouped by Autism Spectrum Quotient (AQ-10) score of <6 or ≥ 6 and compared for significant differences in tested variables. This analysis was repeated with patients grouped by alexithymia status. Simple effects were tested using pairwise comparisons. Multistep regression models tested direct relationships between autistic traits and psychiatric comorbidity scores, and mediation by alexithymia.

Results: 36 patients (40%) were AQ-10 positive (scoring ≥ 6 on AQ-10). A further 36 patients (across AQ-10 positive and AQ-10 negative groups) (40%) screened positive for alexithymia. AQ-10 positive patients scored significantly higher for alexithymia, depression, generalised anxiety, social phobia, ADHD, and dyslexia. Alexithymia positive patients scored significantly higher for generalised anxiety, depression, somatic symptoms severity, social phobia, and dyslexia. Alexithymia score was found to mediate the relationship between autistic trait and depression scores.

Conclusion: We demonstrate a high proportion of autistic and alexithymic traits, in adults with FND. A higher prevalence of autistic traits may highlight a need for specialised communication approaches in FND management. Mechanistic conclusions are limited. Future research could explore links with interoceptive data.

1. Introduction

Functional Neurological Disorder (FND) and Autism Spectrum Disorder (ASD) are two conditions commonly seen in neuropsychiatric settings with potential for high levels of disability. Symptoms manifest through the nervous system and, in the case of FND, do not relate to underlying structural neurological pathology. Despite common features, very little work has explored ASD is adults with FND.

1.1. Functional neurological disorder

In FND neurological symptoms demonstrate clinical features incompatible with structural pathology, there is abnormal function a system that is capable of normal function [1].

The current model of understanding focuses on strong ideas and expectations about a sensitising event (e.g., medical illness, physical trauma, psychophysiological events) alongside abnormal predictions of sensory data and body-focused attention [2]. Processing alterations reported in FND include limbic (amygdala) hyperactivation, excessive

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affective (autonomic) arousal and threat-related hypervigilance. There is also wider evidence in somatisation disorders of a higher prevalence of alexithymia and impaired interoception of bodily emotional responses, resulting in a reduced emotional awareness with limited integration of affective, cognitive and viscerosomatic experiences [3].

Psychiatric comorbidities are common in FND including depression, anxiety, panic disorder, personality disorders and obsessive compulsive personality disorders' as are functional somatic syndromes (such as irritable bowel, chronic fatigue and fibromyalgia) [1]. Common neurological comorbidities include epilepsy, migraine, and traumatic brain injury.

1.2. Autism spectrum condition / disorder

Autism Spectrum Disorder (ASD) is a neurodevelopmental condition characterized by persistent difficulties in social communication and interaction, restricted repetitive patterns in behaviour, interests and activities, and hyper or hyporeactivity to sensory stimuli. [3] The symptoms are present early on in development and affect daily functioning. However, they may not become fully manifest until social demands exceed limited capacities or may be masked by learnt strategies later in life.

The prevalence of ASD with mild or no intellectual disability in the UK is estimated at 1%. It has a male to female ratio of 2–3:1 in non-referred samples but this increases to 4:1 in clinical samples suggesting an ascertainment bias in the latter groups [4]. Common psychiatric co-morbidities include ADHD (attention-deficit/hyperactivity disorder), anxiety, depression, eating disorders, self-harm and obsessive compulsive traits [4]. The National Institute for Clinical Guidelines (NICE) recommended screening tool is the 10 item Autism Spectrum Quotient (AQ-10), with diagnostic assessment recommended for scores of 6 or above [5].

1.3. Alexithymia

Alexithymia is a dimensional personality trait characterized by difficulties in identifying and describing one's own emotional state [6]. As well as difficulty identifying feelings, it entails externally oriented thinking and a limited imaginal capacity. Rates in the general and autistic populations are estimated at 10 and 50% respectively [6,7]. Alexithymia is also prevalent in FND, with rates reported between 35%–75% [8]. Alexithymic individuals may show significantly higher levels of "functional" somatic and psychiatric symptoms such as anxiety and depression compared to those without alexithymia [9].

1.4. ASD and FND

The relationship between ASD and FND is relatively unexplored (see Box 1) [10–20], One study looking at odds ratios (OR) of childhood diagnoses in autistic adults, reported an OR of 5.9 for dissociative and conversion disorders compared to adults without autism. [17] Another population analysis reported an adjusted relative risk of 3.42 for somatoform disorder, and 6.45 for dissociative disorder. [11]

Certain features of autism, or associated traits, might act as perpetuating or precipitating factors. This includes differences in sensory processing (e.g., pain), cognition (attention, alexithymia) higher than average rates of psychiatric co-morbidity and risk of adverse life events, including bullying, abuse and exploitation. [21–23]

Sensory processing patterns such as low neurological threshold, or sensory over responsivity (extreme sensitivity to or avoidance of sensory stimuli (e.g., loud sounds)) have both been reported in FND and ASD [14,24,25]. Relevant to this are the known interoceptive differences associated with both ASD and FND. Interoception is the process by which the nervous system senses, interprets and integrates signals originating from within the body at conscious and unconscious levels [26].

Nisticò et al. explored this and suggested that difficulties translating interoceptive signals into higher-order brain representations might result in poor integration of physiological responses to emotional cues. Adding to this the role of alexithymia, the failure to interpret autonomic arousal as anxiety occurring during a physical precipitating event might result in the interpretation of these sensations as symptoms of physical illness [14,27]. In their paper on emotional processing in FND, Pick et al. highlight a transdiagnostic role of interoception, suggesting reduced integration between conscious emotional experience and somatic responses [28]. Relevant to this is the possible therapeutic role of interoceptive training explored in samples of autistic and somatoform patients [28–30].

