Received: 11 December 2023 | Accepted: 11 April 2024

DOI: 10.1111/ene.16318

european journal of neurology
the official journal of the european academy of neurology

ORIGINAL ARTICLE

Perspectives on the diagnosis and management of functional cognitive disorder: An international Delphi study

Verónica Cabreira[1] | Jane Alty[2] | Sonja Antic[3] | Rui Araújo[4,5] | Selma Aybek[6] | Harriet A. Ball[7] | Gaston Baslet[8] | Rohan Bhome[9,10] | Jan Coebergh[11] | Bruno Dubois[12] | Mark Edwards[13] | Saša R. Filipović[14] | Kristian Steen Frederiksen[15,16] | Thomas Harbo[3] | Bradleigh Hayhow[17,18] | Robert Howard[19] | Jonathan Huntley[19,20] | Jeremy Isaacs[11] | William Curt LaFrance Jr.[21,22] | Andrew J. Larner[23] | Francesco Di Lorenzo[24] | James Main[25] | Elizabeth Mallam[26] | Camillo Marra[27] | João Massano[4,5] | Emer R. McGrath[28] | Laura McWhirter[1] | Isabel Portela Moreira[29] | Flavio Nobili[30] | Catherine Pennington[31,32,33] | Miguel Tábuas-Pereira[34,35,36] | David L. Perez[37] | Stoyan Popkirov[38] | Dane Rayment[39] | Martin Rossor[9] | Mirella Russo[40] | Isabel Santana[35,36] | Jonathan Schott[9] | Emmi P. Scott[41] | Ricardo Taipa[42] | Michele Tinazzi[43] | Svetlana Tomic[44] | Sofia Toniolo[45] | Caroline Winther Tørring[3] | Tim Wilkinson[1] | Lisbeth Frostholm[46,47] | Jon Stone[1] | Alan Carson[1]

Correspondence
Verónica Cabreira, Centre for Clinical Brain Sciences, University of Edinburgh, Edinburgh, UK.
Email: veronica.cabreira@ed.ac.uk

Funding information
European Commission, Grant/Award Number: 956673

Abstract

Background: Current proposed criteria for functional cognitive disorder (FCD) have not been externally validated. We sought to analyse the current perspectives of cognitive specialists in the diagnosis and management of FCD in comparison with neurodegenerative conditions.

Methods: International experts in cognitive disorders were invited to assess seven illustrative clinical vignettes containing history and bedside characteristics alone. Participants assigned a probable diagnosis and selected the appropriate investigation and treatment. Qualitative, quantitative and inter-rater agreement analyses were undertaken.

Results: Eighteen diagnostic terminologies were assigned by 45 cognitive experts from 12 countries with a median of 13 years of experience, across the seven scenarios. Accurate discrimination between FCD and neurodegeneration was observed, independently of background and years of experience: 100% of the neurodegenerative vignettes were correctly classified and 75%–88% of the FCD diagnoses were attributed to non-neurodegenerative causes. There was <50% agreement in the terminology used for FCD, in comparison with 87%–92% agreement for neurodegenerative syndromes. Blood

For affiliations refer to page 11.

tests and neuropsychological evaluation were the leading diagnostic modalities for FCD. Diagnostic communication, psychotherapy and psychiatry referral were the main suggested management strategies in FCD.

Conclusions: Our study demonstrates the feasibility of distinguishing between FCD and neurodegeneration based on relevant patient characteristics and history details. These characteristics need further validation and operationalisation. Heterogeneous labelling and framing pose clinical and research challenges reflecting a lack of agreement in the field. Careful consideration of FCD diagnosis is advised, particularly in the presence of comorbidities. This study informs future research on diagnostic tools and evidence-based interventions.

KEYWORDS
Alzheimer's disease, cognitive disorders, consensus, Delphi, functional cognitive disorder

INTRODUCTION

Functional cognitive disorder (FCD) is a common cause of non-neurodegenerative memory complaints seen in primary care, memory clinics and other specialised services [1–3]. In 2020, efforts to improve diagnostic uniformity culminated in the publication of operationalised diagnostic criteria [1]. According to the proposed criteria, FCD is characterised by positive features and 'internal inconsistency' (reflecting differences in automatic versus explicit processing within the same cognitive domain) [4–6], is persistent and distressing, and not explained by another condition (e.g., a neurodegenerative or psychiatric disorder). To date, the diagnostic criteria have not yet been externally validated, and inter-rater reliability for positive features of FCD (Box 1) has not been established [7–12].

The current FCD model is partially centred on attentional dysregulation that results in memory lapses including difficulty concentrating or completing tasks, retrieval blocks for overlearned information, word-finding difficulties, effortful thinking, or inability to follow conversations [2, 13]. For instance, patients might be able to perform a task well at certain times, but with significantly impaired ability at other times, particularly when the task is the focus of attention. Recent studies have elucidated other mechanisms including memory perfectionism and highly valued memory self-efficacy and intolerance to minor memory lapses, increased anxiety about the symptoms, increased vigilance over cognitive complaints, and impaired global metacognition (i.e., the way one's own thoughts are appraised) [5, 6, 14, 15], all feeding into a loop of symptom perpetuation [6, 12, 16].

Diagnosing FCD is challenging due to its similarity, at least at presentation, to neurodegenerative disorders, some mental health conditions, and multifactorial concentration difficulties (e.g., due to poor sleep, vascular disorders or medication or alcohol toxicity). Contemporary views acknowledge that FCD can coexist with these conditions if cognitive symptoms are in excess of what would be expected in these cases, and with an inconsistent pattern and hypervigilance for cognitive symptoms [5, 17]. Studies also indicate that FCD is likely to be underdiagnosed, perhaps in part because these patients usually concurrently meet descriptive criteria for either mild cognitive impairment (MCI) or subjective cognitive impairment (SCI),

BOX 1 Functional cognitive disorder (FCD) diagnostic criteria [1] and characteristic history and clinical signs informed by the literature [7–12] that inspired the FCD clinical vignettes (B, C and F). Some of these symptoms and signs require further validation

FCD diagnostic criteria

1. One or more symptoms of impaired cognitive function.
2. Clinical evidence of internal inconsistency (intact performance at certain times with demonstration of impaired ability at other times).
3. Symptoms or deficit that are not better explained by another medical or psychiatric disorder.
4. Symptoms or deficit that cause clinically significant distress or impairment in social, occupational or other important areas of functioning, or warrant medical evaluation.

Examples of history details and clinical signs observed in FCD patients

- Predominant attention and concentration problems.
- Losing track during tasks (ability to perform a task well at certain times, but with significant impairment at other times).
- Ability to detail several examples of memory failures clearly.
- Forgetting people's names and overlearned information (e.g., own telephone number), despite intact ability to recall it at a later point.
- Difficulties with immediate but not with delayed recall or other cognitive profiles inconsistent with cognitive performance, or incongruent with other neurological disorders.
- Struggling to complete easy parts of the cognitive tests, looking anxious and often making self-deprecating comments.
- Attending clinical appointments alone.

which are etiologically agnostic concepts with multiple possible underlying causes [18]. This challenge in differentiation may vary between countries and could be amplified by a limited access to clinical expertise and diagnostic investigations.

In this position paper, a panel of international experts who routinely assess patients with cognitive complaints aimed to analyse the panorama of FCD diagnosis and management. We explored the application of the proposed diagnostic criteria of FCD in light of the current clinically available tools with the purpose of gauging the current perspectives of cognitive specialists in the diagnosis, workup and management of FCD in comparison with neurodegenerative conditions. A survey using case vignettes was chosen to investigate the inter-rater agreement across different practice settings, backgrounds and clinical experience.

METHODOLOGY

We used an online modified Delphi methodology which is a group facilitation iterative technique that seeks to obtain consensus based on the opinions of 'experts' through a series of consecutive rounds [19]. This method is especially well suited to areas of inquiry for which there is little empirical evidence or clinical agreement. Participants assessed seven fictional not definitive clinical scenarios (informed by clinical experience and designed to represent commonly encountered clinical scenarios), developed by two of the authors (V.C. and A.C.), and piloted with two experts in cognitive disorders (Table 1). These authors assigned research study reference diagnoses to the different cases as follows: Alzheimer's disease (AD), three cases of FCD (terminology adopted), multifactorial cognitive impairment, primary progressive aphasia (PPA) and cognitive symptoms post-mild traumatic brain injury (TBI)/'post-concussion syndrome'. Scenarios varied in terms of age, spectrum of cognitive symptoms, comorbidities, and physical or bedside cognitive assessments.

