Functional cognitive disorders: a systematic review

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Cognitive symptoms are common, and yet many who seek help for cognitive symptoms neither have, nor go on to develop, dementia. A proportion of these people are likely to have functional cognitive disorders, a subtype of functional neurological disorders, in which cognitive symptoms are present, associated with distress or disability, but caused by functional alterations rather than degenerative brain disease or another structural lesion. In this Review, we have systematically examined the prevalence and clinical associations of functional cognitive disorders, and related phenotypes, within the wider cognitive disorder literature. Around a quarter of patients presenting to memory clinics received diagnoses that might indicate the presence of functional cognitive disorders, which were associated with affective symptoms, negative self-evaluation, negative illness perceptions, non-progressive symptom trajectories, and linguistic and behavioural differences during clinical interactions. Those with functional cognitive disorder phenotypes are at risk of iatrogenic harm because of misdiagnosis or inaccurate prediction of future decline. Further research is imperative to improve diagnosis and identify effective treatments for functional cognitive disorders, and better understanding these phenotypes will also improve the specificity of diagnoses of prodromal degenerative brain disease.

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Introduction

Increasing numbers of people seek help for memory problems, and yet many symptomatic patients attending memory clinics do not have degenerative brain disease, and do not progress to dementia.[1,2] Cognitive symptoms or impairment might be caused by other medical and neurological disorders, or by prescribed or non-prescribed drugs, but the experience of cognitive failure can also arise through purely functional disturbances to cognitive and introspective processes.

Functional cognitive disorders are a group of overlapping conditions in which cognitive symptoms are present, which are genuine, distressing, and often disabling, but experienced inconsistently and not related to systemic or brain disease (panel).[3] They can be included under the umbrella of functional neurological disorders, one of the most common causes of neurological disability.[5,6] Although historically defined in terms of psychological stress and absence of disease, functional neurological disorders are now also understood in neurobiological terms, with evidence of dysregulated attention, sensorimotor prediction, self-agency, and emotional processing.[7,8] Psychological stressors are no longer required for the diagnosis of functional neurological disorder, which, crucially, is only made on the basis of positive clinical features showing characteristic internal inconsistency; misdiagnosis is rare.[9]

Functional cognitive symptoms have received less research attention than other functional symptoms, although the interest in the field is developing. Teodoro and colleagues[4] systematically reviewed the literature on so-called brain fog, and cognitive symptoms in functional neurological disorders, fibromyalgia, and chronic fatigue syndrome, suggesting a unifying theory in which excessive attention towards physical symptoms and cognitive processes generates symptoms. Bailey and colleagues[10] systematically reviewed patterns of communication in memory clinics, identifying features with potential to discriminate between functional and neurodegenerative disorders: individuals with functional disorders were more likely to attend alone, to be worried about their memory, and to provide a detailed account of personal history and memory failures than patients with neurodegenerative disease.

However, despite increasing interest in identifying early prodromes of degenerative brain diseases, no detailed examination has been done of the prevalence and clinical

Panel: Definition and subtypes of functional cognitive disorders

Definition

- One or more symptoms of impaired cognitive function are present
- Clinical findings show evidence of internal inconsistency: with observed or measured function, or between different situations
- Symptoms or impairment are not better explained by another medical disorder, although might be comorbid with another medical disorder
- Symptoms or impairment cause clinically substantial distress or impairment in social, occupational, or other important areas of function, or warrant medical evaluation

Proposed overlapping subtypes (after Stone and colleagues[3]):

- Excessive attentional focus on normal cognitive symptoms
- Health anxiety about dementia, with perceived cognitive deficit
- Isolated functional cognitive symptoms with or without impairment on cognitive tests
- Cognitive symptoms as part of anxiety or depression
- Cognitive symptoms in other functional disorders—eg, functional neurological disorders (ie, dissociative seizures and functional movement disorders), chronic fatigue syndrome, and fibromyalgia (in which cognitive symptoms are often described as brain fog; not included in this Review—see Teodoro and colleagues[4])
- Dissociative cognitive states (eg, dissociative amnesia, fugue, Ganser syndrome)

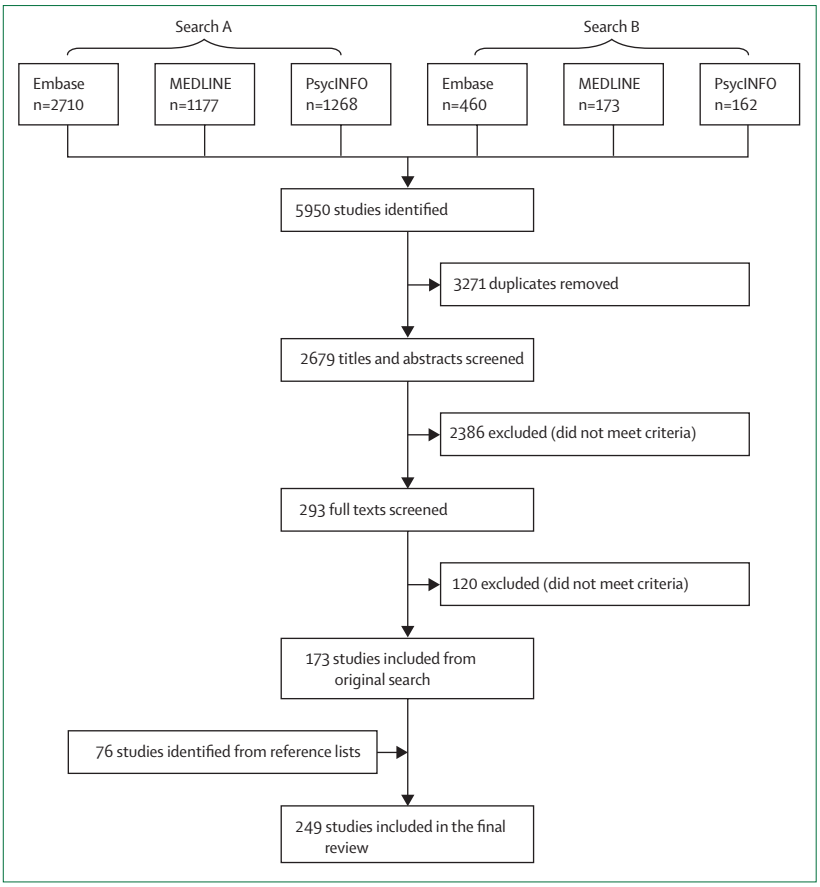

Figure 1: **Selection of included studies**

associations of functional cognitive disorders (an important differential diagnosis) in the cognitive disorder literature. One reason for this scarcity of such research might be that the scientific literature concerning functional cognitive disorders is a tangled landscape of overlapping terminology.