These factors highlight a need to further explore whether an association exists; this paper focuses on the prevalence of autistic traits and alexithymia in participants of a 5-week outpatient based (day-case) individualised treatment programme for FND in the UK [31]. Autistic traits may act as underlying factors which could be considered in the treatment strategy.

We aim to:

1. Report the prevalence of autistic traits in an outpatient group of adults diagnosed with FND.
2. Report the prevalence of alexithymia
3. Report differences in symptom severity of psychiatric comorbidity between those scoring below 6 versus 6 and above on the AQ-10, and by alexithymia status

Box 1
Prior evidence from the literature on autistic traits and FND

Very little work exploring the prevalence of autistic traits in patients with FND has been done. The existing literature is heterogeneous in terms of country, terminology, sample demographics, aims, methodology (whether autistic traits were assessed in people with FND, or whether functional symptoms were assessed in autistic people) and findings. Research has focused largely on the child and adolescent populations and/or on psychogenic non-epileptic seizures (PNES rather than wider functional symptoms (See Supplementary Material Fig. 1 and Table 1 for search strategy and results)).

Two studies found no significant difference in autistic *traits* between adults with FND and controls [14], with another finding the same in samples of children [10]. Retrospective analyses assessing rates of comorbid diagnoses of ASD in patients with FND have reported higher rates compared to the general population [12,13,16,18,20]. One study assessing comorbidity in autistic individuals found higher rates of FND compared to controls [11]. Higher rates of somatoform dissociation and alexithymia in an autistic sample compared to controls has been reported by one study [19]. Finally another study looking at odds ratios (OR) of childhood diagnoses in autistic adults, reported an OR of 5.9 for dissociative and conversion disorders compared to adults without autism [17]. Full details of the systematic review can be found in the supplementary material.

4. Explore the association between autistic traits and psychiatric comorbidities as mediated by alexithymia scores

2. Methods

2.1. Ethical approval

The study was approved as a service evaluation by the departmental audit lead, registered with the quality and safety forum of UCLH NHS Foundation trust, thus not requiring ethics committee approval.

2.2. Sample/participants

This study was set in the Neuropsychiatry department of a tertiary neurological hospital. All patients had been diagnosed with FND by a neurologist and presented with functional movement, sensory and non-epileptic seizures and combinations of these. Further details of the referral pathway are found in Petrochilos et al. 2020 paper. [32] See Table 3 in supplementary material for DSM-V diagnostic criteria for FND. [33,34]

Data was collected between December 2019 and December 2021 when 105 patients participated in a 5-week individualised MDT outpatient (day-case) treatment programme for FND in the department of Neuropsychiatry at NHNN in the UK. Complete data sets were obtained from 91 (87%) patients for self-report measures of autistic traits, alexithymia, generalised anxiety, depression, somatic symptom severity, social phobia, panic phobia, work and social adjustment scale, dyslexia, and ADHD. Service referral letters for the 91 patients were reviewed retrospectively for the predominant functional symptom.

2.3. Self-report measures

Measures (described in Supplementary Material Table 2) were collected at the start of the program. These included the AQ-10, Toronto Alexithymia Scale (TASS-20), Patient Health Questionnaire (PHQ9), Generalised Anxiety Disorder-7 (GAD-7), Social Phobia Inventory, Work and Social Adjustment Scale (WSAS), Somatic Symptom Questionnaire (PHQ-15), Adult ADHD Self-Report Scale (ASRS v.1.1), The Adult Dyslexia Checklist, and the IAPT phobia scale. The cuttoffs for the TASS-20 were: ≤ 50 = no alexithymia, 51–60 = borderline alexithymia, and ≥ 61 = alexithymia.

2.4. Statistical analysis

Patients were grouped between those scoring <6 or ≥ 6 on the AQ-10 and compared for significant differences on the other measures. Data were tabulated and analysed descriptively using SPSS version 25. For each of the measures, a Mann-Whitney *U* test was performed to explore differences between the patient groups scoring <6 or ≥ 6 on the AQ-10.

