

Mild behavioral impairment is related to frailty in non-dementia older adults: a cross-sectional study

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Abstract

Background: Frailty and cognitive decline are highly prevalent among older adults. However, the relationship between frailty and mild behavioral impairment (MBI), a dementia risk syndrome characterized by later-life emergence of persistent neuropsychiatric symptoms, has yet to be elucidated. We aimed to evaluate the associations between MBI and frailty in older adults without dementia.

Methods: In this cross-sectional study, a consecutive series of 137 older adults without dementia in the Anti-Aging Study, recruited from primary care clinics, were enrolled. Frailty was estimated using the Fried phenotype. MBI was evaluated by the Mild Behavioral Impairment Checklist (MBI-C) at a cut-off point of >8. Cognition was assessed with the Chinese versions of the Montreal Cognitive Assessment (MoCA-BC) and Mini-mental State Examination (MMSE). Multivariable logistic regression was performed to estimate the relationship between MBI and objective cognition with frailty status.

Results: At baseline, 30.7% of the older adults had frailty and 18.2% had MBI (MBI+ status). Multivariable logistic regression analysis demonstrated that compared to those without MBI (MBI- status), MBI+ was more likely to have frailty (odds ratio [OR] = 7.44, 95% CI = 1.49-37.21, $p = 0.02$). Frailty and MBI were both significantly associated with both MMSE and MoCA-BC score ($p < 0.05$).

Conclusions: Both frailty and MBI status were associated with higher odds of cognitive impairment. MBI was significantly associated with an increased risk of having frailty in the absence of dementia. This association merits further study to identify potential strategies for the early detection, prevention and therapeutic intervention of frailty.

Background

Frailty is a common geriatric condition presenting as a clinical state of decreased physiological reserve, increased vulnerability to death and increased susceptibility to even small stressors(1). It is associated with an increased risk of adverse health-related outcomes, including falls, disability and mortality(2). The prevalence of frailty is 3.9% to 51.4% among community-dwelling people aged 60 years and older, and the incidence increases with age(3). As population aging has become a global phenomenon, frailty has become an emerging public health issue. To date, most definitions have prioritized the physical dimension of frailty, which includes symptoms and signs such as weight loss, muscle weakness, slower gait speed, and sedentary behavior(4). Frailty has been most commonly operationalized using a phenotypic approach or a deficit accumulation approach(5, 6). In research, a commonly used approach to capture frailty is the Fried phenotype, which has been extensively tested for its validity(7, 8).

Frailty that combines a range of diverse deficits is increasingly recognized as a fundamental determinant of an individual's vulnerability or resilience to stressors(9) and has been linked to impaired cognition(10, 11). Cognitive impairment has been shown to improve the predictive value of frailty, measured using the Fried phenotype, for adverse health outcomes(11). Various neurocognitive disorders, including late-life cognitive impairment(12, 13), mild cognitive impairment (MCI)(14), dementia(15) and Alzheimer's disease

(AD)(16, 17), have shown associations with frailty. Indeed, frailty moderates the association between AD pathology and the clinical expression of dementia, such that in the presence of frailty, even low AD pathological burden may manifest as dementia(17). Researchers have also found that frailty and cognitive decline might share common physiological mechanisms, with greater frailty being associated with worse cognition and a faster rate of cognitive decline(18). Thus, associations between frailty and other risk markers for cognitive decline are warranted.

Similar to frailty, neuropsychiatric symptoms (NPS) have demonstrated associations with cognitive decline and have been linked to known dementia biomarkers, thus also suggesting common underlying mechanisms. The Mayo Clinic Study of Aging reported that the presence of NPS (particularly agitation, apathy, anxiety, irritability or depression) was associated with an increased risk of developing MCI in cognitively normal older adults(19). More recent evidence from a large sample in the National Alzheimer Coordinating Center dataset demonstrated that in 59% of dementia cases, NPS emerged in advance of cognitive symptoms, including 30% of people who developed AD, reinforcing the notion that later-life onset of NPS can be an early marker of dementia(20). To operationalize the assessment of NPS as risk markers for dementia, the International Society to Advance Alzheimer's Research and Treatment developed criteria for mild behavioral impairment (MBI)(21), which is a neurobehavioral syndrome characterized by later-life emergent NPS as an at-risk state for incident cognitive decline and dementia. Although MBI and MCI can co-occur, MBI can also precede MCI, manifesting in older adults with subjective cognitive decline or even normal cognition, in whom MBI has demonstrated an increased risk of cognitive decline and dementia(22-26). MBI may be the initial manifestation of neurodegeneration for some, and has been connected with known biomarkers for dementia including amyloid beta(27), tau(28, 29), neurofilament light(30), cortical atrophy(31, 32), white matter atrophy(33), and AD risk genes(34, 35). MBI has also been used in machine learning models to predict neurocognitive diagnostic category 40 months later(36). These findings suggested that the early recognition of the NPS that constitute MBI may contribute to earlier detection of neurodegeneration, and may represent a clinical entity and premorbid treatment target to explore for intervention strategies to prevent or delay the onset of dementia(37). The Mild behavioral impairment Checklist (MBI-C) is the validated brief screening instrument developed to capture MBI in accordance with the criteria(38-42).