Physicians from the early 20th century used the term pseudodementia to describe a wide range of clinical syndromes with the appearance of dementia but rather caused by depression, conversion disorders (also sometimes called hysteria), dissociative states (including so-called Ganser states), or disordered personality.[11–13] The broader pseudodementia concept has been superseded by depressive pseudodementia (ie, cognitive impairment associated with severe depression), although with better recognition of the frequency of depression and anxiety in prodromal degenerative brain disease, this clinical group is seen to be aetiologically heterogenous.

See Online for appendix

During the past 10 years, researchers investigating subjective cognitive decline have been strongly invested in identifying early clinical markers of neurodegenerative disease, rarely focusing on alternative causes of symptoms. People with subjective cognitive complaints but normal cognitive examination are sometimes described, unhelpfully, as worried well (ie, describing worry about experiences that fall within the range of normal, and that are not because of disease). Of equal concern, people with both subjective cognitive complaints and impairment on testing (therefore defined as having mild cognitive impairment), or with subjective cognitive complaints and biomarkers suggestive of an underlying disease process, might receive life-changing predictions or diagnoses of dementia that are retained even when inconsistent symptom experience and subsequent cognitive trajectory are more consistent with a functional disorder than dementia.[14]

An almost universal tendency exists in dementia research to view subjective cognitive symptoms as a preliminary to mild cognitive impairment and later dementia. However, an as yet undefined proportion of those individuals with symptoms described in terms of subjective cognitive decline, subjective memory impairment, pseudodementia, or as the worried well, might be better described in positive terms as having the inclusively generated diagnosis of functional cognitive disorders—challenging the prevailing model that subjective cognitive decline always leads to mild cognitive impairment, which in turn always leads to dementia. We aimed to systematically search and review the literature incorporating these diverse terms to assess their usage, and the prevalence and clinical associations of functional cognitive disorders in people with cognitive symptoms.

Methods

Search strategy and selection criteria

We did two simultaneous searches (A and B) of the published peer-reviewed English language literature in MEDLINE, Embase, and PsycINFO databases from inception to March 14, 2019. We included observational studies describing the cross-sectional diagnoses of those assessed for possible dementia in memory clinics or similar services, and observational studies (excluding treatment studies) that included (albeit not necessarily as a primary focus) original data on at least ten adults (>18 years) with subjective cognitive symptoms, arising de novo, who did not receive a diagnosis of dementia, delirium, or other medical or neurological causes of symptoms. Exclusion criteria (not applied to cross-sectional studies of memory clinics) were: primary diagnosis of non-cognitive functional neurological disorder, chronic fatigue syndrome, fibromyalgia, major psychiatric disorder other than depressive or anxiety disorders, or cognitive symptoms after physical illness or injury. The search, screening, and data extraction were done by one author (LM). Data were synthesised into a narrative review. Full list of terms used can be found in the appendix (p 30).

Results

Of the 249 included studies (figure 1, appendix pp 19–29), 185 studies had a cross-sectional design, 59 studies had

a longitudinal design, and five studies described case series with at least ten people; 59 studies included at least one control group. A wide range of terms was used to describe non-dementia cognitive symptom profiles and diagnoses (appendix p 1).

Population prevalence and outcomes

38 studies (appendix pp 2–6) described diagnoses in 40 clinical populations attending cognitive assessment services: all were in memory clinic (or similar) settings except for the earliest three studies of inpatients investigated for suspected dementia, reflecting clinical practice at the time.[15–17] The 38 studies included 13 637 people (57% female),[15–51] excluding Wright and Lindesay's survey[52] in which the number of participants was not reported. Studies used various terminologies and reported various degrees of descriptive detail. Of these 38 studies, 35 studies reported dementia diagnoses in 7173 (54%) of 13 353 patients, 32 studies reported presence of clinical syndromes of subjective cognitive impairment, pseudodementia, or functional cognitive disorders in 2832 (24%) of 12 003 patients, 30 studies reported both functional cognitive disorder prevalence (2808 [24%] of 11 807 patients) and dementia prevalence (6285 [53%] of 11 807 patients; figure 2), and five studies reported no cognitive disorder in 616 (47%) of 1324 patients.

Cognitive disorders might also emerge during treatment of medical illness: 8 (5%) of 166 medical inpatients (mean age 82·9 years) with severe acquired cognitive deficits suggestive of dementia were ultimately diagnosed with depressive pseudodementia.[53]

If cognitive symptoms always represent steps on a trajectory towards dementia, every person with subjective cognitive impairment would be expected to progress to mild cognitive impairment, then dementia, with ongoing decline from the point of dementia diagnosis. Atypical trajectories (ie, non-progressive, remitting, or fluctuating) are potential markers of functional cognitive disorders. Although complete meta-analysis of the longitudinal outcome of subjective cognitive symptoms was outside of the scope of this Review, we examined the included studies to consider whether, in broad terms, non-progressive cognitive problems were common or rare in those presenting for clinical assessment.

Three studies from before 1980 examined stability of dementia diagnoses. In Kendell's study[54] of the temporal stability of psychiatric diagnoses in 2000 patients first admitted to a psychiatric bed in 1964, dementia was the most stable of all psychiatric diagnoses at 75%; indicating, however, that 25% of those diagnosed with dementia severe enough to lead to hospital admission were ultimately rediagnosed with something else. In another 10-year, case-note review of 35 inpatients diagnosed with presenile (<65 years) dementia, 15 (43%) deteriorated as expected of whom ten died, but 20 (57%) did not deteriorate of whom 18 improved and two were unchanged; with revised diagnoses, including depression

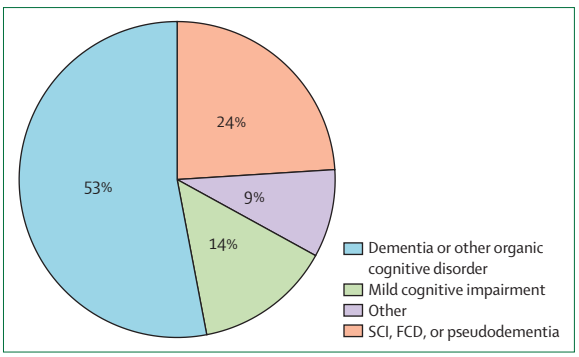

Figure 2: **Diagnoses in people attending memory clinics**
This chart shows the pooled distribution of diagnoses in the 30 included studies (n=11 807) of memory clinics (and similar services) that include adequate description of dementia and non-dementia diagnoses. Studies reporting not dementia without alternative description are not represented in this chart. SCI=subjective cognitive impairment. FCD=functional cognitive disorder.

for three patients, anxiety state for three patients, somatic symptoms without organic basis for six patients, and hysterical reaction for one patient, the authors stated that the non-progressors consisted mainly of "people with marked personality difficulties and neurotic symptoms or affective disorder".[55]