Patients were then grouped by alexithymia status (no alexithymia,

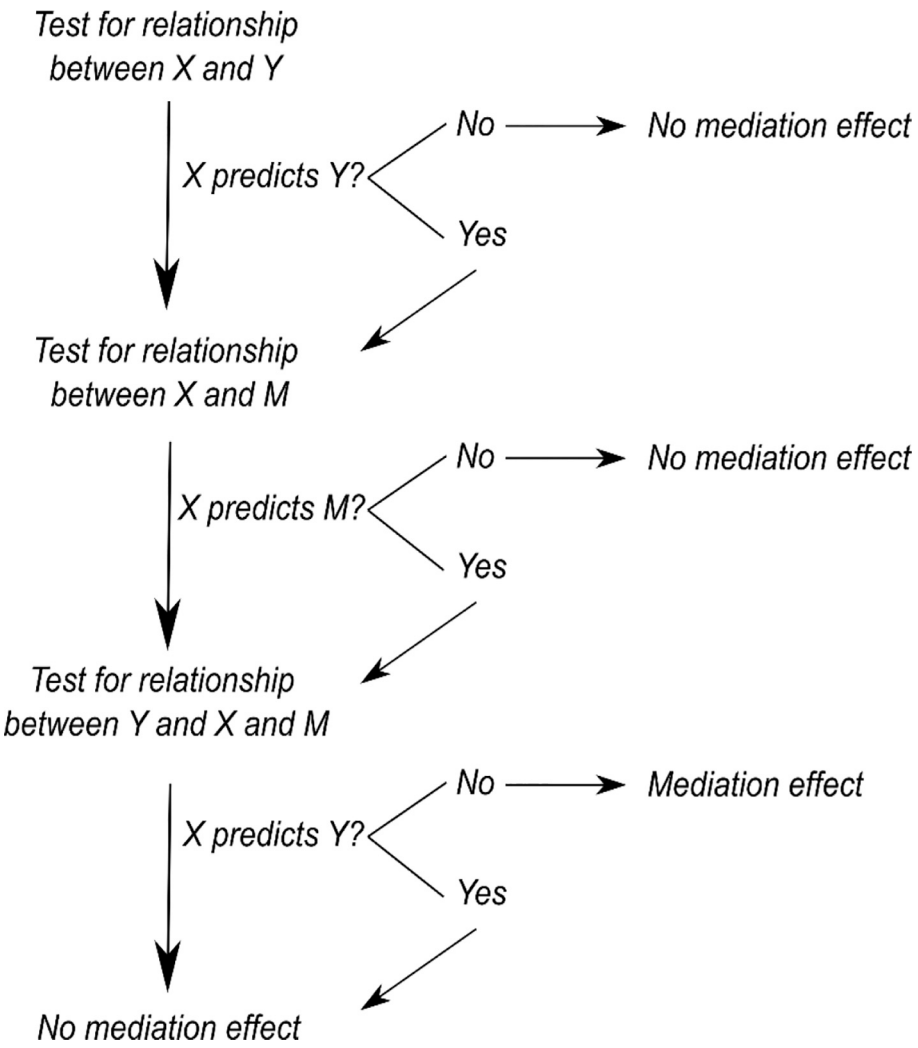

Fig. 1. Flow chart of mediation analysis model. The figure shows the multistep regression algorithm applied in the mediation analysis model, to test the direct and indirect relationships between the predictor (X), the outcome (Y) and the mediator (M) variables.

possible alexithymia, and alexithymia) and a Kruskal-Wallis H was performed to explore differences in self-report measured between alexithymia status groups, using standard clinical cut-offs. Simple effects were explored by performing pairwise comparisons.

We further sought to explore the association between autistic traits and psychiatric co-morbidities as mediated by alexithymia scores.

The proposed mediation models were tested in a single, bootstrapping-based model with 5000 iterations to assess the significance of the indirect effects between the independent (X: AQ-10) and the dependent (Y: psychiatric comorbidities) variable at the levels of the mediator variable (M: TAS-20). Age and sex were entered as covariates [35] (see Fig. 1). Table 4 provides the statistical results obtained from each of the models performed.

3. Results

Data was analysed for 91 patients, 69 (75.8%) were female and 22 (24.2%) were male. 39.6% scored AQ-10 positive and 60.4% scored AQ-10 negative. Of the AQ-10 positive group, 61.1% were females and 38.9% of males (Table 1). The probability of scoring AQ-10 positive was 31.9% for females and 63.63% for males (Table 2). Of the AQ-10 negative group (60.4% of the total), 85.5% were female and 14.5% were male. The probability of scoring AQ-10 negative was 68% for females and 36% for males. (See Table 1b.)

For the whole group, 37% were unemployed, 24% were full-time employed and 16.5% were part-time employed. Compared to the AQ-10 negative group, the AQ-10 positive group, rates for unemployment were higher (50%) and rates of full-time employment (19.4%) and part-time employment (11.1%) were lower.

Regarding highest education attainment level, the AQ-10 positive group had highest rates for GCSEs (38.9%), followed by HND/NVQ/BTEC (19.4%) and university degree (22%). The AQ negative group was highest for university degree (40%) A levels (16.4%) and then GCSEs (9.1%), HND (9.1%) and University master's degree (9.1%). Regarding predominant symptoms, the AQ-10 positive group compared to the AQ-10 negative group had similar rates of functional motor symptoms (47.2% and 50.9% respectively). However, they had slightly lower rates of non-epileptic episodes (16.7% versus 23.6%) and higher rates of other functional symptoms (e.g., PPPD, cognition and sensory) (36.1% versus 25.5%) compared to the AQ-10 negative group.

3.1. AQ-10 group differences

Differences in psychiatric comorbidity scores were explored between patients with a positive vs. negative AQ-10 status. A Mann-Whitney test was performed for each of the contrasts. Table 2 summarises the descriptive and statistical results.

3.2. TAS-20 group differences

Differences in psychiatric comorbidity scores were explored between patients with a (1) negative (2) possible or (3) positive TAS-20 status by performing Kruskal Wallis tests. . Table 3 summarises the statistical results.

3.2.1. AQ-10

The model returned a significant effect for differences in AQ-10 median ranks scores as a function of TAS status ($\chi^2 = 3.21$, $p = 0.01$). Simple effects analysis revealed that TAS-20 negative had a lower mean rank compared with TAS-20-positive patients (35.03 vs 53.81, $p < 0.01$).

3.2.2. PHQ9

The model returned a significant effect for differences in PHQ9 median ranks scores as a function of TAS status ($\chi^2 = 10.67$, $p = 0.005$). Simple effects analysis revealed that TAS-20 negative had a lower mean rank compared with TAS-20-positive patients (37.79 vs. 56.74, $p < 0.01$). Further, patients with possible alexithymia had lower PHQ9 scores compared with those with a positive status (39.18 vs 56.74, $p < 0.05$).

3.2.3. PHQ15

The model returned a significant effect for differences in PHQ15 median ranks scores as a function of TAS status ($\chi^2 = 8.13$, $p = 0.017$). Simple effects analysis revealed that TAS-20 negative had a lower mean rank compared with TAS-20-positive patients (41.02 vs. 55.13, $p < 0.05$).

Table 1
Demographic and Clinical Characteristics.