Participant identification and recruitment

Experts with an interest and experience in cognitive disorders were selected to ensure that the information obtained reflected current scientific evidence and/or clinical expertise [20]. A method of purposive sampling was applied because participants' knowledge was required for the investigation being carried out [21]. Members of the European Academy of Neurology Scientific Panel on Dementia and Cognitive Disorders were also invited to participate to ensure representativeness. Using a 'snowball' recruitment method, participants could forward information to other eligible individuals.

Survey

Experts anonymously completed structured questionnaires through a series of two rounds and were fed back theirs and the group's responses after each round (Figure 1). In this way they were always informed of the current status of their collective opinion and could identify any missed items [20]. The surveys included an open-answer question regarding the presumed diagnosis, two multiple-choice questions regarding investigations and treatment approaches (multiple answers) and a yes/no question regarding follow-up for each of the seven scenarios (Table 1). Demographic data including participants' country, specialty, setting of practice, years of experience dealing with patients with cognitive complaints, and questions regarding experience with FCD were also collected.

The survey was formatted using the Online Survey system (https://www.onlinesurveys.ac.uk) and took approximately 15 min to complete. Reminder emails were employed to improve retention rates.

Analysis

Data were collected, stored, processed and analysed using Microsoft Excel and SPSS (IBM, version 28). Demographics were expressed as frequencies and percentages. In the first round, qualitative responses were manually analysed to adjust the language of the clinical scenarios including any missing information most often requested by the participants in a first clinical encounter. Responses obtained in the second round were considered for the analysis. The distribution of variables was examined using histograms and the Shapiro–Wilk normality test. Nominal variables were compared using chi-square or Fisher's exact test. Fleiss' kappa was run to determine inter-rater agreement for diagnostic investigations and treatment approaches (<0: no agreement; 0–0.20: slight; 0.21–0.40: fair; 0.41–0.60: moderate; 0.61–0.80: substantial; 0.81–1: almost perfect agreement) [22]. Spearman coefficient was calculated to assess for a correlation between variables and ANOVA test to analyse differences between groups. Significance was set as <0.05.

Ethics and privacy

No ethical approval was required as these were not real case scenarios and only amalgamated results were reported to the group rather than individual responses, thus guaranteeing anonymity. Unique identifiers were used to enable personalised emails containing a survey link to be sent to participants to aid survey administration and to allow monitoring of responses and issue of timely reminders to non-respondents. All participants gave their informed consent digitally before participation.

RESULTS

The study was conducted over 6 months (March 2022–August 2022). Forty-five valid responses were obtained in the first round, from 12 countries, with the highest number of participants from the UK

TABLE 1 Clinical scenarios and actions to be appraised.

Clinical scenarios presented to each Delphi participant in Round 2	Clinical actions to be appraised for each scenario
Case A: 78-year-old woman, academic retired. Complains of "bad memory" that has been progressing during the last 2 years. When questioned she states "I forget everything", without giving any further examples. The patient appreciates the presence of her husband in the clinic, as "he is someone who she can always check with". Family is very concerned as she already lost her way home a couple of times and is no longer able to leave the house alone, but she does not seem to recognise her impairment. No changes in sleep habits or appetite, and no behavioral disturbances were noted. Neurological examination is unremarkable except for copying hand movements. She scores 22/30 on MMSE (failing orientation to time and delayed recall).	Identify the presumptive diagnosis Choosing between a set of investigations to conduct in each patient (assuming all are available) • Blood tests (namely, thyroid, renal, and hepatic function, B12 and folate levels, electrolytes and glucose) • Propose to the patient an assessment of CSF biomarkers • Psychometric testing done by a neuropsychologist • Bedside cognitive testing (physician-led) • Cognitive nursing assessment • CT brain scan • MRI brain scan • EEG • PET (FDG-PET or PET-PiB/PET-tau) scan • Clinical history and neurological examination are sufficient for the diagnosis • Other Choosing a treatment strategy potentially relevant to each individual patient • Cognitive stimulating activities (brain games, puzzles, reading, etc.) • Psychological therapy • Psychiatric assessment • Add an antidepressant drug (e.g., SSRI) • Add an anti-dementia drug (e.g. cholinesterase inhibitor) • Occupational therapy and/or physiotherapy • Speech and language therapy • Other Deciding on the need of a follow-up appointment? (Yes/No)
Case B: 64-year-old woman, recently retired, independent, living on her own. Complains of "forgetfulness" (e.g., what she was going to pick up from the kitchen and people's names on TV), although she recognises the ability to recall it moments later. She describes a single episode when she went "blank" and couldn't remember her telephone number. She must check her things regularly to not leave them on the bus or at the supermarket. As she is so worried about her memory, she stopped attending the swim classes and rarely leaves the house now. She attends the clinic alone and there is no collateral source of information. Neurological examination is entirely normal. On bedside cognitive testing, she struggles to complete the required tasks, looking anxious and concerned about a potential failure, but ends up scoring appropriately for her norm.	
Case C: 44-year-old woman, university teacher, reports herself as an easily distractable person but now her memory is "worse than ever". She says it is now very hard to focus during important meetings although she keeps working. Her work colleagues apparently did not notice these difficulties. Attends the clinic alone. She also mentions that she used to run marathons but now gets tired after a few miles, feeling a constant fatigue and the need to sleep at least 10 h/night. No family history of dementia. On bedside cognitive testing, she takes more time to complete digit span and other attention tasks but performs correctly. Struggles with immediate recall but can remember all the words on delayed recall, with no other deficits. The neurological examination is unremarkable.	
Case D: 61-year-old man, overweight, history of diabetes and chronic back pain, recently retired after decreased performance and some disputes with his colleagues at work. Reports episodic memory lapses and word-finding difficulties, generally exacerbated by fatigue and insomnia. He is being prescribed tramadol and amitriptyline, as well as benzodiazepines at times. On examination, no neurological signs were evident. His wife reports reduced empathy and lower ability to manage complex tasks at home, like he is "unable to think properly" at times. No changes in appetite or food preferences. Blood tests are normal and a prior MRI brain scan, from 2 years ago, showed small vessel disease and mild generalised atrophy. On neuropsychological testing, he performs on the average range except for concrete interpretation of proverbs. No failure is identified on performance validity tests. The patient and his wife pre-emptively deny mood disturbances.	
Case E: 56-year-old woman, reports losing the thread of a conversation, forgetting a work colleague's name or mislaying keys in the fridge. Her thinking feels sluggish and effortful, "not like it used to be". She continues working with minor difficulties handling finances. She had a few episodes when she felt "spacy" or "confused" last year after her mother died from cancer. In the clinic, she rated her own memory as being significantly impaired. She says she very much enjoys reading thrillers despite sometimes she is not able to recall the ending of a novel she has read months before. There is no family history of dementia. Neurological examination entirely normal. On bedside cognitive testing, she performs on the normative range.	
Case F: 53-year-old woman reports a 1-year history of decreased ability to find words and reduced verbal output. Family reports word choice errors (e.g., reversal of yes and no, shorter sentences overall, missing words or words left out). No difficulties with memory, no mood or personality changes and no motor complaints. On examination, she is moderately aphasic, there is an inability to repeat long sentences and to perform complex actions. She had a MoCA test score of 17/30 with a prominent decline on language subtests. Semantic memory and prosody appear unimpaired. Also, she has no features of apraxia or dysarthria. The remaining neurological examination is normal.	
Case G: A 19-year-old male student, complains of headache, dizziness, and fatigue after having suffered a concussion while playing rugby months ago. That day he lost consciousness for a few seconds. Upon awakening, he remembers feeling dazed and confused, not being able to recall the brief moments immediately before the fall. He was taken to the hospital and discharged the same day after exclusion of neurological abnormalities. He no longer enjoys playing videogames and is not able to hang out with his friends without feeling exhausted. Even simple tasks now take a longer time. During consultation, he expresses himself very worried that these might be signs of brain damage.	