Ten studies followed up clinical populations assessed at baseline as having subjective cognitive symptoms of uncertain or benign cause: none reported rates of progression to dementia greater than 10% during 2–4 years of follow up.[56–66] If subjective cognitive decline were mostly due to degenerative brain disease, this condition would be expected to be associated with early death. However, two included studies reported no reduction in life expectancy in individuals with subjective cognitive decline over mean follow-up periods of 3·5–4 years.[67,68]

Poor outcomes were reported in pseudodementia cohorts, both in terms of incident dementia and non-neurodegenerative mortality, although progression to dementia varied from 0–89%. Bulbena and Berrios[69] followed up 22 individuals (mean age 73·3 years) with pseudodementia: ten patients were subsequently diagnosed unipolar depression, five patients with bipolar disorder, five patients with psychosis, and two patients with personality disorder. After 15–47 months, eight (36%) had died, and six (27%) of 14 survivors had developed dementia.[69] Sachdev[70] followed up 19 individuals (mean age 53 years) with pseudodementia: eight patients were subsequently diagnosed with depression, three patients with bipolar depression, five patients with schizophrenia, two patients with mania, and one patient with schizophreniform disorder. Over 12 years, eight (42%) died but none of the 11 (58%) survivors developed dementia.[70] However, Kral and Emery[71] reported onset of dementia within 8 years in 39 (89%) of 44 individuals (mean age 76·5 years) with pseudodementia, despite initial resolution of affective and cognitive symptoms.[72] Similarly, 15 (71%) of 21 individuals (mean age 78 years) with depressive

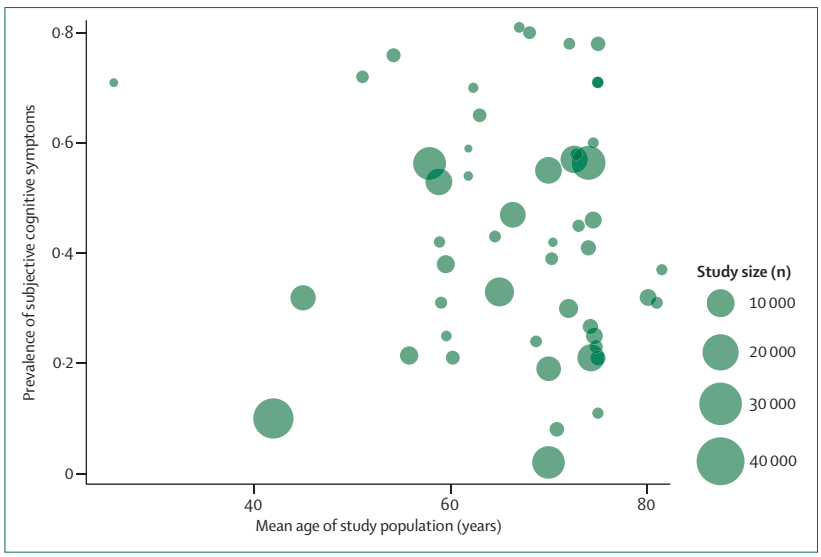

Figure 3: **Cognitive symptom prevalence versus mean sample age**
Subjective cognitive symptom prevalence in 49 included studies of community populations (not including seven included studies in which sample age was not reported).

pseudodementia followed up over 5–7 years developed dementia.[71]

Schmidtke and colleagues[73] reported outcome (symptom severity, subjective memory function, and psychosocial burden) in the 46 individuals who attended a follow-up examination (total of 73; mean age 55·2 years) diagnosed with functional memory disorder: 39 (85%) had persistent symptoms at mean follow-up of 20·1 months, symptoms had resolved in six (13%), and one (2·1%) had dementia. Risk of incident dementia therefore was low (nonetheless present) but symptom persistence was the notable finding here, suggesting functional memory disorder is not a benign condition.

Cognitive symptoms

Estimation of the general prevalence of cognitive symptoms is important to understand who presents for clinical assessment and why. 56 studies described prevalence of cognitive symptoms in community populations (appendix pp 7–11), with a variety of assessment methods, finding symptoms in between 8% and 80%, with overall 30% of the 245 654 individuals included reported to have cognitive symptoms.[74–129]

Of those cross-sectional studies including objective measures of cognitive function, 18 studies found a positive association between symptoms and objective impairment,[74–77,84–87,89,92,97,98,102,105,106,108,120,127] but 14 studies did not.[79,81,83,95,96,99,104,113,117,119,126,128–130] Some reported symptom association with impairment in subgroups: specific rather than global cognitive symptoms,[80] subjective cognitive decline with additional clinical or biomarkers suggesting neurodegenerative disease but not subjective cognitive decline alone,[103] and only in male participants.[91] No correlation was found between prevalence of reported cognitive symptoms and mean study population age, although this result must be interpreted with caution given the different measures used (figure 3).

We established in our first step that cognitive symptoms are common. In our second step towards understanding why people present with cognitive symptoms, we identified seven studies reporting factors associated with seeking help for cognitive symptoms.

When comparing patients in memory clinics who were self referred with those who were physician referred, self-referrers reported greater decline, had more depressive symptoms, more trait anxiety, higher estimated premorbid intelligence quotient, and were more likely to have had previous depression requiring treatment.[131,132] Four other studies reported that help-seekers (those who have sought medical help for subjective memory symptoms) had poorer memory self-efficacy, poorer quality of life, were more often worried because of a family history of dementia,[133] were more likely to perceive a biological or medical (rather than social) cause of memory problems,[134] and had more depressive symptoms and hippocampal atrophy than symptomatic non-help-seekers (people identified from population surveys as experiencing subjective memory symptoms but who had not sought medical help for these) despite similar cognitive scores, anxiety scores, and cerebral amyloid deposition.[135] Haussman[136] found that intrinsic motivation (ie, attending because of self rather than others) reduced likelihood of dementia diagnosis. In a case study of Ganser syndrome,[137] every presentation was assessed to be motivated by external circumstances, including avoidance of murder trial, head injury compensation, and dissatisfaction with army life. Those seeking help for subjective cognitive symptoms are more likely to be distressed, depressed, and anxious, and to be more concerned than others about their memory; they cannot be considered worried well. A possibility exists that a substantial proportion have functional cognitive disorders (panel).