	Total ($N = 91$)	AQ-10 Positive ($n = 36$)	AQ-10 Negative ($n = 55$)
Age (SD)	43.42 (13.39)	41.5 (12.92)	46.4 (13.61)
Female (%)	69 (75.8)	22 (61.1)	47 (85.5)
Male (%)	22 (24.2)	14 (38.9)	8 (14.5)
In receipt of illness-related benefits (%)	47 (51.6)	20 (55.6)	27 (49.1)
Employment status (%)			
Full time employed	22 (24.2)	7 (19.4)	15 (27.3)
Part time employed	15 (16.5)	4 (11.1)	11 (20)
Unemployed	34 (37.3)	18 (50)	16 (29.1)
Full time student	1 (1.1)	1 (2.8)	0 (0)
Retired	14 (15.4)	4 (11.1)	10 (18.2)
Homemaker/carer	3 (3.3)	1 (2.8)	2 (3.6)
Unknown	2 (2.2)	1 (2.8)	1 (1.8)
Education (%) (highest level obtained)			
Primary (%)	0 (0)	0 (0)	0 (0)
Secondary lower	4 (4.4)	0 (0)	4 (7.3)
GCSE, O level, CSE	19 (20.9)	14 (38.9)	5 (9.1)
Further education	1 (1.1)	0 (0)	1 (1.8)
HND/NVQ/BTEC	12 (13.1)	7 (19.4)	5 (9.1)
Secondary higher (A levels)	10 (11)	1 (2.8)	9 (16.4)
University degree	30 (33)	8 (22.2)	22 (40)
University masters	7 (7.7)	2 (5.6)	5 (9.1)
University doctorate	2 (2.2)	1 (2.8)	1 (1.8)
Unknown	6 (6.6)	3 (5.5)	3 (5.5)
Predominant symptom (%)			
Functional motor	45 (49.4)	17 (47.2)	28 (50.9)
Non-epileptic episodes	19 (20.9)	6 (16.7)	13 (23.6)
Other (PPPD, cognition, sensory)	27 (29.7)	13 (36.1)	14 (25.5)
TAS: Alexithymia (%)			
Alexithymia positive	36 (40.0)	21 (58.3)	17 (30.9)
Probable alexithymia	15 (16.4)	6 (16.7)	9 (16.4)
No alexithymia	37 (40.7)	9 (25.0)	28 (50.9)
Unknown	2 (2.2)	1 (2.7)	1 (1.8)
GAD7: Anxiety (%)			
Severe anxiety (>15)	28 (30.8)	15 (41.7)	13 (23.6)
Moderate anxiety (10–14)	18 (19.7)	7 (19.4)	11 (20)
No anxiety (<10)	45 (49.5)	14 (38.9)	31 (56.4)
PHQ9: Depression (%)			
Severe depression (20–27)	18 (19.8)	10 (27.8)	8 (14.5)
Mod-severe depression (15–19)	23 (25.2)	13 (36.1)	10 (18.2)
Moderate depression (10–14)	16 (17.6)	6 (16.7)	10 (18.2)
No depression (<10)	34 (37.4)	7 (19.4)	27 (49.1)
SPIN: Social phobia (%)			
Very severe social phobia (>51)	7 (7.7)	3 (8.3)	4 (7.3)
Severe social phobia (41–50)	7 (7.7)	4 (11.1)	3 (5.5)
Moderate social phobia (31–40)	16 (17.6)	10 (27.8)	6 (10.9)
or no social phobia (⟨31)	61 (67)	19 (52.8)	42 (76.3)
Adult Dyslexia Checklist: Dyslexia (%)			
Moderate-severe dyslexia (>60)	7 (7.7)	4 (11.1)	3 (5.5)
Mild dyslexia (45–60)	35 (38.5)	18 (50)	17 (30.9)
No dyslexia (<45)	49 (53.8)	14 (38.9)	35 (63.6)
ASRS: ADHD (%)			
Warrant assessment for ADHD (>4)	16 (17.6)	8 (22.2)	8 (14.5)
No ADHD (<4)	75 (82.4)	28 (77.8)	47 (85.5)

Table 2
Group Comparison between AQ-10 Positive and AQ-10 Negative in Psychiatric Comorbidities[1].

	Descriptive				Statistics	
	AQ-10[2] Positive ($n = 36$)		AQ-10 Negative ($n = 55$)		Mann Whitney Test	
Measure	Median	Mean Rank	Median	Mean Rank	Standardised U Statistic[3]	p value
ASRS[a]	2	56.28	1	39.27	3.07	0.002
TAS-20[b] (Total)	63.5	58.69	51	37.69	3.71	**<0.001**
Describing feelings	17	55.58	14	39.73	2.80	0.005
Identifying feelings	24	54.44	19	40.47	2.47	0.013
Externally oriented thinking	21.5	56.68	18	39.01	3.12	0.002
PHQ9[c]	17	56.26	10	39.28	3.00	0.003
PHQ15[d]	13.5	51.33	12	42.51	1.56	n.s[4]
GAD7[e]	12	53.88	8	40.85	2.30	0.021
SPIN[f]	27	53.81	19	40.89	2.28	0.023
IAPT Phobia Scale[g] (Total)	11.5	53.11	6	41.35	2.08	0.037
Social Phobia	3	51.99	2	42.08	1.7	n.s
Panic Phobia	3.5	52.57	2	41.70	1.95	n.s
Object and Action Phobia	3	51.01	1	42.72	1.51	n.s
WSAS[h]	22	54.43	17	40.48	2.46	0.014
Adult Dyslexia Checklist	48	57.53	38	38.45	3.37	0.001

Compared to AQ-10 negative, AQ-10 positive patients had higher median ranks of ASRS (56.28 vs. 39.27, U = 3.07, $p = 0.002$), TAS-20 (58.69 vs. 37.69, U = 3.71, $p < 0.001$), PHQ-9 (56.26 vs. 39.28, U = 3, $p = 0.003$), GAD-7 (53.88 vs. 40.85, U = 2.30, $p = 0.003$), SPIN-7 (53.81 vs. 40.89, U = 2.28, $p = 0.023$), IAPT Phobia Scale (53.11 vs. 41.35, U = 2.08, $p = 0.037$), WSAS (54.43 vs. 40.48, U = 2.46, $p = 0.014$), and the Adult Dyslexia Checklist (57.53 vs. 38.45, U = 3.37, $p = 0.001$. No differences were obtained for PHQ15, and social, panic and object and action phobias IAPT subscales.