Abbreviations: CSF, cerebrospinal fluid; CT, computed tomography; EEG, electroencephalogram; FDG, fluorodeoxyglucose; MMSE, Mini-Mental State Examination; MoCA, Montreal Cognitive Assessment; MRI, magnetic resonance imaging; PET, positron emission spectroscopy; PiB, Pittsburgh compound B; SSRI, selective serotonin reuptake inhibitor.

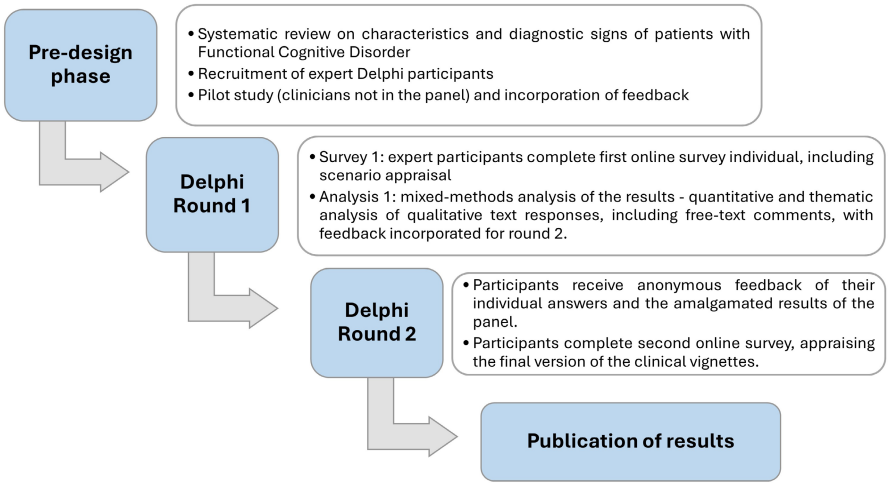

FIGURE 1 Flowchart of this modified Delphi study and activities conducted across the two rounds.

TABLE 2 Demographics of Delphi participants.

Characteristic	*N* (%)
Age (years), median (range)	44 (29–71)
Female sex	14 (32)
Years of experience assessing patients with cognitive disorders, median (range)	13 (2–45)
Country	
Australia	2 (4)
Croatia	1 (2)
Denmark	5 (11)
France	1 (2)
Germany	1 (2)
Ireland	1 (2)
Italy	5 (11)
Portugal	6 (13)
Serbia	1 (2)
Switzerland	1 (2)
UK	17 (38)
USA	4 (9)
Specialty	
Neurology[a,b]	36 (80)
Psychiatry[a]	10 (22)
Neuropsychiatry	3 (7)
Old age psychiatry	5 (11)
Neuropsychology[b]	2 (4)

Note: Unless stated otherwise values are given as numbers (percentages).
[a]Two participants are specialists in both neurology and psychiatry.
[b]One participant is a specialist in neurology and neuropsychology.

($n=17$; 38%). Thirty-six (80%) were neurologists. The median age was 44 years, and 32% were female. The median (range) of years of experience in cognitive disorders was 13 (2–45) years (Table 2). Forty-three (96%) of the participants reported recognising the term 'functional cognitive disorder'. Thirty-one (69%) reported seeing between 10 and 100 patients with FCD over the last year For 31 (69%) participants FCD was responsible for 5% to 30% of patients with memory complaints assessed in their clinics, with the largest age group being patients aged between 45 and 65 years (53%) (Table 3). Most of the participants were based in cognitive/neurology clinics in tertiary/referral hospitals. None worked in primary care. Thirty-nine (87% retention rate) responses were obtained in the second round.

Terminology

A total of 18 diagnostic categories (terminologies were merged whenever possible) were recorded by participants across the

TABLE 3 Experience in care of patients with functional cognitive disorder and other cognitive disorders among the 45 clinicians who responded to the survey.

Clinical experience domain	*N* (%)
Recognising the term FCD	43 (96)
Patients with a functional cognitive disorder diagnosed in the last year (*n*)	
<10	12 (27)
10–50	21 (47)
>50	10 (22)
>100	2 (4)
Proportion of patients with FCD among the patients with memory complaints seen in the clinic (%)	
<5	3 (7)
5–30	31 (69)
30–50	3 (7)
>50	7 (16)
NA	1 (2)
Commonest age group of FCD patients (years)	
<30	–
30–45	16 (36)
45–65	24 (53)
>65	1 (2)
NA	4 (9)
Using a self-help or remote treatment (e.g., app, chatbot, book, website) for patients with FCD	8 (18)
Willingness to try remote interventions in FCD	42 (93)

Abbreviations: FCD, functional cognitive disorder; NA, not applicable.

seven scenarios (Figure 2 and Table S1). When compared with the research study reference diagnosis, 41%–49% of the respondents labelled the three FCD cases as FCD, followed by mood disorder (anxiety/low mood) (13%–31%), subjective cognitive impairment/MCI (13%–26%) and stress/normal (3%–5%) (Figure 2). Similar heterogeneity was observed for the multifactorial cognitive impairment case and for post-TBI cognitive symptoms (Figure 2). This contrasts with the labelling of neurodegenerative cases (Cases A and F): 92% of respondents agreed with the diagnosis of PPA and 87% with the diagnosis of AD. Most importantly, despite the heterogeneity in terminology, 75%–88% of the FCD diagnoses were attributed to non-neurodegenerative causes and 100% of the neurodegenerative vignettes were classified as such, highlighting a clear-cut distinction between the two spectra of patient groups solely on clinical grounds (Table 4 and Figure 2). The clinical specialty (neurologists vs. non-neurologists), number of years of experience dealing with cognitive disorders ($p = 0.40$) and the number of patients diagnosed with FCD by the experts in 1 year ($F = 0.81$, $p = 0.50$) did not affect the chance of attributing a diagnosis matching the research study reference diagnosis for any of the fictional scenarios.

Investigation

History and examination were deemed as sufficient to make the diagnosis of post-TBI cognitive symptoms by 21% of the participants, 8%–10% in the FCD cases, only 5% for AD or PPA, and by none of the participants for the multifactorial cognitive impairment. Blood tests were widely supported in all the diagnostic scenarios, especially in those with a higher likelihood of neurodegeneration (54%–95%).

Regarding diagnostic biomarkers, structural brain imaging was the most popular (62%–95% would require a magnetic resonance imaging (MRI) scan, most frequently in the AD and PPA cases). While 62% of the participants would request a cerebrospinal fluid (CSF) study for AD biomarkers for PPA, only a third of the participants required CSF biomarkers for AD, and a minority (up to 13%) suggested these for FCD (Table 5; Table S1). Regarding functional neuroimaging, fluorodeoxyglucose positron emission tomography (FDG-PET) would only be requested for neurodegenerative cases (15% and 41% for AD and PPA, respectively) and multifactorial cognitive impairment (28%), and almost never for FCD/post-TBI symptoms (<5%). Electroencephalography (EEG) and computed tomography (CT) scan were only rarely selected, possibly due to their more historical relevance, low diagnostic yield and perhaps sequential role in the investigation timeline. Only a minority of participants (n=1–3) would request amyloid/tau-PET or apolipoprotein E (APOE) genotyping for diagnostic purposes, probably a reflection of their limited availability outside of research protocols as will be discussed later. Interestingly, 18%–28% of the clinicians would not request any imaging method for the FCD scenarios, while all would require at least one of these tests to make a diagnosis of neurodegenerative condition (Table 5).

Additional bedside cognitive testing was mentioned by around a third of the participants for FCD cases, and slightly higher for AD (54%). Participants suggested further formal neuropsychological evaluation for 46%–69% of the FCD cases (up to half of those not performing bedside testing would request in-depth cognitive testing), and this was similar to AD (49%) (almost all not performing bedside testing for AD do so because they privilege a neuropsychological evaluation). Less than a third would use cognitive testing to diagnose post-TBI cognitive symptoms, while 80% would use it to confirm PPA. Full psychiatric assessment aiming at an in-depth exploration of psychosocial factors was requested more often in situations where FCD, mood disorder or behavioural disturbances were considered in the differential diagnoses (around a third vs. 3%–10% for neurodegeneration).