Comprehensive meta-analysis of longitudinal outcomes was outside of the scope of this Review, but we aimed to summarise the range of outcomes in included studies to consider broadly whether cognitive symptoms, in those who do not necessarily seek help, frequently or infrequently progress to dementia. 26 studies reported outcomes in non-clinical populations with subjective cognitive symptoms after 1–10 years. In 13 cohorts, baseline subjective cognitive symptoms were associated with increased risk of future cognitive decline, although in studies reporting incident dementia rather than decline on cognitive tests, patients progressing to dementia were few, and at most 19 (11%) of 166 patients with subjective cognitive impairment who were followed up over 7 years[138] in any individual study population.[76,77,87,97,98,115,127,139–143] Two studies, in 453 individuals (mean age 80·5 years) and 1990 individuals (mean age 80·1 years), reported increased risk of progression in stable but not unstable (ie, relapsing and remitting) subjective cognitive decline.[92,121]

Amariglio and colleagues[144] found symptoms predictive of decline only in individuals with amyloid-positive profiles on Pittsburgh compound B (PiB) PET in a cohort of 279 individuals (mean age 73·7 years). In a cohort of 1416 individuals (mean age 75·3 years), subjective cognitive decline no longer predicted decline after adjusting for baseline cognitive performance.[127]

Six studies (all mean age >65 years) reported that symptoms did not predict future decline.[58,106,119,145–147] Three studies described predictors of future increases in cognitive symptoms: low control beliefs (corresponding to low or external locus of control),[148] female sex, fear of falling, anxiety and depression,[149] and longitudinal change in cognitive performance.[150]

In keeping with systematic reviews assessing this specific question,[151] although some individuals with subjective cognitive symptoms progress to dementia, the majority (around 90%) do not.

We hypothesise that a substantial proportion of those with subjective cognitive symptoms in clinical populations have functional cognitive disorders. By including biomarker studies, we intended not to assess the predictive value of these biomarkers per se, but rather to consider the clinically important question of what patterns of results might be found in those with functional cognitive disorders.

Structural and functional neuroimaging and metabolic imaging

As cerebral atrophy is a key marker of degenerative brain disease, and medial temporal lobe atrophy a marker of Alzheimer's disease, functional cognitive disorders (assumed here to represent many of those with subjective cognitive symptoms) might be expected to be associated with an absence or relatively small degree of atrophy. Several included studies confirmed this hypothesis by finding a degree of global atrophy unrelated to measures of cognitive function in individuals complaining of memory loss[47] or to symptom severity,[152] and three studies reported no difference in brain volume between groups with subjective memory complaints and healthy controls.[153–155]

Two studies reported greater medial temporal volumes in patients with depressive pseudodementia than in patients with Alzheimer's disease, and one reported greater hippocampal volumes in patients with subjective cognitive impairment than in patients with Alzheimer's disease or mild cognitive impairment.[156–158] One study defined a non-neurodegenerative subjective memory impairment subtype with minimal atrophy.[159] However, six studies reported smaller hippocampal volumes in patients with subjective memory impairment and smaller hippocampi in less depressed patients with subjective memory impairment,[160] in patients with subjective memory impairment and a family history of Alzheimer's disease,[161] and in association with help-seeking,[135] than in healthy controls.[135,157,160–162] A study of dementia syndrome of depression reported atrophy intermediate between unimpaired depressed individuals and those with Alzheimer's disease.[163] In 60 patients (mean age 72·6 years) assessed at a memory clinic, white matter lesion severity was associated with subjective memory symptoms and depression severity.[164] Superior temporal gyrus atrophy was associated with depressive symptoms in unaccompanied attenders to memory-clinics, the authors proposing that depression was the cause of atrophy, rather than the result.[165]

Just as the absence of atrophy cannot exclude degenerative disease, the presence of atrophy is not specific: a study describing an atrophy pattern like that of Alzheimer's disease present in 13% (11/86) of those with subjective memory impairment reported that 27% (3/11) of symptomatic individuals with this pattern did not progress within 90 months.[166]

Rodda and colleagues[167] reported increased functional MRI activation in the left medial temporal lobe, bilateral thalamus, posterior cingulate, and caudate in patients with subjective cognitive impairment compared with healthy controls. Kawagoe and colleagues[152] described increased resting-state functional connectivity, related to symptom severity, in the lingual gyrus, anterior insula, and superior parietal lobe. The authors of both studies suggest the observations might reflect compensatory activity in early neurodegenerative disease. By contrast, Hu and colleagues[168] described absent hippocampal activation during a choice-making task in people with subjective cognitive decline compared with healthy controls.

A PET study of regional cerebral blood flow reported decreased flow in left anterior medial prefrontal cortex and increased flow in the cerebellar vermis in patients with major depression and substantial cognitive impairment compared with depressed patients without cognitive impairment.[169] Gucuyener and colleagues[170] reported no differences in cerebral blood velocities as measured by transcranial doppler ultrasound between patients with depressive pseudodementia and controls with Alzheimer's disease, but impaired vasoneural reactivity to visual stimuli only in Alzheimer's disease.

If subjective cognitive symptoms often represented a prodrome of Alzheimer's disease, an association with increased cerebral amyloid deposition would be expected, although amyloid is not specific to Alzheimer's disease.[171] Five studies examined cerebral amyloid burden in subjective cognitive impairment or decline, with PiB or ^{18}F-florbetapir ligands.

Results were mixed. One study reported no difference in amyloid between community participants with subjective cognitive impairment and healthy controls.[154] Another reported more amyloid in clinical and community participants with subjective cognitive decline than in healthy controls.[135]

Three studies examined amyloid in relation to cognitive symptoms, finding no association with global cognitive

symptom scores, although Amariglio and colleagues[149] reported a specific association with impaired memory and Perrotin and colleagues[135] reported, somewhat tenuously, that although those with higher PiB uptake did not report inferior memory, they were less likely to report superior memory than than those with lower PiB uptake.[149,170,173]

Overall, of the five included amyloid PET studies, only Perrotin and colleagues[135] reported a clear association with presence of subjective cognitive symptoms, reporting amyloid-positivity in three (9%) of 35 controls, eight (29%) of 28 clinical participants with subjective cognitive decline, and 12 (34%) of 35 community participants with subjective cognitive decline.

Neurophysiological measures, genetic variables, and cerebrospinal fluid biomarkers

The authors of two papers from before 1990 (one paper describing pseudodementia, and the other mixed depression and dementia) described diagnostic use of electroencephalography (EEG): EEG results were more often abnormal in patients with Alzheimer's disease than in those with depression alone.[12,174] Hutton[175] reported worse eye tracking in Alzheimer's disease compared with pseudodementia and healthy controls. Examining the P300 late-evoked potential in passive listening and oddball tasks, Gottlieb[176] reported no difference between individuals with pseudodementia and healthy controls. Cespon and colleagues[177] reported greater medial frontal negativity (a correlate of conflict monitoring) in those with higher levels of subjective memory complaints than those with lower levels.