[1] As data were not normally distributed, the descriptive values are the median and mean ranks.
[2] Autism Spectrum Quotient. A total score of ≥6 is positive.
[3] for the test statistics value the mean ranks were used.
[4] non-significant.
[a] Adult Self-Report ADHD Scale (v1.1).
[b] Total Alexithymia Score.
[c] Patient Health Questionnaire-9.
[d] Patient Health Questionnaire-15.
[e] Generalised Anxiety Disorder Assessment.
[f] Social Phobia Inventory.
[g] Improving Access to Psychological Therapies Phobia Scale.
[h] Work and Social Adjustment Scale.

Table 1b
Probability of scoring positive or negative on the AQ-10 by gender.

	AQ Positive n = 36	AQ Negative n = 55
Females $n = 69$	31.8%	68%
Males $n = 22$	63%	36%

3.2.4. GAD7

The model returned a significant effect for differences in GAD7 median ranks scores as a function of TAS status ($\chi^2 = 12.25$, $p = 0.002$). Simple effects analysis revealed that TAS-20 negative had a lower mean rank compared with TAS-20-positive patients (34.61 vs. 56.69, $p < 0.01$).

3.2.5. SPIN

The model returned a significant effect for differences in SPIN median ranks scores as a function of TAS status ($\chi^2 = 17.91$, $p < 0.001$). Simple effects analysis revealed that TAS-20 negative had a lower mean rank compared with TAS-20-positive patients (33.03 vs. 59.46, $p < 0.001$). Further, patients with possible alexithymia had lower SPIN scores compared with those with a positive status (42 vs. 59.46, $p < 0.05$).

3.2.6. IAPT phobia scale

The model returned a significant effect for differences in phobia median ranks scores as a function of TAS status ($\chi^2 = 17.25$, $p < 0.001$). Simple effects analysis revealed that TAS-20 negative had a lower mean rank compared with TAS-20-positive patients (33.05 vs. 59.03, $p < 0.001$). Similar trends were obtained for the social, object and action and panic sub-scales (Table 3).

3.2.7. Adult dyslexia checklist

The model returned a significant effect for differences in dyslexia median ranks scores as a function of TAS status ($\chi^2 = 6.46$, $p = 0.039$). Simple effects analysis revealed that TAS-20 negative had a lower mean rank compared with TAS-20-positive patients (36.83 vs. 52.86, $p < 0.05$).

Table 4|Statistical results of the direct (c′) and indirect (m) regression models applied to determine if a mediation relationship exists between the predictor variable (X: AQ-10), the psychiatric comorbidities outcome variables (Y) and the mediator variable (M: TAS scores).

3.2.8. PHQ9

Overall model returned a significant, positive direct association between AQ-10 and PHQ9 scores (c′ = 0.7749, $t = 2.15$, $p = 0.0339$, 95% CI = [0.06, 1.48]) so that higher AQ-10 scores were associated with higher PHQ9 scores, accounting for 69.4% of the variability in PHQ9 scores. The mediation model was supported (m = 0.3425, 95% CI = [0.04, 0.79]) with a moderate effect size of 30.6% (**Fig. 1a**).

3.2.9. PHQ15

Overall model returned a significant, positive direct association between AQ-10 and PHQ15 scores (c′ = 0.7086, $t = 2.38$, $p = 0.0194$, 95% CI = [0.06, 1.48]) so that higher AQ-10 scores were associated with higher PHQ15 scores, accounting for 83.3% of the variability in PHQ9 scores. The mediation model was not supported (m = 0.1422, 95% CI = [0.04, 0.79]) (**Fig. 1b**).

3.2.10. GAD7

Overall model returned a non-significant direct association between AQ-10 and GAD7 scores (c′ = 0.6956, $t = 0.92$, $p = 0.361$, 95% CI =

Table 3
Group Comparison between Alexithymia Status Groups in Psychiatric Comorbidities[1] 2, b.

	Descriptive						Statistics		
	(I) No Alexithymia (n = 33)		(II) Possible Alexithymia (n = 22)		(III) Alexithymia (n = 36)		Independent Samples Kruskal Wallis Test		Post-hoc analysis
Measure	Median	Mean Rank	Median	Mean Rank	Median	Mean Rank	χ^2 Test Statistic[3]	p value	Pairwise Comparisons
ASRS[a]	1	39.35	2	47.25	2	50.29	3.21	0.2	Not applicable
AQ-10	4	35.03	5	47.98	6	53.81	9.25	0.01*	I = II; I < III**; II = III
PHQ9[c]	9	37.79	12	39.18	17	56.74	10.67	0.005**	I = II; I < III**; II < III*
PHQ15[d]	11	41.02	11.5	36.91	15	55.13	8.13	0.017*	I = II; I < III*; II = III
GAD7[e]	6	34.61	9.5	44.05	15	56.69	12.25	0.002**	I = II; I < III**; II = III
SPIN[f]	14	33.03	19	42.00	30	59.46	17.91	**<0.000*****	I = II; I < III***; II < III*
IAPT Phobia Scale[g] (Total)	4	33.05	6.5	42.66	12	59.03	17.25	**<0.000*****	I = II; I < III***; II = III
Social Phobia	2	35.17	2	43.30	6	56.63	12.04	0.002**	I = II; I < III**; II = III
Panic Phobia	1	31.29	2.5	44.43	4	59.57	20.59	**<0.000*****	I = II; I < III***; II = III
Object and Action Phobia	1	38.95	1	41.36	3	54.27	6.99	0.03*	I = II; I < III*; II = III
WSAS[h]	17	42.68	20	48.39	21	46.34	0.69	0.708	Not applicable
Adult Dyslexia Checklist	39	36.83	41.5	46.80	46	52.86	6.46	0.039*	I = II; I < III*; II = III

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$.
[1] As data were not normally distributed, the descriptive values are the mean ranks.
[2] Autism Spectrum Quotient. A total score of ≥6 is positive [5].
[3] For the test statistics value the mean ranks were used.
[a] Adult Self-Report ADHD Scale (v1.1).
[b] Total Alexithymia Score [6].
[c] Patient Health Questionnaire-9.
[d] Patient Health Questionnaire-15.
[e] Generalised Anxiety Disorder - 7.
[f] Social Phobia Inventory.
[g] Improving Access to Psychological Therapies Phobia Scale.
[h] Work and Social Adjustment Scale.