Besides the standard investigations, a qualitative analysis of participants' responses revealed an emphasis on the collateral history, screening of mood disorders and vascular risk factors, sleep studies (for obstructive sleep apnea) and cardiovascular assessment (especially in the presence of fatigue).

On the whole, the median number of investigations suggested was minimal for post-TBI symptoms (median: 2, range: 6) (with the caveat that this vignette reported neurological abnormalities having been excluded after the injury) and maximal for PPA (median: 5, range: 6) (Table 5). The median number of investigations requested

FIGURE 2 Diagnostic labels for each clinical scenario. Research study reference diagnoses are named under each bar. AD, Alzheimer's disease; CFS, chronic fatigue syndrome; FCD, functional cognitive disorder; FTD, frontotemporal dementia; MCI, mild cognitive impairment; OSA, obstructive sleep apnea; PCS, 'post-concussion syndrome'; PPA, primary progressive aphasia; PTSD, post-traumatic stress disorder; SCI, subjective cognitive impairment; TBI, traumatic brain injury.

for FCD was 3–4, with blood tests, MRI scan and neuropsychological evaluation leading the preferences. Neurologists were more likely to ask for a CSF study ($p < 0.001$), but not other tests, in comparison with non-neurologists.

Treatment and follow-up

Overall, 80%–95% of participants highlighted the importance of diagnostic and prognostic communication regardless of the underlying disorder. Although it was not the focus of this study, the participants emphasised the importance of including brief discussions about the nature of the symptoms, rates of 'normal forgetting', and explanation on memory functioning and contributing and maintaining factors (e.g., poor sleep, fear avoidance, medication management) during diagnostic formulations. Some mentioned support and encouragement for behavioural activation and gradual return to normal activities with pacing strategies if fatigue present, both in FCD and post-mld TBI cases. Regarding non-pharmacological interventions, psychotherapy or psychiatry were selected by up to 72% of participants for FCD and post-TBI cognitive symptoms. Particularly, cognitive-behavioral therapy or acceptance and commitment therapy might be useful in selected cases. A complementary role of exercise, vascular risk factor control and medication adjustment was deemed as particularly relevant for the multifactorial cognitive impairment (72%, 90% and 95%, respectively). Around a third would also recommend cognitively stimulating activities (e.g., reading, puzzles, board games) for all cases. Speech and language therapy was suggested by 92% for PPA. The role of physiotherapy/occupational therapy was particularly emphasised for post-TBI cognitive symptoms.

Regarding pharmacological interventions, only one participant (3%) would prescribe anticholinesterase inhibitors in FCD (Case E), while 90% would recommend it for AD, 36% for PPA and 5% for multifactorial cognitive impairment. Patients with FCD and post-TBI symptoms had a chance between 23% and 46% of being prescribed an antidepressant, even without fulfilling formal diagnostic criteria for a depressive disorder.

Twenty-seven participants (69%) would schedule a follow-up for FCD, and 25 (64%) for post-TBI-cognitive symptoms, with the clinicians highlighting that in the remaining cases the patients are instructed to be referred for a new assessment should the situation change, new symptoms appear, or in the advent of symptom progression. In contrast, 90%–100% of the participants would follow-up patients with PPA, AD and multifactorial cognitive impairment in their clinic.

TABLE 4 Proportion of the responses matching the operationalised definition for each clinical scenario.

Reference diagnosis	Agreement between experts' diagnoses and research study reference diagnoses (%)	Non-neurodegenerative diagnoses (%)	SCI/MCI (%)[a]	Fisher's exact test (neurologists vs. non-neurologists)
AD	87	0	3	0.976
FCD	41	75	26	0.820
FCD	49	88	13	0.695
Multifactorial cognitive impairment	46	6	3	0.702
FCD	44	78	18	1.00
PPA	92	0	0	0.508
'Post-concussion syndrome'	41	100	0	0.694

Abbreviations: AD, Alzheimer's disease; FCD, functional cognitive disorder; MCI, mild cognitive impairment; PPA, primary progressive aphasia; SCI, subjective cognitive impairment.

[a]Mild cognitive impairment and subjective cognitive impairment were considered independently (neither neurodegenerative or non-neurodegenerative) as they represent clinical syndromes which are in theory etiologically agnostic.

TABLE 5 Investigations requested by the experts for each of the case scenarios.

Investigation (%)	AD (Case A)	FCD (Case B)	FCD (Case C)	Multifactorial cognitive impairment (Case D)	FCD (Case E)	PPA (Case F)	Post-concussion (Case G)
Blood tests	95	87	87	82	85	90	54
MRI brain scan	82	72	62	85	69	95	69
Bedside cognitive testing	54	39	36	26	28	36	31
Neuropsychological evaluation	49	54	46	64	69	80	23
CSF studies	33	8	0	13	13	62	0
Psychiatric assessment	3	31	28	18	33	10	21
CT scan	18	8	10	3	13	0	5
EEG	3	3	3	6	8	3	10
PET scan (PiB/tau)	8	3	0	6	5	18	0
FDG-PET	15	3	0	28	5	41	3
No brain imaging requested	0	21	28	10	18	0	26
APOE genotype	8	–	–	–	5	8	–
Cognitive nursing assessment	10	3	3	5	0	5	3
History/examination are sufficient	5	10	8	0	10	5	21
Other relevant tests	–	–	Cardiology assessment (23)	Sleep studies (64)	Sleep studies (8) Vascular risk factor screening (31)	–	–
Investigations requested (median, range)	3 (5)	3 (7)	3 (6)	4 (6)	4 (9)	5 (6)	2 (6)

Note: Unless stated otherwise values are given as numbers.

Abbreviations: AD, Alzheimer's disease; APOE, apolipoprotein A; CSF, cerebrospinal fluid; CT, computed tomography; EEG, electroencephalogram; FCD, functional cognitive disorder; FDG, fluorodeoxyglucose; MRI, magnetic resonance imaging; PET, positron emission spectroscopy; PiB, Pittsburgh compound B; PPA, primary progressive aphasia.

Overview

We analysed the inter-rater agreement regarding investigations, treatment and follow-up approaches (multiple responses) across participants. We found moderate global inter-rater agreement for PPA ($k=0.50$), AD ($k=0.46$) and multifactorial cognitive impairment ($k=0.45$). Fair inter-rater agreement was recorded for FCD (κ ranging between 0.31 and 0.35) and post-TBI symptoms ($k=0.32$). The overall agreement across all case scenarios and options posed was fair–moderate ($\kappa=0.41$; 95% CI 0.40–0.41) in this expert population.

DISCUSSION

This online modified Delphi study analysed how a group of 45 clinicians with expertise in cognitive disorders, from 12 countries, assesses case vignettes illustrative of different cognitive scenarios, with an emphasis on the identification and management of patients with FCD. This study is unique as it was conducted 2 years after the publication proposing the FCD diagnostic criteria, and we surveyed clinicians with expertise in the cognitive and functional disorders subspecialties. So, this study represents the first step towards establishing inter-rater reliability of the current diagnostic criteria for FCD based on relevant positive clinical features [1]. This comes at a time when models of brain health clinics are being discussed to replace traditional cognitive clinics focused on a neurodegenerative paradigm and to incorporate the new complementary tools and biomarkers in the diagnosis of neurocognitive disorders [23, 24].

The clinicians demonstrated high discriminative ability in distinguishing between neurodegenerative and non-neurodegenerative/functional cognitive symptoms using patient characteristics, history and bedside examination only, with comparable accuracy between neurologists and non-neurologists, regardless of years of experience or number of patients diagnosed with FCD per year. They assigned a neurodegenerative diagnosis to 100% of neurodegenerative cases and a non-neurodegenerative diagnosis to 75%–88% of the three FCD (reference diagnosis) vignettes (excluding MCI and SCI as they are in theory etiologically agnostic). A much lower agreement was observed for how clinicians label and frame non-neurodegenerative cognitive presentations, with 41%–49% of the participants labelling the case vignettes with a reference diagnosis of FCD as such, versus 87%–92% agreement in terminology for the two neurodegenerative vignettes. The FCD case vignettes were labelled with diverse terms including mood/anxiety, SCI or MCI, or stress, in line with findings from previous surveys conducted across European countries and the USA [25–27] (Table S2). The heterogeneity in labelling reflects a different conceptualisation of the non-neurodegenerative cognitive symptoms, confusion about the most appropriate diagnostic labels, and overlapping clinical presentations [16, 28]. In addition, multiple contributing factors can account for someone's symptoms, and an assumption was brought forward of a higher chance of comorbidities in the FCD group versus neurodegenerative conditions. Currently it is unclear whether the FCD label should be reserved for patients with purely distressing cognitive symptoms with demonstrable internal inconsistency in cognitive performance, or if it should be split in different subtypes [5, 16], considering its common occurrence with other functional neurological/functional somatic disorders, comorbid mood and anxiety symptoms often with excessive concern about cognitive performance and intolerance to memory lapses [5, 17, 29], obstructive sleep apnea or medication cognitive side effects. Similar discussions have been held regarding conceptualisation of functional motor phenotypes [30, 31], and a FCD classification had been previously proposed by Stone et al. [5, 28]. Interestingly, hesitancy in labelling and consideration of comorbidities because of fears of misdiagnosis were not among the prevailing concerns when considering the neurodegenerative vignettes.