Prodromal (indeed, preclinical) Alzheimer's disease would be expected to be associated with an increased risk of carrying the *APOE* ε4 allele, the most penetrant genetic risk factor for sporadic Alzheimer's disease; but in keeping with systematic reviews of this specific question, five included studies of people with subjective memory symptoms found no increase in *APOE* ε4 allele prevalence.[129,155,158,173,178,179]

One study found an Alzheimer's disease profile of cerebrospinal fluid (CSF; pathological $A\beta_{42}$:total-tau ratio) more frequent in patients with subjective cognitive impairment (52%; 31/60) than in healthy controls (31%; 28/89).[59] Eckerstrom and colleagues[180] and Garcia-Ptacek and colleagues[158] found CSF biomarkers more frequently normal in patients with subjective cognitive impairment than in patients with mild cognitive impairment or Alzheimer's disease. Overall, those with subjective cognitive symptoms appear more likely to have pathological biomarkers than controls, but less likely than those with objective impairment; many described as having subjective cognitive impairment do not have markers of degenerative brain disease.

Six included studies reported outcomes of subjective cognitive symptoms in relation to CSF biomarkers for Alzheimer's disease. Visser[59] reported that no patients with subjective cognitive impairment progressed to dementia (including those with a pathological $A\beta_{42}$:total-tau ratio) by 2·3 years. Van Harten and colleagues[62] reported that low $A\beta_{42}$ alone (without abnormal total or phosphorylated tau) predicted progression in a clinical population with subjective complaints, but numbers were small: of 132 people with subjective complaints, ten had low $A\beta_{42}$, of whom two (18%) declined over 2 years.

In another cohort described by the same authors, 12 (10%) of 115 patients with subjective complaints had low $A\beta_{42}$, of whom eight (62%) declined.[63] Sierra-Rio and colleagues[181] reported that pathological $A\beta_{42}$:phosphorylated tau ratio was associated with progression in subjective cognitive decline; but out of 55 patients, 11 patients had this profile of whom only three (27%) declined.

Although CSF profiles for people with Alzheimer's disease might be slightly more common in subjective cognitive decline than in normal controls, the predictive value for any individual is uncertain, as eloquently shown by a longitudinal study in which CSF biomarkers did not improve clinicians' diagnostic or prognostic accuracy in suspected cognitive disorder; sensitivity was the same but specificity lower when CSF biomarker status was available, with most resulting in false positive predictions in those with subjective complaints only.[182]

Neuropsychological test performance and interactional and linguistic features

Neuropsychological tests are a prerequisite in all dementia diagnostic criteria. How people with functional cognitive disorders perform in such tests is important to consider to understand when and how to use them in diagnosis. 13 studies examined neuropsychological test performance in people with subjective symptoms (a proportion of whom are likely to have functional disorders) in comparison with healthy, mild cognitive impairment, or dementia groups (appendix p 12):[133,155,156,168,183–192] participants generally did similarly to or worse than healthy controls, but better than groups with mild cognitive impairment or dementia.

19 studies examined the relationship between subjective cognitive symptoms and objective cognitive performance (appendix p 13),[152,164,173,184,192–208] with ten reporting a relationship between symptom report and measured cognition in at least a subset of participants and nine finding no relationship. In cases of discord, the memory complaint exceeded impairment. Three studies reported that neuropsychological tests had predictive value in subjective cognitive symptoms, reporting associations with decline at 1 year, 2 years, and 7 years.[57,209,210] However, although analysis of specific tests and forgetting index in one study identified 79% (128/162) of those with cognitive complaints converting to dementia within 5–6 years, this model also incorrectly predicted dementia in a substantial 20% (18/88).[211]

Jansen and colleagues[212] did not find that neuropsychological assessment improved the dementia

classification in 221 patients attending memory clinics, increasing false positive predictions of decline in those with subjective cognitive impairment. Although mild baseline impairment seems more probable in those with degenerative brain disease than in those without, the predictive value of neuropsychological testing for any individual is inaccurate.

Specific cognitive features were described in ten patients with Ganser syndrome: amnesia, approximate answers (ie, incorrect answers that show knowledge of the correct answer), fugue or trance-like state, and hallucinations.[137] The approximate answer shows internal inconsistency, and can be considered a (rarely described) positive sign of functional cognitive disorder. Validity tests also show internal inconsistency, although the utility of validity test failure in discriminating prodromal degenerative brain disease from functional disorders is unclear. One study reported that 11 (6·5%) of 170 (13% of those younger than 65 years) patients in memory clinics with mild cognitive impairment, uncertain diagnosis, or worried well scored in a non-credible range on the Word Memory Test or Test of Memory Malingering or both.[213]

Some groups have examined interactional and linguistic features during the consultation. As clinical consensus was used as the so-called gold standard diagnosis in most studies, we note risk of diagnostic suspicion bias, in which clinicians might use the assessed features consciously or subconsciously to make the diagnosis.

11 studies described observable differences in behaviour or language during the clinical assessment which discriminated functional cognitive symptoms from those due to degenerative brain disease (appendix p 14).[13,31,41,165,214–221] People with functional symptoms were reported to be more likely to attend independently, offer detailed descriptions of complaints and personal history, and produce a written list of complaints; they were less likely to exhibit the "head turning sign" (ie, turning towards the caregiver for help with a question) or otherwise rely on an accompanying adult.[13,31,41,214–219,221]

Cognitive symptom profile, metacognition, and illness perceptions

We considered whether any specific cognitive symptoms increased the likelihood of a functional cognitive disorder. Wells[13] reported that patients with pseudodementia reported memory loss for both recent (ie, memories from recent days or weeks) and remote (ie, memories from earlier in life) events (*vs* relative preservation of remote [ie, from much earlier in life] memory preservation in early Alzheimer's disease), memory gaps for specific periods or events, dated symptom onset precisely, and had symptoms of short duration and rapid progression. Ahmed and colleagues[222] reported, in a 2-year longitudinal study, that baseline complaints did not differ between patients who were classified as worried well, patients with amnestic mild cognitive impairment, and patients with semantic dementia. Haussman and colleagues[136] found initial symptoms of attention deficit or word finding impairment more likely in those with subjective memory impairment and normal objective cognition, compared with those with dementia, in whom first symptoms were more likely to be unspecified, memory impairment, or orientation deficit.

The use of symptom checklists was described in two studies of cognitive impairment in depression: Reynolds and colleagues[223] correctly classified 38 (90·5%) of 42 patients (using a seven-item inventory, in which anxiety, delayed insomnia, and loss of libido supported pseudodementia diagnosis) and Yousef and colleagues[224] correctly classified 43 (98%) of 44 patients with dementia and 60 (95%) of 63 patients with depression.

As described, included studies reported poor concordance between cognitive symptoms and measured performance. Metacognition can be defined as the ability to monitor and evaluate one's own thinking; discordance between memory self-report and performance might therefore represent metacognitive error.