Table 4
Statistical results of direct and indirect effects of AQ-10 scores on psychiatric comorbidities mediated by TAS scores.

Outcome (Y)	Predictor (X) AQ-10 Total					Mediator (M) TAS Total		
	Direct Coefficient X ~ Y (c′)	t	p value	95% CI	Effect size (%)	Indirect Coefficient X ~ Y (m)	95% CI	Effect size (%)
PHQ9	0.7749	2.15	0.0339	[0.06, 1.48]	69.4	0.3425	[0.04, 0.79]	30.6
PHQ15	0.7086	2.38	0.0194	[0.04, 0.79]	83.3	0.1422	0.07, 0.44]	16.7
GAD7	0.6689	1.92	0.0579	0.02, 1.36]	68.1	0.3140	[0.01, 0.76]	31.9
SPIN	0.6956	0.91	0.3610	0.81, 2.20]	37.8	1.1479	[0.38, 2.34]	62.2
IAPT Phobia Scale	0.4910	1.36	0.1756	0.22, 1.20]	49.8	0.4950	[0.18, 0.96]	50.2
WSAS	0.8651	1.78	0.0785	[−0.1, 1.83	79.2	0.2276	[−0.09, 0.61]	20.8
Adult Dyslexia Checklist	1.5689	2.54	0.0127	[0.34, 2.79]	77.0	0.4696	[−0.001, 1.15]	23.0

[−0.02, 1.36]) so that higher AQ-10 scores were not associated with higher GAD7 scores. Given the absence of significant relationship between the predictor and the outcome variable, mediation effect is not applicable (**Fig. 1** and Fig. 2c).

3.2.11. SPIN

Overall model returned a non-significant direct association between AQ-10 and SPIN scores ($c' = 0.7086$, $t = 2.38$, $p = 0.0579$, 95% CI = [−0.81, 2.20]) so that higher AQ-10 scores were not associated with higher SPIN scores (**Fig. 1** and Fig. 2d).

3.2.12. Phobia

Overall model returned a non-significant direct association between AQ-10 and phobia total scores ($c' = 0.4910$, $t = 1.36$, $p = 0.1756$, 95% CI = [−0.22, 1.20]) so that higher AQ-10 scores were not associated with higher phobia scores (see **Fig. 1** and Fig. 2e).

3.2.13. WSAS

Overall model returned a non-significant direct association between AQ-10 and WSAS scores ($c' = 0.8651$, $t = 1.78$, $p = 0.0785$, 95% CI = [−0.1, 1.83]) so that higher AQ-10 scores were not associated with higher WSAS scores (**Fig. 1** and Fig. 2f).

3.2.14. Dyslexia

Overall model returned a significant, positive direct association between AQ-10 and dyslexia scores ($c' = 1.5689$, $t = 2.54$, $p = 0.0127$, 95% CI = [0.34, 2.79]) so that higher AQ-10 scores were associated with higher dyslexia scores, accounting for 77% of the variability in dyslexia scores. The mediation model was not supported (m = 0. 4696, 95% CI = [−0.001, 1.15]), suggesting that there is no ground for a mediation effect of level of alexithymia scores on the direct relationship between autistic traits and dyslexia levels (see **Fig. 1g**).

4. Discussion

This study aimed to assess the prevalence of autistic traits and alexithymia in a group of adults with FND, as well as explore associated psychopathology and the mediating role of alexithymia.

- We report new evidence of high rates of autistic traits, as measured by the AQ-10, in a group of adults with mixed FND symptoms. 40% of participants were AQ-10 positive, meeting the recommended