Equally, it is currently admitted that mood and behavioural changes can potentially represent a prodrome to neurodegeneration, and the same might apply to a subset of individuals with FCD [13, 28, 29, 32], as observed for Parkinson's disease and multiple sclerosis [33, 34] with FND overlap, often preceding these conditions.

The seven clinical vignettes analysed in this study represent common clinical scenarios, relevant to neurologists, psychiatrists, psychologists, geriatricians and other healthcare professionals assessing patients with cognitive presentations. These were selected based on the need of individual diagnostic formulations and availability of different treatments, which makes their clinical distinction paramount. We also included post-TBI cognitive symptoms which are prevalent [35], and are thought to be reinforced via similar mechanisms to those recognised in FCD including memory perfectionism, phobic anxiety, behaviour avoidance, hypervigilance, and concern about an underlying brain damage [36–38]. The participants adopted similar approaches to investigation and management of 'post-concussion symptoms' and FCD, and terminologies overlapped, with 26% of the participants labelling post-TBI cognitive symptoms as FCD. The emphasis on occupational therapy and physiotherapy in this scenario perhaps supports the adoption of interventions commonly used post-TBI and targeting maladaptive beliefs, dysfunctional coping strategies and disease comorbidities [35–37] to FCD cases. In support of this formulation, individuals with functional seizures and cognitive symptoms post-TBI displayed objective cognitive improvement after receiving neurobehavioral therapy [39], a multimodality, integrative psychotherapy used in patients with other functional neurological disorder subtypes [3, 40].

Of note, although only a third of participants said they would perform bedside cognitive testing, this is confounded by the fact that the vignettes already contained some information on cognitive testing, so the chances of performing a cognitive screening test or some mental tasks are probably substantially higher than reported. Some participants nevertheless highlighted that bedside cognitive testing is not always needed if the history strongly suggests FCD, reflecting heterogeneity in clinical practice. They also acknowledge the limitations of the tests, first because people with FCD may sometimes underperform in cognitive tests, especially in the attention and executive control domains [2, 16, 29, 41], and second because highly educated patients will often not be reassured by normal cognitive scores, which can occur in both scenarios of FCD and neurodegeneration. Participants agreed that the reasoning for cognitive testing needs to be carefully explained to the patient. Further evaluation should focus on the role of bedside cognitive testing and neuropsychological evaluation in FCD, specifically in the pursuit of more positive signs (e.g., incongruence between a significant drop in a domain where there is a demonstration of function on a daily basis, as in fluency tests with preserved conversational ability) [12].

In line with this, while on one hand, CSF and neuroimaging biomarkers hold promise for a distinction between preclinical AD and non-neurodegenerative symptoms [42], as auxiliary tests, many participants pointed out their limited specificity and sensitivity, high costs, invasiveness and the limited availability of PET

scans/radiotracers in certain countries [43]. A greater demand for these methods is anticipated with the possible approval of new amyloid-modifying agents in Europe/UK [44, 45]. Caution and shared decision-making are advised for FCD, especially given that significant AD neuropathology does not equal cognitive decline, and coexisting FCD with unrelated incidental positive amyloid/tau biomarkers (especially with aging) is possible [43, 46, 47]. Further studies and solid recommendations are welcomed to reduce the chances of incidental findings and dementia misdiagnosis, which implies significant patient risks such as inclusion in clinical trials for dementia and being exposed to hazardous therapies. The present development of diagnostic tools and digital biomarkers might be of assistance in the future [48–50].

Notably, around two-thirds of the experts would follow up patients with functional cognitive symptoms, in contrast to over 90% for neurodegeneration. In this simulated scenario, follow-up was presented as a binary yes/no option. However, it is important to note that in many countries the actual practice often involves an open follow-up system, wherein further assessments are offered in response to new symptoms or clinical progression. Therefore, the presented results should be interpreted cautiously. Yet, we suspect that this discrepancy may be attributed partially to the perception of FCD as a more benign condition compared with neurodegenerative disorders, partially in the absence of currently available effective treatments, and to the need to avoid raising false expectations about prognosis. Equally, in some cases follow-up is used as a safety net for clinicians and patients to delay the diagnosis, but an earlier diagnosis of FCD potentially offers therapeutic advantages, accepting that in rare cases a neurodegenerative pathology could be missed [28]. In the rare event of a neurodegenerative disorder unfolding, timely administration of disease-modifying therapy might be considered [44].

Finally, at first glance, a fair to moderate inter-rater agreement regarding investigation and treatment approaches in FCD cases may seem low. However, this reflects an agreement on multiple aspects of diagnosis and treatment practices by a broad panel of participants, for which a strict guidance is currently lacking.

The use of vignettes and survey methodology incurs certain limitations. These include the absence of patient–clinician interaction, being a poor proxy for clinical contact where the amount of clinical information gathered is superior. The lack of longitudinal follow-up hinders diagnostic clarification in some cases, and the inclusion of preliminary cognitive or imaging data, or clinical findings on examination (e.g., “aphasia” in Case F) in the vignettes may affect the diagnostic formulation and choice of further investigation tests, deviating from real-life stepwise procedures. Analysing data based on discrete versus narrative diagnoses underestimates the agreement on FCD diagnosis by not acknowledging the existence of overlap between functional cognitive symptoms and a second aetiology. The diagnoses assigned to vignettes are not definitive as multidisciplinary assessment might be needed to clarify the diagnosis, the importance of which we demonstrated in this study. A potential response bias may exist, with clinicians more motivated to participate, those with prior knowledge of FCD, and domain of English language being overrepresented, limiting generalisability. Despite efforts to recruit experts from a geographically dispersed area and a diverse professional background, all of which may increase the content validity of these results, there is overrepresentation of male participants, high-income countries and predominantly neurologists. The possibility of discussions among participants, although unlikely since the participants remained anonymous during the whole iterative process, cannot be completely excluded, particularly in the minority recruited through the ‘snowball’ method (<5%). Choice of tests could also be affected by costs in countries not covered by public health systems or medical insurance.

Position statement

Our study reinforces the importance of having multidisciplinary teams involved in the care of patients with cognitive complaints, especially in the field of FCD. Given that FCD is a likely common cause for cognitive symptoms, continuous work is required to improve screening and diagnostic workflow [18]. In an era when efforts to improve diagnostic specificity of neurodegenerative conditions are increasing [23, 24], this should also apply to the non-neurodegenerative field. Namely, further positive diagnostic signs and diagnostic decision tools to support clinicians in differentiating FCD from other causes of cognitive impairment are needed to enable earlier diagnosis and increase specificity [48, 51]. Variability in the framing of early stages of SCI/MCI and FCD, with competing conceptual models, underscores the need for better guidance and efforts to produce standardised diagnostic approaches. Importantly, identifiable clinical characteristics that enabled diagnostic formulation of a reasonably consistent diagnosis among these experts merit further consideration and clearer classification, and should be further validated and operationalised, including in settings with less expertise in cognitive neurology [10, 12]. Neuropsychology can be very informative both in diagnosis and treatment of FCD [12, 52], particularly in cases where history and bedside testing are incongruent; however, there is a lack of consensus and regional variability regarding how and when this should be used. Given the complexity of cognitive syndromes, an FCD diagnosis needs careful consideration, particularly in the presence of comorbidities such as sleep disorders, fatigue, mood disorders and anxiety. It should not be a ‘dustbin diagnosis’ for everything that is not neurodegeneration. However, clinical experience and published reports of improvement in patients with FCD who underwent various treatments [53–55] support the need for better awareness about this condition and the establishment of an accurate diagnosis and subsequent treatment planning. Further studies are needed to understand the trajectory of FCD and its relationship with other conditions such as depression and anxiety, FND and neurodegeneration. Moreover, contemporary views admit that FCD and neurodegeneration are not mutually exclusive, both because FCD can occur in the prodromal stage in a subset of patients, and AD pathology can coexist with FCD as an incidental finding as