A small number of studies examined metacognitions in those with functional or subjective cognitive symptoms. Two studies of functional memory disorders found poorer memory self-efficacy (evaluation of one's own ability) in patients compared with healthy controls.[225,226] Larner[42] found memory self-rating of poor or fair to be sensitive (correctly identified 87% [61/70]), but not specific, with some patients with dementia also reporting poor or fair memory (5-point Likert scale). Elsey and colleagues[216] reported that those with functional memory disorder were more concerned about memory symptoms than were their companions. Mogle and colleagues[227] found higher memory ratings compared with others the same age to be associated with better psychological wellbeing. Chin and colleagues[197] reported that, in those with normal cognitive testing, subjective memory symptom severity was associated with increased self-focused attention.

Three studies suggested that illness perceptions influence symptom severity. Negative ageing stereotypes were associated with more subjective memory complaints (and depressive symptoms), whereas factors contributing to meaning in life were associated with fewer complaints.[130,196] The effect of knowledge of genetic risk was explored by Lineweaver and colleagues:[228] participants informed of their *APOE* ε4 positive status rated their memory worse and did worse than those who were unaware that they were *APOE* ε4 positive. Hurt and colleagues[134] reported that helplessness, illness identity, serious perceived consequences, emotional representation, and negative comparison with peers were strong determinants of distress and anxiety in adults with subjective memory complaints.

Non-cognitive symptom profile

We examined reported associations between functional cognitive disorders and non-cognitive symptoms to consider whether a distinct phenotype could be defined in those with primary cognitive symptoms, having

excluded studies of those with primary (ie, non-cognitive) functional neurological disorder, chronic fatigue syndrome, fibromyalgia, major psychiatric disorder other than depressive or anxiety disorders, or cognitive symptoms after physical illness or injury.

The most striking association was between depressive symptoms and cognitive symptom severity, in both clinical and community populations; anxiety symptoms and personality traits (particularly neuroticism) were also frequent associations (appendix pp 15–18).[12,13,69,123,130–133,135,164,165,173,180,184,186,192,194,195,197,198,202–204,207,208,225,226,229–251] Depressive symptoms were in some studies associated with objective cognitive impairment.[195,204,232,240,252]

Kawagoe and colleagues[152] reported a positive association between apathy scores and cognitive symptom severity scores. Cognitive symptoms were also reported to be associated with self-reported multimorbidity,[253] physical health complaints,[233] additional pain and analgesia use,[114,254] and psychosomatic complaints as measured by symptom checklist 90 (known as SCL-90).[225,226] Five studies reported an association between functional or subjective cognitive symptoms and reported stress.[180,198,225,226,255] Three studies reported an association between severity of subjective memory complaint and general measures of poor psychological wellbeing,[116,186,227,253] two with poorer quality of life than those with less severe memory complaint,[188,201] and one qualitative study reported that presence of subjective memory symptoms had a variable impact on wellbeing.[256]

Nine studies described sleep disturbance in association with functional cognitive symptoms. Reynolds and colleagues[244] described more delayed insomnia, longer recording periods, early-morning waking, and higher rapid eye movement intensity in those with depressive pseudodementia compared with those with dementia.[223,244] Self-report of poor-quality sleep was associated with symptoms in functional cognitive disorder, subjective memory complaint without objective impairment or biomarkers for Alzheimer's disease, patients without dementia in memory clinics, and in population cohorts with subjective memory complaint or perceived decline.[32,39,252,255,257,258] However, in 181 adults (mean age 74 years), sleep actigraphy showed less sleep disruption in those with higher, compared with lower, complaint of subjective memory decline; the authors suggested a "non-linear trajectory between sleep and memory decline in aging".[259] An alternative explanation supported by the other studies identified would be that although sleep is more measurably disordered in degenerative brain disease than in functional cognitive disorders, greater experience of disturbed sleep in those with functional cognitive disorders than those with degenerative brain disease reflects differences in self-monitoring and expectation.

Age and family history

Age is the most important risk factor for degenerative brain disease. If subjective cognitive symptoms were most often prodromal, a close relationship between symptom prevalence and advancing age would be expected, but this correlation was not confirmed by the included studies as a whole. Rowell and colleagues[242] reported that prevalence of subjective memory complaint was similar across all age groups (18–99 years old) in 3798 individuals.

Derouesne and colleagues[234] reported that of those self-referring to a memory clinic, younger patients (range 20–49 years; mean age 39 years) rated their symptoms as major and of longer duration than older patients (range 50–85 years; mean age 61 years). Apolinario and colleagues[239] similarly reported that younger patients (from an older cohort; mean age 74 years [SD 7·2]) reported a higher number of complaints than older patients. Sinforiani and colleagues[236] and Gallassi and colleagues[260] both reported that symptomatic patients without impairment tended to be younger (mean age 68·6 years [SD 8·5] for symptomatic patients without cognitive impairment *vs* 73·5 years [7·0] for patients with mild cognitive impairment;[236] 63·3 years [11·17] for patients with subjective cognitive complaints and no cognitive impairments *vs* 71·06 years [8·07] for patients with subjective complaints and mild cognitive impairment[260]), whereas Arbabi and colleagues[240] found no difference in age between impaired and unimpaired subjective memory complaint.

Family history is a risk factor for degenerative brain disease but experience of dementia in the family might also influence self-evaluation and help-seeking. Four studies examined memory symptoms in relation to family history of dementia: in a study by McPherson and colleagues[261] symptom report was similar overall, but relatives of people with early-onset Alzheimer's disease reported worse memory than controls, correlating with impairment; in a study by La Rue and colleagues[262] relatives had more memory complaints and depressive symptoms than controls, explained as a possible mediator of slightly poorer performance; Cutler and colleagues[263] found that although relatives were more concerned about developing Alzheimer's disease than controls, this concern was not reflected by memory self-ratings. Arbabi and colleagues[240] found no difference in family history between impaired and unimpaired patients with subjective memory complaint. Bharambe and colleagues[43] found higher rates of family history of dementia in patients attending a memory clinic with functional cognitive disorder than in patients with degenerative brain disease attending the same clinic.

Haussman and colleagues[161] reported more subjective impairment in healthy adults with family history than in those without, an association not present in the mild cognitive impairment group, and Hill and colleagues[256] reported that equivalent levels of subjective memory impairment had a greater effect on emotional wellbeing in those with personal experience with dementia than in those without.

Discussion

Cognitive symptoms are common: according to this Review they are present in around a third of the population, with no clear relation to age. This finding alone confirms that not all cognitive symptoms are caused by degenerative brain disease. In studies of people presenting to memory clinics, we found that only 53% (6285 of 11807) received dementia diagnoses, and in studies including adequate description of diagnoses, 24% (2832 of 12003) were described as having subjective cognitive impairment (with or without primary psychiatric disorder), pseudodementia, functional cognitive disorder, or a primary psychiatric disorder, and not degenerative brain disease or other medical cause. Many of these individuals could probably be described as having functional cognitive disorders.