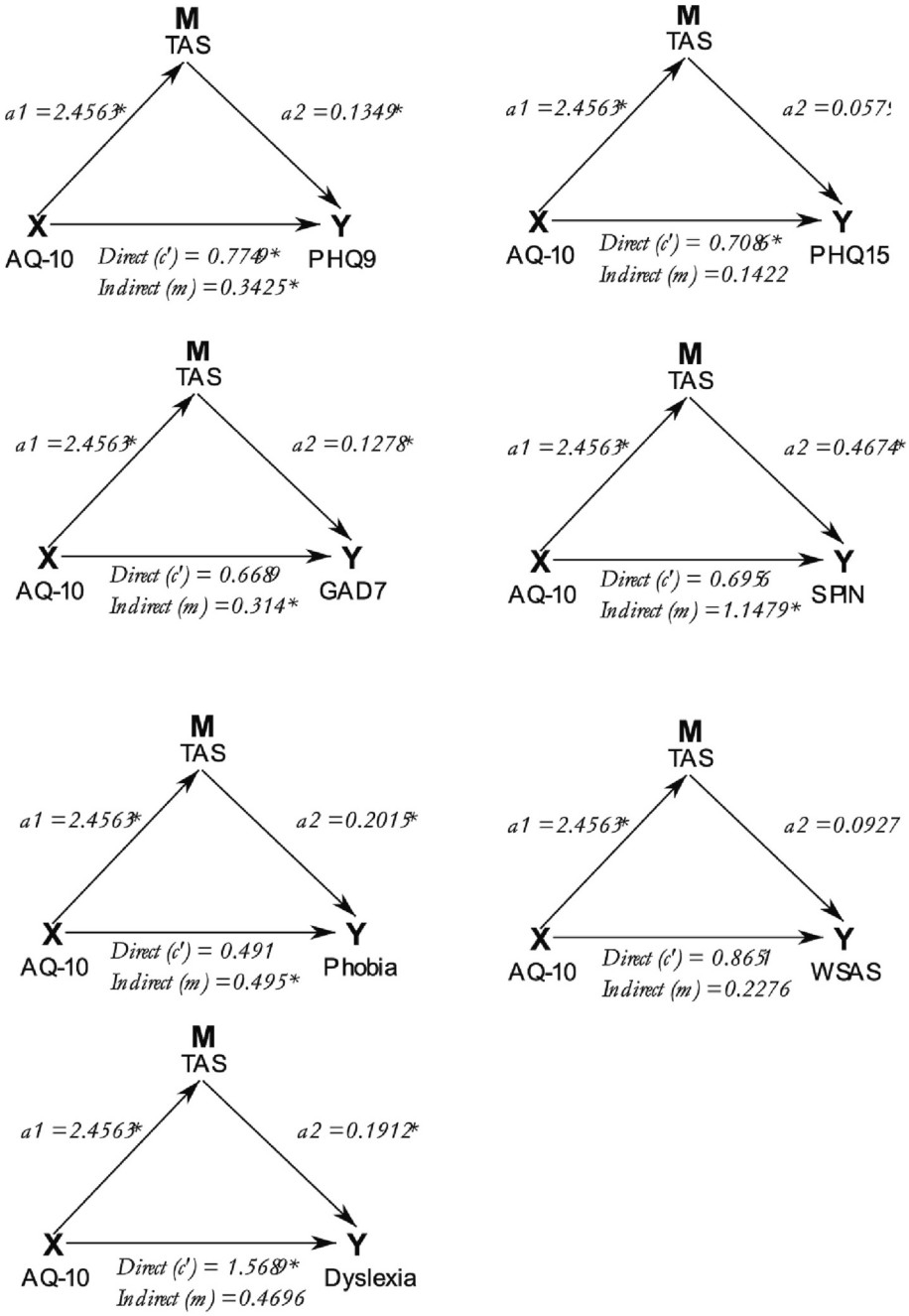

Fig. 2. Coefficients obtained from the multi-step regression analyses employed to establish the presence of a mediation effect (M) between the predictor variable (X: AQ-10), outcome variable (Y: psychiatric comorbidities) by TAS scores (M). pairwise effects are shown (*a1* [X ~ M], *a2* [M ~ Y], c′ [direct effect X ~ Y], and m [indirect effect X ~ Y]). Statistically significant effects are labelled with *. TAS score was found to mediate the relationship between AQ-10, PHQ-9, PHQ-15 and dyslexia scores.

threshold for consideration of a formal autism diagnostic assessment [34]. [36]

- The probability of scoring AQ-10 positive was 31.8% for females and 63.6% for males.
- We report further evidence of FND associated with high prevalence of alexithymia (40%) with a group mean TASS-20 score of 54.87.
- Alexithymia mediates the association between AQ-10 and PHQ9 (depression) scores with a moderate effect size of 30.6%.

4.1. Prevalence of ASD traits in FND

Our findings differ from Nisticò et al.'s finding of no patients in an FND sample being AQ-50 positive [14]. This may be due to a different study design; their use of the AQ-50 and a smaller sample of FND patients. They did however report that 86.7% of their sample with diagnosed ASD reported at least one functional neurological symptom, a prevalence significantly higher than the one encountered in their neurotypical sample (35.6%). They also found that tactile hypersensitivity was a risk factor for functional weakness and paraesthesia.

The AQ positive group (i.e. scoring 6 or above) had a higher proportion of males, higher rates of unemployment, alexithymia, severe generalised anxiety and severe depression, more moderate-severe dyslexia and a higher proportion meeting threshold for a recommended ADHD assessment (see Table 2). Statistical analysis revealed that this group also scored significantly higher on self-report measures of alexithymia, depression, generalised anxiety, social phobia, total phobia, day-to-day functional impairment, ADHD, and dyslexia. These findings are consistent with known comorbidities in the ASD population [14].

4.2. Prevalence of alexithymia and its mediation effect on AQ-10 positive scores and depression

We report further evidence of FND associated with a high prevalence of alexithymia (40%) with a group mean TASS-20 score of 54.87. This is higher than a reported prevalence of 34.5% (mean score of 55.38) in a previous NHNN outpatient group with functional motor symptoms (FMS) [27].

The differing prevalence of alexithymia between our AQ-10 positive group (55.6%) and AQ-10 negative group (30.9%), may reflect that alexithymia is known to be common in ASD and reportedly up to 49.93% [6]. However, our finding of a 30.9% prevalence in our AQ-10 negative group may reflect alexithymia's association with depression and FND [27]. By contrast, alexithymia prevalence in neurotypical individuals has been found to be much lower at 4.89% [37], and 10% in the general population [6].

The alexithymic group scored significantly higher on self-report measures of autistic traits, depression, somatic symptom severity, generalised anxiety, social phobia, total phobia and dyslexia. When assessing for the presence of a mediation effect of TAS-20 (alexithymia) score in the association between autistic traits and scores of psychiatric comorbidities, it was found to be true for the depression (PHQ9) score with a moderate effect size (30%). This supports the previously reported strong associations between alexithymia and depression [38].

The strong association between AQ-10 score and PHQ15 score (accounting for 83.3% of the variability) suggests that autistic traits are associated with high severity of somatic symptoms.

4.3. Use of the AQ-10

The AQ-10 was developed as a brief screen for ASD for use with adults with average or above average intellectual functioning and it is important to note that it is not a diagnostic tool. [36] However those with elevated autistic traits can experience similar difficulties to diagnosed autistic people, such as sensory hypersensitivity and difficulties with social communication and sensorimotor skills [22]. Previous research has also confirmed an increased prevalence of psychiatric diagnoses in both autistic people and those with elevated autistic traits [23,39,40].