Frailty, as a substantial moderator in the clinical expression of dementia, could be a predictor of cognitive decline over time(43-45). However, the association between frailty and cognition in pre-dementia has yielded mixed results(46-48). MBI is associated with a significantly faster rate of cognitive decline and progression along the continuum of neurodegenerative pathology compared to late life psychiatric disorders, and compared to those without MBI. Thus predictive value of MBI appears to be early in the neuropathological course of disease, in advance of cognitive impairment for some(22).

Identifying at-risk populations is an important public health issue, in order to explore risk reduction. The possible association between MBI and frailty, both independent risk factors for dementia appearing early in the disease course, should also be further investigated. In this cross-sectional study, we aimed to: 1) determine the prevalence of frailty and of MBI; 2) replicate prior findings linking frailty to worse objective

global cognition; 3) determine the association between MBI and global cognition; and 4) assess the relationships between MBI total and domain scores, and frailty, in a primary care sample of older adults with at most mild cognitive impairment. We hypothesized that MBI would predict greater frailty burden.

Methods

Participants and Setting

A consecutive series of 185 volunteers aged 60 or older were recruited from the Anti-Aging Study, which investigates health and frailty. Participants were recruited from advertisements in GPs clinics and Medical Management Centers in Guangzhou (the capital of the Guangdong, South-East of China). Inclusion criteria were: 1) aged 60 or older; 2) ability to speak Chinese; 3) having adequate auditory and visual acuity; and 4) ability to provide informed consent to participate in the study. At eligibility assessment, participants underwent a detailed medical history, record review, and neuropsychological assessment. Exclusion criteria included the following: 1) history of neurological and psychiatric diseases (cerebrovascular disease, Parkinson's disease or dementia); 2) head injury with loss of consciousness longer than 5 minutes; and 3) a systemic or terminal illness affecting follow-up participation. At enrollment, participants completed a comprehensive evaluation including but not limited to a physical examination, frailty assessment, medical record and medication review, clinical interview with questionnaires, emotional assessment and a neuropsychological assessment. Participants were excluded if they had a cognitive score consistent with dementia, defined as a Mini Mental State Examination (MMSE) cut-off score ≥ 24 (49, 50). Participants were also excluded if they were missing covariate data (Figure 1 lists exclusion details).

Sociodemographic and Clinical Characteristics

The sociodemographic characteristics analyzed were age, sex and education. Nutritional status was measured and classified based on body mass index (BMI), calculated as weight in kilograms divided by height in meters squared (kg/m^2) (51). To identify polypharmacy and multimorbidity, the participants were asked if they had a physician-determined diagnosis of heart disease, hypertension, stroke, diabetes, cancer, rheumatic disease, lung disease, osteoporosis, neurologic disease, urinary incontinence or fecal incontinence. Polypharmacy has generally been defined as concurrent administration of more than 5 medications and multimorbidity as the presence of >2 chronic diseases (52).

Emotional Assessment

Anxiety and depression were measured by the Generalized Anxiety Disorder 7-item (GAD-7) scale (53) and the 9-item Patient Health Questionnaire (PHQ-9) (54), respectively. The GAD-7 measures the frequency of each anxious mood item from never (0) to nearly every day (3). A total score of ≥ 10 indicates the presence of an anxiety symptomatology (55). The PHQ-9 measures the frequency of each depressed mood item from not at all (0) to nearly every day (3). The standard cut-off score of 10 or greater maximized the combined sensitivity and specificity in the primary studies (56). We used a GAD-7 score of

≥ 10 and a PHQ-9 score of ≥ 10 as the cut-off points for indicating clinically significant anxiety and depression, respectively(57).