A striking number of terms used to denote cognitive symptoms in the studies included here denoted only a few concepts: cognitive complaints without presumption of cause (eg, subjective memory complaints), perceived cognitive impairment in the absence of measured impairment or disease (eg, worried well or clinically or cognitively normal), progressive symptoms (eg, subjective cognitive decline), and symptoms with positive evidence of non-degenerative cause (eg, functional cognitive disorder or depressive pseudodementia).

As terminology varies, so do methods used to ascertain presence and severity of subjective cognitive symptoms: a substantial limitation of this body of research is that even of those studies using the same terminology, few used the same measure, and even those using a single question address such a variety of aspects (eg, perceived memory decline, poor memory compared with others, worry about memory, and having a poor memory; appendix pp 7–11) that it seems unlikely that different studies are describing similar subjective experiences. Historical use of terms such as pseudodementia introduces additional confusion, with these having been used to describe a wide range of clinical syndromes and causes.

The concepts implied by these terms are important. Authors of studies of subjective cognitive decline and mild cognitive impairment tend to view these states as steps on a trajectory towards dementia, paying less attention to possible alternative causes. A dominant linear trajectory (ie, subjective cognitive decline to mild cognitive impairment to dementia) is not supported by this Review or by other, more comprehensive analyses of outcome, which instead suggest multiple overlapping symptom trajectories (figure 4).

In a review by Jonkers and colleagues[264] of the relationship between memory complaints and dementia, complaints in the younger so-called elderly people were most often related to depression, anxiety, or personality factors, predicting dementia only in a small subset. The description by Reisberg and colleagues[265] of a robustly identifiable clinical entity lasting 15 years before progressing to mild cognitive impairment is at odds with the observed frequency of cognitive complaints in the general population and absence of excess mortality.[67,68,265] A systematic review reported increased risk of incident dementia per year in subjective cognitive impairment, but 86% of patients who were followed up beyond 4 years did not progress to dementia.[151] Although not explored here, the prevalence of functional cognitive disorders in individuals meeting the criteria for mild cognitive impairment will be an interesting topic for future research: meta-analysis of mild cognitive impairment progression in 41 cohort studies found that most individuals with mild cognitive impairment did not progress to dementia even after 10 years.[266]

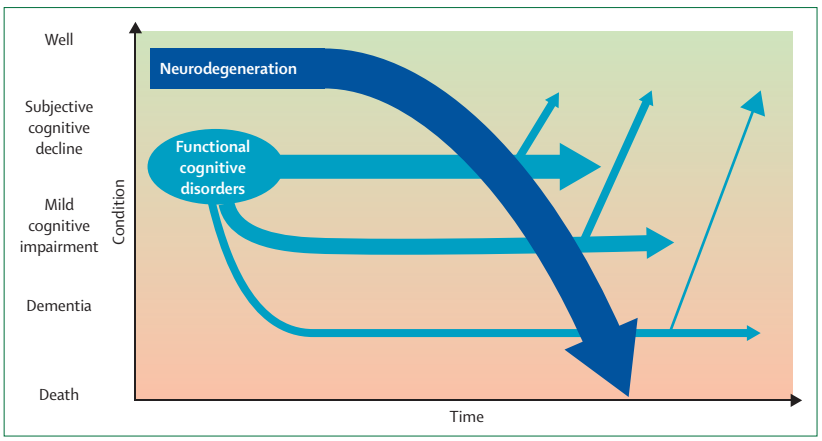

Figure 4: **Degenerative brain disease and functional cognitive disorder trajectories**

With the ongoing dominance of the subjective cognitive decline to mild cognitive impairment to dementia model, assumptions of cause have become attached to descriptive terms, restricting the range of interpretation of research findings. As examples, researchers finding an inverse relation between measured sleep quality and severity of subjective cognitive decline suggest a non-linear trajectory between sleep and memory decline, and researchers finding opposite patterns of resting state functional MRI in subjective cognitive decline to those seen in Alzheimer's disease hypothesise that these patterns are compensatory responses to neurodegeneration: neither group considering that their findings might indicate functional, rather than Alzheimer's disease, pathology.[152,167,259]

Although mild cognitive impairment is outside of the scope of this Review, efforts to make results fit with the subjective cognitive decline to mild cognitive impairment to dementia model can be seen in studies of biomarkers for Alzheimer's disease in individuals with mild cognitive impairment, where profiles indicating a mildly increased risk of progression to dementia are often described in authors' conclusions as predictive, with little discussion of the frequency or clinical significance of false positives, when meta-analyses of the same biomarkers report poor accuracy.[267–271] For example, review of ^{11}C-labelled PIB-PET as a predictor of mild cognitive impairment conversion to dementia reported test specificity of between 46% and