we age. So, further work is required to improve the early separation of these diagnostic entities and identify those individuals who might be at a hypothetical higher risk of future neurodegeneration. The growing knowledge on FCD allows for effective communication of the diagnosis, mechanistically informed discussions about prognosis, and incorporation of education elements and other strategies such as behavioural activation in treatment plans [12, 18]. This study also emphasises the striking need for evidence-based interventions for FCD that might improve prognosis and influence decisions about follow-up, so we encourage future research which can inform such interventions [56]. Non-neurodegenerative cognitive syndromes have been traditionally overlooked and under-researched but require further attention, especially given the growing numbers of people attending memory clinics with non-neurodegenerative causes for memory symptoms [24, 28, 57].

AUTHOR CONTRIBUTIONS

Verónica Cabreira: Conceptualization; investigation; writing – original draft; methodology; software; formal analysis; data curation. **Jane Alty:** Writing – review and editing; investigation. **Sonja Antic:** Investigation; writing – review and editing. **Rui Araújo:** Investigation; writing – review and editing. **Selma Aybek:** Investigation; writing – review and editing. **Harriet A. Ball:** Investigation; writing – review and editing. **Gaston Baslet:** Investigation; writing – review and editing. **Rohan Bhome:** Investigation; writing – review and editing. **Jan Coebergh:** Investigation; writing – review and editing. **Bruno Dubois:** Investigation; writing – review and editing. **Mark Edwards:** Investigation; writing – review and editing. **Saša R. Filipović:** Investigation; writing – review and editing. **Kristian Steen Frederiksen:** Investigation; writing – review and editing. **Thomas Harbo:** Investigation; writing – review and editing. **Bradleigh Hayhow:** Investigation; writing – review and editing. **Robert Howard:** Investigation; writing – review and editing. **Jonathan Huntley:** Investigation; writing – review and editing. **Jeremy Isaacs:** Investigation; writing – review and editing. **William Curt LaFrance Jr.:** Investigation; writing – review and editing. **Andrew J. Larner:** Investigation; writing – review and editing. **Francesco Di Lorenzo:** Investigation; writing – review and editing. **James Main:** Investigation; writing – review and editing. **Elizabeth Mallam:** Investigation; writing – review and editing. **Camillo Marra:** Investigation; writing – review and editing. **João Massano:** Investigation; writing – review and editing. **Emer R. McGrath:** Investigation; writing – review and editing. **Laura McWhirter:** Investigation; writing – review and editing. **Isabel Portela Moreira:** Investigation; writing – review and editing. **Flavio Nobili:** Investigation; writing – review and editing. **Catherine Pennington:** Investigation; writing – review and editing. **Miguel Tábuas-Pereira:** Investigation; writing – review and editing. **David L. Perez:** Investigation; writing – review and editing. **Stoyan Popkirov:** Investigation; writing – review and editing. **Dane Rayment:** Investigation; writing – review and editing. **Martin Rossor:** Investigation; writing – review and editing. **Mirella Russo:** Investigation; writing – review and editing. **Isabel Santana:** Investigation; writing – review and editing. **Jonathan Schott:** Investigation; writing – review and editing. **Emmi P. Scott:** Investigation; writing – review and editing. **Ricardo Taipa:** Investigation; writing – review and editing. **Michele Tinazzi:** Investigation; writing – review and editing. **Svetlana Tomic:** Investigation; writing – review and editing. **Sofia Toniolo:** Investigation; writing – review and editing. **Caroline Winther Tørring:** Investigation; writing – review and editing. **Tim Wilkinson:** Investigation; writing – review and editing. **Lisbeth Frostholm:** Funding acquisition; writing – review and editing; supervision. **Jon Stone:** Funding acquisition; writing – review and editing; supervision. **Alan Carson:** Conceptualization; funding acquisition; methodology; writing – review and editing; supervision.

AFFILIATIONS

[1]Centre for Clinical Brain Sciences, University of Edinburgh, Edinburgh, UK
[2]Wicking Dementia Research and Education Centre, University of Tasmania, Hobart, Tasmania, Australia
[3]Department of Neurology, Aarhus University Hospital, Aarhus, Denmark
[4]Department of Neurology, Centro Hospitalar Universitário São João, Porto, Portugal
[5]Department of Clinical Neurosciences and Mental Health, Faculty of Medicine University of Porto, Porto, Portugal
[6]Neurology, Faculty of Sciences and Medicine, Fribourg University, Fribourg, Switzerland
[7]Population Health Sciences, Bristol Medical School, Bristol, UK
[8]Department of Psychiatry, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts, USA
[9]Dementia Research Centre, UCL Queen Square Institute of Neurology, London, UK
[10]Centre for Medical Image Computing, University College London, London, UK
[11]Department of Neurology, St George's University of London, London, UK
[12]Department of Neurology, Institut de la mémoire et de la maladie d'Alzheimer (IM2A), AP-HP, Brain Institute, Sorbonne University, Paris, France
[13]Department of Basic and Clinical Neurosciences, Institute of Psychiatry Psychology and Neurosciences, Kings College London, London, UK
[14]University of Belgrade Institute for Medical Research, Belgrade, Serbia
[15]Clinical Trial Unit, Rigshospitalet, University of Copenhagen, Copenhagen, Denmark
[16]Department of Clinical Medicine, University of Copenhagen, Copenhagen, Denmark
[17]Department of Neurology, Fiona Stanley Hospital, Murdoch, Western Australia, Australia
[18]School of Medicine, University of Notre Dame Australia, Fremantle, Western Australia, Australia
[19]Division of Psychiatry, University College London, London, UK
[20]Camden and Islington NHS Foundation Trust, London, UK
[21]Alpert Medical School, Brown University, Providence, Rhode Island, USA
[22]Neuropsychiatry and Behavioral Neurology, Rhode Island Hospital, Providence, Rhode Island, USA
[23]Cognitive Function Clinic, Walton Centre for Neurology and Neurosurgery, Liverpool, UK
[24]Department of Clinical and Behavioural Neurology, Santa Lucia Foundation IRCCS, Rome, Italy
[25]Bristol Dementia Wellbeing Service, Devon Partnership NHS Trust, Bristol, UK
[26]Neurology Department, North Bristol NHS Trust, Bristol, UK
[27]Department of Neuroscience, Catholic University of the Sacred Heart, Memory Clinic - Fondazione Policlinico Agostino Gemelli IRCCS, Rome, Italy
[28]School of Medicine, University of Galway, Galway, Ireland
[29]Neurology Department, Private Hospital of Gaia of the Trofa Saúde Group, Vila Nova de Gaia, Portugal

[30]IRCCS Ospedale Policlinico San Martino, Genoa, Italy
[31]Clinical Lecturer, University of Edinburgh, Edinburgh, UK
[32]Neurology Department, NHS Forth Valley, Larbert, UK
[33]Department of Clinical Neurosciences, NHS Lothian, Edinburgh, UK
[34]Neurology Department, Centro Hospitalar e Universitário de Coimbra, Praceta Prof. Mota Pinto, Coimbra, Portugal
[35]Faculty of Medicine, University of Coimbra, Coimbra, Portugal
[36]Center for Innovative Biomedicine and Biotechnology, University of Coimbra, Coimbra, Portugal
[37]Department of Neurology and Psychiatry, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA
[38]Department of Neurology, University Hospital Essen, Essen, Germany
[39]Rosa Burden Centre for Neuropsychiatry, Southmead Hospital, Bristol, UK
[40]Department of Neuroscience, Imaging and Clinical Sciences G. d'Annunzio University of Chieti-Pescara, Chieti, Italy
[41]Medical University of South Carolina, Charleston, South Carolina, USA
[42]Neuropathology Department, Centro Hospitalar Universitário de Santo António, Porto, Portugal
[43]Department of Neurosciences, Biomedicine and Movement, University of Verona, Verona, Italy
[44]Department of Neurology, University Hospital Center Osijek, Medical School on University of Osijek, Osijek, Croatia
[45]Cognitive Disorder Clinic, Nuffield Department of Clinical Neurosciences, University of Oxford, Oxford, UK
[46]Department of Clinical Medicine, Aarhus University Hospital, Aarhus, Denmark
[47]Department of Functional Disorders and Psychosomatics, Aarhus University Hospital, Aarhus, Denmark

ACKNOWLEDGEMENTS

The authors thank Allan Thimsen for his contribution to the scientific content of this study as a Delphi participant.