Frailty Assessment

The diagnosis of frailty was based on the Fried phenotype and included five indicators: exhaustion, which was measured by self-report questionnaire based on two items extracted from the Center for Epidemiology Studies Depression (CES-D)(58); unintentional weight loss (≥ 10 pounds or $\geq 5\%$ of body weight in last year); weak grip strength measured by the grip-strength of the dominant hand using a hand grip dynamometer and defined based on established cutoffs by gender and body mass index (BMI); slow walking speed (speeds below an established cutoffs adjusted for sex and height), which was directly measured by walking time of 15 feet; and low energy expenditure (low physical activity in the past two weeks, adjusted for sex), which was estimated using the Minnesota Leisure Time Physical Activity (MLTA) questionnaire(4). Based on these scores, individuals with 0-2 criteria present were categorized into the no-frailty group and those with 3 or more criteria were in the frailty group(59).

Neuropsychiatric and Neuropsychological Assessment

MBI was assessed using the Chinese version of MBI-C developed by Cui(60), a scale developed specifically for functionally independent community-dwelling older adults. The MBI-C(42) includes 34 items in five domains: 1) decreased motivation (apathy); 2) emotional dysregulation (mood and anxiety symptoms); 3) impulse dyscontrol (agitation, aggression, impulsivity); 4) social inappropriateness (impaired social cognition); and 5) abnormal perception or thought content (psychotic symptoms, i.e. hallucination and delusions). Only symptoms that were characterized by later life onset, representing a change from longstanding patterns of behavior, and that have been present no less than 6 months were assessed as “yes”, and their severity was rated (1 to 3 points)(42). MBI-C domain total scores were calculated by adding the severity scores. MBI+ status was based on a total score > 8 , its optimal cut-off point for MBI case detection in a primary care population, with good sensitivity and specificity(39).

As part of the objective cognitive assessment, the participants completed brief objective cognitive screening tools. The Chinese versions of the Montreal Cognitive Assessment (MoCA-BC)(61) and the Mini-mental State Examination (MMSE)(62) were used to measure global cognitive function. The MoCA test includes more attention-executive items than the MMSE and was included to be sensitive for mid cognitive impairment, whereas the MMSE was used primarily to screen out dementia(63). The MMSE and the MoCA were administered in a random order to avoid a fatigue effect bias. Potential scores range from 0 to 30, with higher values indicating better cognition. As both the MMSE and MoCA include similar items for orientation and calculation, these items were included only in the MMSE.

Statistical Analysis

Continuous variables were reported as the mean \pm standard deviations (SD), and categorical variables were presented as frequency and percentage. Independent samples t-tests were performed to determine

the differences between the frailty and no-frailty groups, with respect to the continuous variables. Chi-square (χ^2) tests were used to identify the group differences for the categorical variables. The total and domain-specific questionnaire scores for the MBI-C were calculated. The distribution of the scores in MBI-C and the prevalence of MBI diagnosis were determined using frequency and descriptive analyses. Logistic regression was used to estimate the odds ratios (ORs) and 95% confidence intervals (CIs) for the association between frailty status with age, education, depression, MBI and objective cognition. All analyses were conducted using the statistical analysis software SPSS version 18.0, and a two-sided *P*-value of 0.05 was set as the level of significance.

Results

Participant Characteristics

There were 137 older adults enrolled in this study; the mean age was 69.6 ± 7.6 years, and the age range was 60 to 90 years old. Among these, 94 (68.6%) were female, 21 (15.3%) had a primary school education or lower, 43 (31.4%) had multimorbidity, the presence of more than 2 comorbid conditions, and 27 (19.7%) had polypharmacy (took five or more oral medications daily). Of these enrolled individuals, the mean BMI was 22.5 ± 3.3 kg/m² and 31 (22.7%) had depression symptoms, while 14 (10.4%) had anxiety symptoms. According to the definition, 42 participants were categorized into the frailty group (30.7%) and 95 into the no-frailty group (69.3%). The frailty group showed worse performance on the MMSE (26.7 vs 28.0, $p < 0.05$) and MoCA (25.2 vs 26.1, $p < 0.05$) scores than the no-frailty group. The two groups also presented significant differences in age, education, comorbid conditions >2 , polypharmacy and depression. No significant differences were found between the two groups with respect to sex, BMI and anxiety symptoms (Table 1).