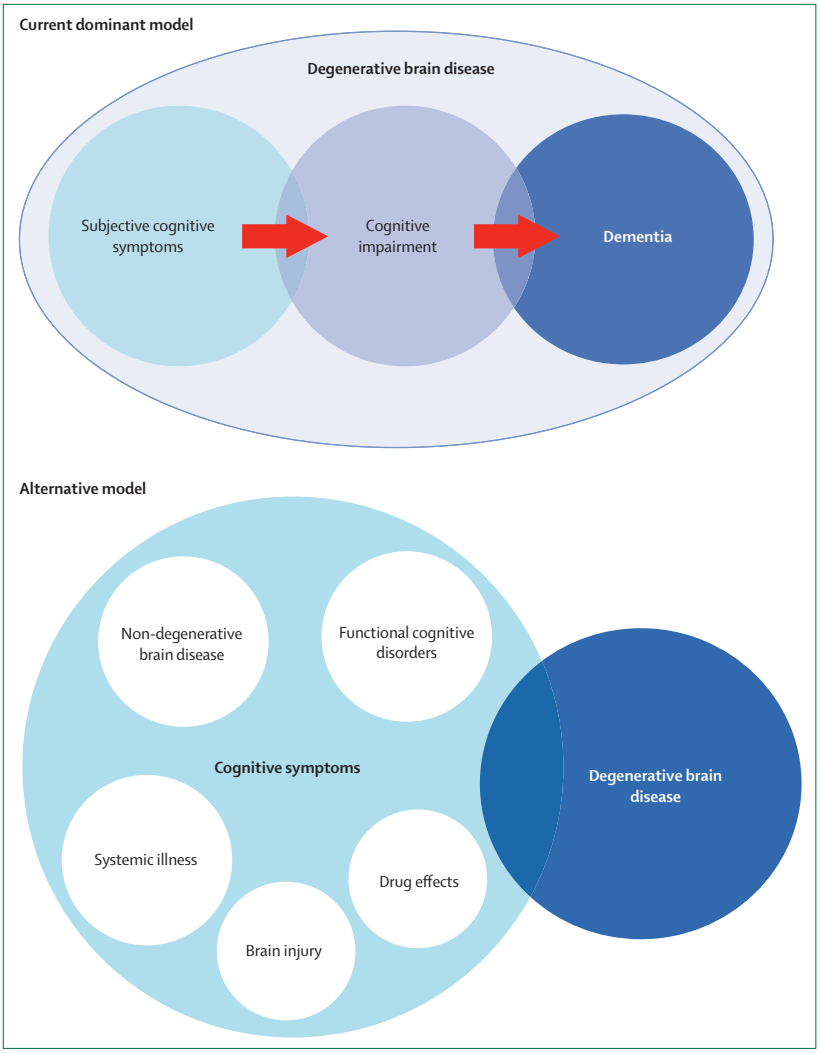

Figure 5: **An alternative model for the cognitive symptoms and degenerative brain disease relationship**
The current dominant model (A) places subjective cognitive symptoms within the realm of degenerative brain disease and at the start of a linear trajectory towards dementia. An alternative model (B) acknowledges that cognitive symptoms have multiple causes, including functional cognitive disorders, and only a minority of cognitive symptoms are the result of degenerative brain disease (and many individuals with degenerative brain disease do not complain of symptoms). Identifying positive diagnostic profiles for functional cognitive disorders might improve accuracy of diagnoses of clinical prodromes of degenerative brain disease.

Functional cognitive disorders	Degenerative brain disease
Attends alone	Attends with someone
Patient more aware of the problem than others	Others more aware of the problem than patient
Answers independently	Turns to accompanying adult for support in answering questions (eg, head turn sign)
Gives a detailed description of complaints (might bring written list)	Absence of details when describing symptoms
Frequently offers elaboration and detail	Unlikely to give spontaneous elaboration or detail
Can answer questions with multiple components	Can only answer single-component questions
Detailed account of personal history, drugs, and previous interactions with doctors	Less detailed account
Loss of recent and remote autobiographical memories	Relative preservation of remote autobiographical memories
Complaint of memory gaps for specific periods and events	Complaint of specific memory gaps unusual
Memory symptoms might be within most people's normal experience	Memory symptoms are often outside normal experience
Approximate answers (ie, answers that indicate that the correct answer is known at some level)	No approximate answers
Dates symptom onset with precision	Difficult to pinpoint date of onset
Unstable longitudinal course	Cognitive impairment progressive over time
Marked variability	Less variability

These clinical features are often observed in functional cognitive disorder and are candidate positive features of functional cognitive disorder that merit prospective examination in mixed clinical cohorts.

Table: **Clinical features more common in functional cognitive disorders versus features more common in degenerative brain disease (adapted from Griem and colleagues[3] and Stone and colleagues[276])**

88%, estimating that for every 100 PIB scans in people with mild cognitive impairment, 28 people with a positive scan would not progress to Alzheimer's dementia.

This problem is not only theoretical. Reliance on biomarker investigations without a keen awareness of the substantial false positive rate risks iatrogenic harm through misdiagnoses; this possibility is shown by an included study in which CSF biomarkers did not improve clinicians' prognostic accuracy but resulted in false positive predictions of future decline in patients with subjective complaints.[182]

A small proportion of those with subjective cognitive symptoms progress to dementia, and this progression is more likely with new and progressive symptoms, cognitive impairment (particularly of an amnestic nature), a degenerative brain disease biomarker profile, or a depressive pseudodementia picture.[272] For some individuals with dementia, identification of a period of prodromal subjective symptoms retrospectively is possible, and (particularly in not-Alzheimer's disease syndromes) this period might last several years before onset of dementia.

Moreover, demonstrably functional cognitive symptoms might result from metacognitive impairment or psychiatric disorder occurring in prodromal Parkinson's disease, Lewy Body dementia, or frontotemporal dementia, just as functional motor symptoms have been reported in the prodrome of Parkinson's disease.[273] This area of overlap and comorbidity will be an important area for future research. However, only a minority of subjective cognitive symptoms progress to dementia, and we suggest that this is in part because many of those with subjective cognitive symptoms have functional cognitive disorders (figure 5).

Clinically, functional cognitive disorders are, if not exactly under-recognised, considered not to be the primary business of the memory clinic. Functional cognitive

disorders are infrequently discussed and rarely investigated in dementia research despite probable ubiquity in midlife and preclinical cohorts, and little evidence exists to guide diagnosis and treatment. The harm associated with an incorrect clinical prediction of dementia cannot be underestimated. Importantly, identifying positive diagnostic profiles for functional cognitive disorders will improve accuracy of early degenerative brain disease diagnoses, so that only those most likely to be on a trajectory towards dementia are included in trials in which relevant causes of degenerative brain disease are a prerequisite for target engagement and amelioration of the disease course.

The diverse studies included in this Review show a broad functional cognitive disorder phenotype. Depressive symptoms are the most common clinical associations, in alignment with other reviews of subjective memory symptoms.[274,275] Metacognitive error, present in most populations, was most marked in those with functional cognitive disorders, who substantially overestimated their deficits. Anxiety, neuroticism, negative self-beliefs, increased self-focused attention, and negative views of ageing are associated with more frequent and severe cognitive complaints. Distinctive patterns of behaviour and language during the clinical assessment (table)[3,10,276] are strong candidate positive diagnostic signs for functional cognitive disorders.

Our findings are consistent with the causative framework proposed in the review by Teodoro and colleagues[4] of cognitive symptoms in functional neurological disorders, fibromyalgia, and chronic fatigue syndrome, which excluded pure cognitive presentations: excessive self-attention and metacognitive error (supported by negative illness beliefs) lead to heightened experience of cognitive failure, effort, and illness (exacerbated by depressive symptoms, anxiety, and neuroticism), and the resulting inattention and cognitive failures maintain the cycle.

An inevitable limitation of this Review results from difficulty in aligning results of studies with widely varying terminology and symptom assessment methods: our analyses of prevalence rates can be considered broadly indicative rather than precise. Our attempt to define and identify functional cognitive disorders from within the wider cognitive disorder is a necessary preliminary to future research. From here, prospective studies will be important to provide evidence for the utility of specific clinical features in making a positive (rather than by-exclusion) diagnosis, to define populations for much needed trials of treatment, reduce iatrogenic harm, and improve accuracy of early degenerative brain disease diagnoses.

Contributors

LM did the literature search, data extraction, and writing. AC, JS, and CR contributed equally to the study design, and to subsequent review and revision of the manuscript.

Declaration of interests

LM provides independent medical testimony in court cases regarding patients with functional disorders. AC 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 and 50% defender basis, is an associate editor of the *Journal of Neurology Neurosurgery and Psychiatry*, and is the treasurer of the International Functional Neurological Disorder Society. JS reports personal fees from UptoDate, outside the submitted work, runs a self-help website for patients with functional neurological symptoms that 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. CR declares no conflicts of interest.

For more on the **self-help website** see www.neurosymptoms.org

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