FUNDING INFORMATION

V.C. has received funding from the European Union's Horizon 2020 research and innovation programme under the Marie Skłodowska-Curie Grant Agreement No. 956673. This article reflects only the author's view, the agency is not responsible for any use that may be made of the information it contains.

CONFLICT OF INTEREST STATEMENT

The authors have stated explicitly that there are no conflicts of interest in connection with this article. J.S. reports personal fees from UptoDate, outside the submitted work, runs a self-help website for patients with functional neurological symptoms (www. neurosymptoms.org) which is free and has no advertising, provides independent medical testimony in personal injury and negligence cases regarding patients with functional disorders, and is secretary of the International Functional Neurological Disorder Society. He is a Chief Scientists Office NHS Research Scotland Career Researcher. A.J.C. is a director of a limited personal services company that provides independent medical testimony in court cases on a range of neuropsychiatric topics on a 50% pursuer 50% defender basis, a paid associate editor of the *Journal of Neurology Neurosurgery and Psychiatry*, and unpaid president elect of the International Functional Neurological Disorder Society. D.L.P. has received honoraria for continuing medical education lectures in functional neurological disorder, royalties from Springer Nature for a functional movement disorder textbook, is on the editorial boards of *Brain and Behavior* (paid), *Epilepsy & Behavior*, and *Journal of Neuropsychiatry and Clinical Neurosciences*, and has received funding from the National Institutes for Health (NIH) and the Sidney R. Baer, Jr. Foundation unrelated to this work. M.J.E. does medical expert reporting in personal injury and clinical negligence cases, including in cases of functional neurological disorder (FND). M.J.E. has shares in Brain & Mind, which provides neuropsychiatric and neurological rehabilitation in the independent medical sector, including in people with FND. M.J.E. has received financial support for lectures from the International Parkinson and Movement Disorder Society and the FND Society (FNDS). M.J.E. receives royalties from Oxford University Press for his book *The Oxford Specialist Handbook of Parkinson's Disease and Other Movement Disorders*. M.J.E has received honoraria for medical advice to Teva Pharmaceuticals. M.J.E. receives grant funding, including for studies related to FND, from the National Institute for Health and Care Research (NIHR) and the Medical Research Council (MRC). M.J.E. is an associate editor of the *European Journal of Neurology*. M.J.E. is a member of the international executive committee of the International Parkinson and Movement Disorder Society and a board member of the FNDS. M.J.E. is on the medical advisory boards of the charities FND Hope UK and Dystonia UK.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ORCID

Verónica Cabreira https://orcid.org/0000-0001-9945-7681
Jane Alty https://orcid.org/0000-0002-5456-8676
Rui Araújo https://orcid.org/0000-0002-3610-3437
Selma Aybek https://orcid.org/0000-0002-7877-6760
Harriet A. Ball https://orcid.org/0000-0002-2137-7582
Gaston Baslet https://orcid.org/0000-0002-3039-582X
Rohan Bhome https://orcid.org/0000-0002-8317-7930
Saša R. Filipović https://orcid.org/0000-0001-8508-3367
Kristian Steen Frederiksen https://orcid.org/0000-0001-5124-4417
William Curt LaFrance Jr. https://orcid.org/0000-0002-4901-3852
Camillo Marra https://orcid.org/0000-0003-3994-4044
João Massano https://orcid.org/0000-0002-5791-7149
Flavio Nobili https://orcid.org/0000-0001-9811-0897
Miguel Tábuas-Pereira https://orcid.org/0000-0002-3988-614X
Stoyan Popkirov https://orcid.org/0000-0001-6168-0036
Mirella Russo https://orcid.org/0000-0002-9937-5923
Emmi P. Scott https://orcid.org/0000-0002-1190-3470
Tim Wilkinson https://orcid.org/0000-0001-8952-0982
Lisbeth Frostholm https://orcid.org/0000-0002-9683-7416
Jon Stone https://orcid.org/0000-0001-9829-8092

REFERENCES

1. Ball HA, McWhirter L, Ballard C, et al. Functional cognitive disorder: dementia's blind spot. *Brain*. 2020;143(10):2895-2903.