A total of 25 (18.2%) participants were MBI+, and 112 (81.8%) were MBI-. Regarding group composition, the mean age of MBI+ participants (72.2 ± 7.7) was higher than that of MBI- participants (69.5 ± 7.0) ($p < 0.05$). The MBI+ individuals had significantly poorer cognition, with lower MMSE (26.8 vs 27.8, $p < 0.05$) and MoCA (24.7 vs 26.1, $p < 0.05$) scores than the MBI- individuals. No significant differences were found between the MBI+ individuals and MBI- individuals in terms of sex, education level, BMI, comorbid conditions >2 , polypharmacy, or depression and anxiety symptoms ($p > 0.05$) (Table 1).

Frailty and Mild Behavioral Impairment

MBI status was significantly different between participants with and without frailty ($p = 0.038$). The MBI-C composite score was associated with frailty ($p = 0.001$). Of the five MBI domains, participants with decreased motivation, affective dysregulation and social inappropriateness MBI domains were more likely to have frailty. Neither impulse dyscontrol nor psychosis differed between frailty groups, although impulse dyscontrol neared statistical significance ($p=0.059$) were found in our study (Table 2).

Multivariable logistic regression analysis indicated that MBI+ status was significantly associated with higher risk of having frailty, with an OR of 3.09 (95% CI = 1.29-9.41; $p = 0.047$) (Table 3). We also

evaluated the associations between frailty status and global cognition, depression, education and age; we found that age and depression were significantly related to a higher risk of having frailty ($p < 0.05$), but the association with education, MMSE and MoCA score was not significant ($p > 0.05$) (Table 3).

Discussion

To our knowledge, this is the first cross-sectional study to evaluate the relationships between frailty, MBI, and cognition. First, we determined that frailty is common in this population, with a prevalence of 30.7%. Second, MBI was also fairly common, with a prevalence of 18.2%. Third, greater burden of frailty was associated with poorer cognition, measured using the MMSE ($p=.01$) and MoCA ($p=.04$). Fourth, compared to those without MBI, MBI+ status was associated with poorer cognition measured using the MMSE ($p=.049$) and MoCA ($p=.01$). Fifth, MBI+ status predicted higher levels of frailty (OR=3.09; 95% CI=1.29-9.41), and this signal was driven by the MBI domains of decreased motivation, affective/emotional dysregulation, and social inappropriateness ($p<0.05$). These results suggest that in non-demented older adults, frailty and MBI are both common and associated with small but significant impairment in global cognition.

The prevalence of frailty was 30.7% in our study, which was relatively high compared with previous estimates, which ranged from 11% up to 26% in community samples(64-66). This difference may be attributed to our study design and to the fact that participants came from primary care clinics. Frailty may increase the risk of future cognitive decline, and that cognitive impairment may increase the risk of frailty, suggesting that cognition and frailty may interact in the cycle of age-related decline(67, 68). Our results indicated that frailty was associated with age-related cognitive decline, describing an at-risk group for the preclinical phase of neurocognitive disorders, consistent with previous studies(11-16). In their seminal study, Solfrizzi and colleagues reported that frail older adults had a higher prevalence of cognitive impairment than those without frailty (77% vs. 54%)(69). Furthermore, components of frailty appeared to be related to pathological findings of AD and vascular dementia, supporting the idea of a possible common biological pathway between frailty and cognitive disorders(70). A previous study found that there was an increase in neurons with cellular senescence and aging of microglia, and therefore, increases in apoptosis, aggregation of protein, and mitochondrial dysfunction, with increased reactive oxygen species, oxidative damage to proteins and lipids, and accumulation of DNA damage(70). Accordingly, increasing frailty may be an indicator of future cognitive impairment.

The prevalence of MBI (18.2%) in our participants was higher than that reported by Creese(22) in the PROTECT study, in which 10% of community-dwelling older adults aged 50 or over ($n = 9,931$) reported MBI, as captured by the MBI-C. In a clinical sample of Spanish primary care patients from which the current cut-points were derived, the prevalence of MBI was 5.8% in older adults with subjective complaints(39) and 14.2% in MCI(71). These estimates collectively, determined using the MBI-C, are considerably lower than previous prevalence estimated generated using the Neuropsychiatric Inventory (72) which ranged from 28-51% in a community population(73, 74), and 49-85% in a cognitive neurology clinic population(73, 75). These differences may be due to the diagnostic frame of reference of one

month of symptoms captured by the Neuropsychiatric Inventory, whereas the MBI-C involves a more rigorous standard of six-month symptom duration and explicit later-life onset of symptoms, in accordance with the MBI criteria. The lower MBI frequency generated using the MBI-C reflects increased diagnostic specificity for MBI, eliminating the inclusion of transient and reactive states, by excluding false positive symptoms.