The AQ-10 is advantageous in that it is self-administered, brief and forced choice with, with Alliston et al. reporting a sensitivity of 0.88, specificity of 0.91, and positive predictive value (PPV) of 0.85 (with a cut off-of 6), whilst Booth et al. reported a sensitivity of 79.87 and specificity of 87.31 [36,41]. More recently however, Ashwood et al. investigated the AQ questionnaire as a predictor of ASD caseness in a large sample of adults and reported a high sensitivity (0.77) but low specificity (0.29), with two-thirds of the patients who scored below the cut-off score of 6 being 'false negatives'.

The fact that we administered the AQ-10 on patients, i.e. not the general population, might increase the risk of false positive. Building on this, a further consideration is whether co-morbidities are inflating the AQ-10 scores. One group's co-morbidity data revealed that in their sample, GAD may 'mimic' ASD and inflate AQ scores, leading to false positives [42]. However, in our study higher GAD-7 scores were not directly associated with higher AQ-10 scores (Table 4, Fig. 2) but PHQ9, PHQ15 and dyslexia were.

There is a long history of sex bias in autism diagnosis, and it is important to consider this with screening measures. Females with ASD may, for example, fail to endorse some items because they refer to more typically male manifestations [43]. Murray et al. evaluated whether the AQ-10 exhibits such a bias, finding that although individual items showed some sex bias, these biases at times favoured males and at other times favoured females. Thus, at the level of test scores the item-level biases cancelled out to give an unbiased overall score. These findings were replicated in a later study [44].

4.4. Interpretation of findings

Whilst we cannot infer causality, nor conclude on the role of diagnosed autism in FND, there are several points to consider from the literature when interpreting the finding of a high prevalence of autistic traits in our FND sample.

Emotional and sensory processing are important factors to consider given their aetiological role in FND and clinical significance in ASD, and it is notable that Nisticò et al. reported tactile hypersensitivity as a risk factor for functional weakness [14]. FND patients are more likely to report physiological markers of panic and anxiety, without reporting an emotional state of anxiety; 'panic attack without panic' [27,45,46]. This is supported by evidence of greater physiological arousal, higher baseline cortisol and greater threat vigilance in FND, alongside higher levels of alexithymia [47,48].

Building from this, the mechanistic relevance of alexithymia to the development of functional symptoms might relate to the failure to correctly recognise autonomic arousal during a precipitating event (or chronically) as anxiety, but rather incorrectly interpreted as symptoms of physical illness [49,50]. A vicious cycle of mislabelling and symptom perception may ensue, exacerbated by a narrowed focus of attention, and reduced mental flexibility (also relevant to autistic traits).

Aberrant emotional processing alongside dysfunctional interoception are important mechanistic factors in FND. Both enhanced and impaired interoception have been reported in ASD, whilst impaired interoception has been strongly correlated with alexithymia [51–53]. When looking at the relationship between alexithymia and ASD, it is reported as common to, but *distinct* from ASD itself (and rather it is alexithymia that is more associated socioemotional difficulties common to ASC - the "alexithymia hypothesis") [54–56]. Building on this, when Shah et al. controlled for autistic traits and diagnosis, they reported that alexithymia, rather than autism, was associated with atypical interoception [57].

Jungilligens et al.'s 2022 perspective article relates the theory of constructed emotion to the FND predictive processing framework, [45] where incoming sensory information from the body and world is compared to features that have already been classified (i.e., a prediction/emotion concept) and can be used to give meaning to the current input (constructed emotion). Similar features from the past are pieced together to give meaning to the present by category construction. In FND they propose there is aberrant emotional construction, such that incoming sensory input might match a prediction that does not have emotion content, and a bodily/illness category is constructed (e.g. 'shaking'). This adds relevance to the role of alexithymia in FND, where non-alexithymics might appropriately use emotion concepts instead bodily and health/illness concepts in moments of arousal.

Jungilligens et al. also formulate that altered neurodevelopment (as well as adverse experiences) may affect the development of conceptual categories of emotion, as well as impact the ability to update prediction models reliant on emotion concepts, proposing: "Deficits in sensory processing, interoceptive accuracy, biased attention and impairments in motor learning among other constructs limit the use of precision signals and predictive errors to improve future predictions… we speculate that developmentally mediated disruptions in emotion construction play a role in the increased propensity for functional neurological symptoms in these populations." [45].

4.5. Limitations

This study did not seek to independently verify an autism diagnosis but rather to identify traits. This may limit the clinical generalisability of our conclusions. A further limitation is the lack of the control group, and that the AQ positive and negative groups were not matched for age and sex.

The generalisability of our findings with regards to the FND population may also be limited as by nature of a tertiary specialist centre. Our

sample may represent cases more likely to agree with the diagnosis and hence participate in treatment at a specialist centre, they may also be more motivated and engaged in their treatment plans compared to the general FND population. Also, although our referral criteria necessitated an FND diagnosed by a neurologist, we were unable to confirm the criteria used.

Another limitation is that we were unable to classify groups by FND subtype. Instead we noted the predominant symptom which was decided subjectively from retrospective review of referral letters. The combination of symptom modality experienced by most patients reflects the naturalistic nature of the study but also limits conclusions about motor/sensory/cognitive/PNES FND subtypes.

5. Conclusion

In conclusion, we have demonstrated new findings of a high prevalence of autistic traits in FND and have explored the role alexithymia plays in mediating ASD traits and depression. We hypothesise a possible role of interoceptive differences to be relevant in our findings. Future research could explore this role, as well as outcomes for AQ positive patients.

Declaration of Competing Interest

On behalf of all authors, the corresponding author states that there is no conflict of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jns.2023.120585.

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