2. McWhirter L, Ritchie C, Stone J, Carson A. Functional cognitive disorders: a systematic review. *Lancet Psychiatry*. 2020;7(2):191-207.
3. Hallett M, Aybek S, Dworetzky BA, McWhirter L, Staab JP, Stone J. Functional neurological disorder: new subtypes and shared mechanisms. *Lancet Neurol*. 2022;21(6):537-550.
4. McWhirter L, Sargent B, Ritchie C, Stone J, Carson A. I think, therefore I forget - using experimental simulation of dementia to understand functional cognitive disorders. *CNS Spectr*. 2020;25(4):511-518.
5. Teodoro T, Koreki A, Chen J, et al. Functional cognitive disorder affects reaction time, subjective mental effort and global metacognition. *Brain*. 2023;146(4):1615-1623.
6. Larner AJ. Functional cognitive disorders (FCD): how is metacognition involved? *Brain Sci*. 2021;11(8):1082.
7. Jones D, Drew P, Elsey C, et al. Conversational assessment in memory clinic encounters: interactional profiling for differentiating dementia from functional memory disorders. *Aging Ment Health*. 2016;20(5):500-509.
8. Bailey C, Poole N, Blackburn DJ. Identifying patterns of communication in patients attending memory clinics: a systematic review of observations and signs with potential diagnostic utility. *Br J Gen Pract*. 2018;68(667):e123-e138.
9. Reuber M, Blackburn DJ, Elsey C, et al. An interactional profile to assist the differential diagnosis of neurodegenerative and functional memory disorders. *Alzheimer Dis Assoc Disord*. 2018;32(3):197-206.
10. Cabreira V, Frostholm L, McWhirter L, Stone J, Carson A. Clinical signs in functional cognitive disorders: a systematic review and diagnostic meta-analysis. *J Psychosom Res*. 2023;173:111447.
11. Larner AJ. Screening utility of the attended alone sign for subjective memory impairment. *Alzheimer Dis Assoc Disord*. 2014;28(4):364-365.
12. Silverberg ND, Rush BK. Neuropsychological evaluation of functional cognitive disorder: a narrative review. *Clin Neuropsychol*. 2024;38(2):302-325.
13. Pennington C, Newson M, Hayre A, Coulthard E. Functional cognitive disorder: what is it and what to do about it? *Pract Neurol*. 2015;15(6):436-444.
14. Bhome R, McWilliams A, Price G, et al. Metacognition in functional cognitive disorder. *Brain Commun*. 2022;4(2):fcac041.
15. Larner AJ. Metacognition in functional cognitive disorder: contradictory or convergent experimental results? *Brain Commun*. 2022;4(3):fcac138.
16. Teodoro T, Edwards MJ, Isaacs JD. A unifying theory for cognitive abnormalities in functional neurological disorders, fibromyalgia and chronic fatigue syndrome: systematic review. *J Neurol Neurosurg Psychiatry*. 2018;89(12):1308-1319.
17. Bhome R, Huntley JD, Price G, Howard RJ. Clinical presentation and neuropsychological profiles of functional cognitive disorder patients with and without co-morbid depression. *Cogn Neuropsychiatry*. 2019;24(2):152-164.
18. McWhirter L, Carson A. Functional cognitive disorders: clinical presentations and treatment approaches. *Pract Neurol*. 2023;23(2):104-110.
19. McKenna HP. The Delphi technique: a worthwhile research approach for nursing? *J Adv Nurs*. 1994;19(6):1221-1225.
20. Goodman CM. The Delphi technique: a critique. *J Adv Nurs*. 1987;12(6):729-734.
21. Polit D, Hungler BF. *Essentials of Nursing Research: Methods, Appraisal, and Utilization*. Lippincott-Raven; 1997.
22. Landis JR, Koch GG. The measurement of observer agreement for categorical data. *Biometrics*. 1977;33(1):159-174.
23. Frisoni GB, Festari C, Massa F, et al. European intersocietal recommendations for the biomarker-based diagnosis of neurocognitive disorders. *Lancet Neurol*. 2024;23(3):302-312.
24. Frisoni GB, Altomare D, Ribaldi F, et al. Dementia prevention in memory clinics: recommendations from the European task force for brain health services. *Lancet Reg Health Eur*. 2023;26:100576.
25. Bertens D, Vos S, Kehoe P, et al. Use of mild cognitive impairment and prodromal AD/MCI due to AD in clinical care: a European survey. *Alzheimers Res Ther*. 2019;11(1):74.
26. Bailey C, Bell SM, Blackburn DM. How the UK describes functional memory symptoms. *Psychogeriatrics*. 2017;17(5):336-337.
27. Saunders S, Ritchie CW, Russ TC, Muniz-Terrera G, Milne R. Assessing and disclosing test results for 'mild cognitive impairment': the perspective of old age psychiatrists in Scotland. *BMC Geriatr*. 2022;22(1):50.
28. Stone J, Pal S, Blackburn D, Reuber M, Thekkumpurath P, Carson A. Functional (psychogenic) cognitive disorders: a perspective from the neurology clinic. *J Alzheimers Dis*. 2015;48(Suppl. 1):S5-S17.
29. Ball HA, Swirski M, Coulthard EJ, Newson M, Pennington CM. Differentiating functional cognitive disorder from early neurodegeneration: a clinic-based study. *Brain Sci*. 2021;11(6):800.
30. Tinazzi M, Geroin C, Marcuzzo E, et al. Functional motor phenotypes: to lump or to split? *J Neurol*. 2021;268(12):4737-4743.
31. Paredes-Echeverri S, Guthrie AJ, Perez DL. Toward a possible trauma subtype of functional neurological disorder: impact on symptom severity and physical health. *Front Psych*. 2022;13:1040911.
32. Pennington C, Ball H, Swirski M. Functional cognitive disorder: diagnostic challenges and future directions. *Diagnostics*. 2019;9(4):131.
33. Pareés I, Saifee TA, Kojovic M, et al. Functional (psychogenic) symptoms in Parkinson's disease. *Mov Disord*. 2013;28(12):1622-1627.
34. Piliavska K, Dantlgraber M, Dettmers C, Jöbges M, Liepert J, Schmidt R. Functional neurological symptoms are a frequent and relevant comorbidity in patients with multiple sclerosis. *Front Neurol*. 2023;14:1077838.
35. Clark CN, Edwards MJ, Ong BE, et al. Reframing postconcussional syndrome as an interface disorder of neurology, psychiatry and psychology. *Brain*. 2022;145(6):1906-1915.
36. Rioux M, Wardell V, Palombo DJ, Picon EL, Le ML, Silverberg ND. Memory for forgetting in adults with persistent symptoms following concussion. *J Clin Exp Neuropsychol*. 2022;44(1):19-30.
37. Picon EL, Todorova EV, Palombo DJ, Perez DL, Howard AK, Silverberg ND. Memory perfectionism is associated with persistent memory complaints after concussion. *Arch Clin Neuropsychol*. 2022;37(6):1177-1184.
38. Potter S, Brown RG. Cognitive behavioural therapy and persistent post-concussional symptoms: integrating conceptual issues and practical aspects in treatment. *Neuropsychol Rehabil*. 2012;22(1):1-25.
39. Van Patten R, Chan L, Tocco K, et al. Improvements in Montreal Cognitive Assessment scores after neurobehavioral therapy in adults with functional (nonepileptic) seizures and traumatic brain injury. *J Psychiatr Res*. 2023;165:282-289.
40. LaFrance WC Jr, Tedesco R, Baird GL, Wincze J, Tocco K, Anderson J. Clinician-rated outcomes of patients with functional neurological disorders treated in an outpatient clinic. *Seizure*. 2023;107:21-27.
41. Mascherek A, Zimprich D, Rupprecht R, Lang FR. What do cognitive complaints in a sample of memory clinic outpatients reflect? *GeroPsych*. 2011;24(4):187-195.
42. Bhome R, Berry AJ, Huntley JD, Howard RJ. Interventions for subjective cognitive decline: systematic review and meta-analysis. *BMJ Open*. 2018;8(7):e021610.
43. Adkins-Jackson PB, Belsky DW. Alzheimer's disease risk biomarkers: progress and challenges. *Lancet Healthy Longev*. 2022;3(9):e575-e576.
44. Cummings J, Lee G, Nahed P, et al. Alzheimer's disease drug development pipeline: 2022. *Alzheimers Dement*. 2022;8(1):e12295.

45. Gregory S. Three promising drugs for treating Alzheimer's disease bring fresh hope. 2023. Accessed November, 2023. https://www.alzheimers.org.uk/blog/three-promising-drugs-for-treating-alzheimers-disease-bring-fresh-hope
46. van Tol M-J, van der Wee NJA, van den Heuvel OA, et al. Regional brain volume in depression and anxiety disorders. *Arch Gen Psychiatry*. 2010;67(10):1002-1011.
47. van Maurik IS, Vos SJ, Bos I, et al. Biomarker-based prognosis for people with mild cognitive impairment (ABIDE): a modelling study. *Lancet Neurol*. 2019;18(11):1034-1044.
48. O'Malley RPD, Mirheidari B, Harkness K, et al. Fully automated cognitive screening tool based on assessment of speech and language. *J Neurol Neurosurg Psychiatry*. 2021;92:12-15. doi:10.1136/jnnp-2019-322517
49. Al-Hameed S, Benaissa M, Christensen H, Mirheidari B, Blackburn D, Reuber M. A new diagnostic approach for the identification of patients with neurodegenerative cognitive complaints. *PLoS One*. 2019;14(5):e0217388.
50. Mirheidari B, Blackburn D, Harkness K, et al. Toward the automation of diagnostic conversation analysis in patients with memory complaints. *J Alzheimers Dis*. 2017;58(2):373-387.
51. Walker T, Christensen H, Mirheidari B, et al. Developing an intelligent virtual agent to stratify people with cognitive complaints: a comparison of human-patient and intelligent virtual agent-patient interaction. *Dementia*. 2020;19(4):1173-1188.
52. Wakefield SJ, Blackburn DJ, Harkness K, Khan A, Reuber M, Venneri A. Distinctive neuropsychological profiles differentiate patients with functional memory disorder from patients with amnestic-mild cognitive impairment. *Acta Neuropsychiatr*. 2018;30(2):90-96.
53. Millstein D, Perez D, Langfitt J. A case of functional neurological disorder with cognitive symptoms: emotion-focused psychotherapeutic insights. *J Acad Consult Liaison Psychiatry*. 2023;64(3):307-308.
54. Godena E, Freeburn J, Silverberg N, Perez D. A case of functional cognitive disorder: psychotherapy and speech and language therapy insights. *Harv Rev Psychiatry*. 2023;31:248-256.
55. Metternich B, Schmidtke K, Dykierek P, Hüll M. A pilot group therapy for functional memory disorder. *Psychother Psychosom*. 2008;77(4):259-260.
56. Norman P, Sarah C, Serena V, et al. Feasibility randomised controlled trial of online group acceptance and commitment therapy for functional cognitive disorder (ACT4FCD). *BMJ Open*. 2023;13(5):e072366.
57. Blackburn DJ, Wakefield S, Shanks MF, Harkness K, Reuber M, Venneri A. Memory difficulties are not always a sign of incipient dementia: a review of the possible causes of loss of memory efficiency. *Br Med Bull*. 2014;112(1):71-81.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Cabreira V, Alty J, Antic S, et al. Perspectives on the diagnosis and management of functional cognitive disorder: An international Delphi study. *Eur J Neurol*. 2024;00:e16318. doi:10.1111/ene.16318