Neuropsychiatric symptoms are associated with an increased risk of cognitive deficits across the lifespan, and MBI is associated with poorer cognition cross-sectionally(76), as well as longitudinally in comparison to those without MBI(23, 24). In agreement with this previous evidence, we also found subtle but significant differences in global cognition reflected by lower scores on both the MMSE and MoCA in patients with MBI. Indeed, the MBI-C might have significantly higher discriminatory power than the MMSE when seeking to detect older adults with subtle cognitive decline(42). Considering that MBI reflects the neurobehavioral axis of pre-dementia at-risk states and is a complement to the neurocognitive risk axis represented by MCI(31), this complementary approach may increase the yield when using both cognitive and behavioral approaches to screen for early-stage neurocognitive disorders.

In this study, we found that MBI was associated with higher levels of frailty, even after adjustment for potential confounders, and that this signal was driven by the MBI domains of decreased motivation, affective/emotional dysregulation and social inappropriateness. Our findings extend the literature by describing different patterns of association of MBI and its components with frailty, a pattern not previously established. Prior studies exploring the link between frailty and cognition have focused on individual functional abilities and assessed only global cognitive ability or limited cognitive domains(14, 77). The mechanisms for the association are not clear, but possibly involve abnormalities in biological processes related to aging(78). A growing body of epidemiological evidence indicates that the mechanisms involved in the onset of frailty are also those that promote neurodegeneration, including chronic inflammation(67) and oxidative stress(79). Other clinical polypharmacy and multimorbidity can increase the risk of both frailty and dementia(80, 81).

MBI may serve as a proxy marker for frailty, or potentially a risk factor of frailty. Thus, MBI assessment may provide an approach to identify frailty early or to determine the risk of frailty in advance of completing a clinical assessment. This approach identifies potentially novel opportunities to prevent or delay frailty, age-related cognitive decline and other associated adverse health outcomes. The ease of administration of the MBI-C, which has been validated for telephone and online administration with high sensitivity and specificity(38, 39, 76), positions it as a simple and cost-effective tool to be administered remotely or at scale for detecting those at clinical risk, in order to flag them for further assessment and work up.

The limitations of our study include the participant population and the sample size. Lower prevalence of MBI and frailty among participants in communities rather than clinical, hospital, or institutional settings are to be expected, and it is unclear if these results can be generalized. We had a limited sample size in this study, and replication with a larger sample is required. Hence, the clinical utility of the cognitive frailty

construct cannot be unequivocally supported by this study, but it should be further investigated in future studies independently undertaken by other investigators in older populations. The frailty instrument may also present another limitation. Due to the constraints related to time, resources, and space, we chose Fried phenotype, combining five physical and physiological burden items, determined simply and quickly. Additional studies with other multi-dimensional and more elaborate objective assessments, representing as many domains as possible, are needed in order to validate these findings.

Conclusion

In conclusion, our findings provide further evidence that MBI and frailty are common among non-demented older adults, with both reflecting subtle but significant deficits in global cognition. MBI, especially in the domains of decreased motivation, affective dysregulation and social inappropriateness, is significantly associated with an increased risk of frailty in those with at most mild cognitive deficits. The MBI-C used in clinical practice could represent a simple and beneficial instrument for the detection of risk prior to the onset of frailty. Overall, these findings emphasize the importance of assessing physical as well as cognitive and behavioral function in older adults to identify risk. The inclusion of these measures in the assessment of frailty can improve the predictive validity of the phenotype regarding adverse health outcomes, and capture an at-risk group for early intervention.

Abbreviations

MBI: mild behavioral impairment; NPS: neuropsychiatric symptoms; MCI: mild cognitive impairment; AD: Alzheimer's disease; NC: normal cognition; MBI-C: Mild Behavioral Impairment Checklist; FP: the Fried phenotype; MoCA-BC: Chinese versions of the Montreal Cognitive Assessment; MMSE: Mini-mental State Examination; BMI: body mass index; ANOVA: analysis of variance; ORs: odds ratios; CIs: confidence intervals.

Declarations

Ethics approval and consent to participate

This study was approved by the Ethics Review Board of Guangdong Provincial Hospital of Chinese Medicine Ethics Committee (reference: B2017-168-01) and all the participants provided written informed consent. For some participants recognized as having cognitive impairment and/or severe illness, we obtained proxy consent from a family member or another supportive adult on their behalf.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

SF, ZY and FX conceived and designed the study. SF, XL and TY recruited the participants, collected the data for the manuscript and provided substantial feedback. SF, ZP, ZI and BH analyzed and interpreted the data. SF, ZI, ZY and FX wrote the first draft of the manuscript. All authors read and approved the final manuscript.

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Tables

Table 1 Characteristics of 137 Participants Aged ≥ 60 Years and Stratified by MBI Status and Frailty Status

Variable †	Full Sample (n=137)	MBI Status			Frailty Status		
		MBI- (n=112)	MBI+ (n=25)	P- value	No-frailty (n=95)	Frailty (n=42)	P- value
Age, mean (SD)	69.6 (7.6)	69.0 (7.5)	72.2 (7.7)	.05	67.9(6.9)	73.2 (8.1)	<.001
Female	94 (68.6)	86(76.8)	18 (72.0)	.61	58 (61.1)	36 (85.7)	.07
Education				.34			.01
Primary or lower	21 (15.3)	15 (13.4)	6 (24.0)		10 (10.5)	11 (26.2)	
Completed high school	75 (54.7)	64 (57.1)	11 (44.0)		50 (52.6)	25 (59.5)	
At least some college	41 (29.9)	33 (29.5)	8 (32.0)		35 (36.8)	6 (14.3)	
Comorbid conditions >2	43 (31.4)	37 (33.0)	13 (52.0)	.08	27 (28.4)	23 (54.8)	.01
Polypharmacy	27 (19.7)	22 (19.6)	5 (20.0)	.97	9 (9.5)	18 (42.9)	<.001
BMI, mean (SD)	22.5 (3.3)	22.5 (3.2)	22.7 (3.9)	.75	22.8 (2.8)	21.9 (4.1)	.14
Depression (PHQ-9≥10)	31 (22.7)	26 (23.2)	5 (20.0)	.73	16 (16.8)	15(35.7)	.02
Anxiety (GAD- 7≥10)	14 (10.2)	12 (10.7)	2 (8.0)	.69	8 (8.4)	6 (14.3)	.30
MMSE, mean (SD)	27.6 (2.4)	27.8 (2.3)	26.8 (2.9)	.049	28.0 (1.8)	26.7 (3.4)	.01
MoCA, mean (SD)	25.8 (2.5)	26.1 (2.2)	24.7 (3.2)	.009	26.1 (2.2)	25.2 (2.9)	.04

Notes: SD: standard deviation; MBI: mild behavioral impairment; MBI-C: Mild Behavioral Impairment Checklist; BMI: body mass index; Results presented as n (%) unless otherwise noted.. Chi-square tests were used for categorical variables, whereas t-tests were used for continuous variables.

Table 2 Frailty and Cognitive and Behavioural Characteristics

	Frailty (n=42)	No-frailty (n=95)	χ^2/F value	p value
MBI, n (%)	12 (28.6)	13 (13.7)	$\chi^2= 4.3$.038
MBI score, mean (SD)	7.3 (5.2)	4.7 (3.6)	F= 5.8	.001
Decreased motivation	2.2 (2.2)	1.3 (1.2)	F= 13.3	.005
Affective dysregulation	1.8 (1.4)	1.3 (1.1)	F= .6	.028
Impulse dyscontrol	2.3 (2.0)	1.6 (1.9)	F= 1.3	.059
Social inappropriateness	0.7 (1.0)	0.4 (.7)	F= 12.5	.041
Psychosis	0.3 (.7)	0.2 (.5)	F= 5.0	.246

MBI: mild behavioral impairment; MBI-C: Mild Behavioral Impairment Checklist.

Table 3. Multivariable logistic regression analysis for the association between frailty status and objective cognition with mild behavioral impairment

	Frailty Status				
	β	Sr	Wal χ^2	p value	Odds ratio (95% CI)
Age	-.09	.04	5.28	.022	.91 (.84-.99)
Education	-.69	1.10	.40	.529	.50 (.06-4.3)
Depression	1.62	.51	10.27	.001	5.04 (1.88-13.58)
MoCA	-.13	.15	.73	.392	.88 (.66-1.18)
MMSE	.18	.16	1.21	.272	1.19 (.87-1.64)
MBI	1.13	.57	3.95	.047	3.09 (1.29-9.41)

Abbreviations: CI: confidence intervals

Figures

Figures

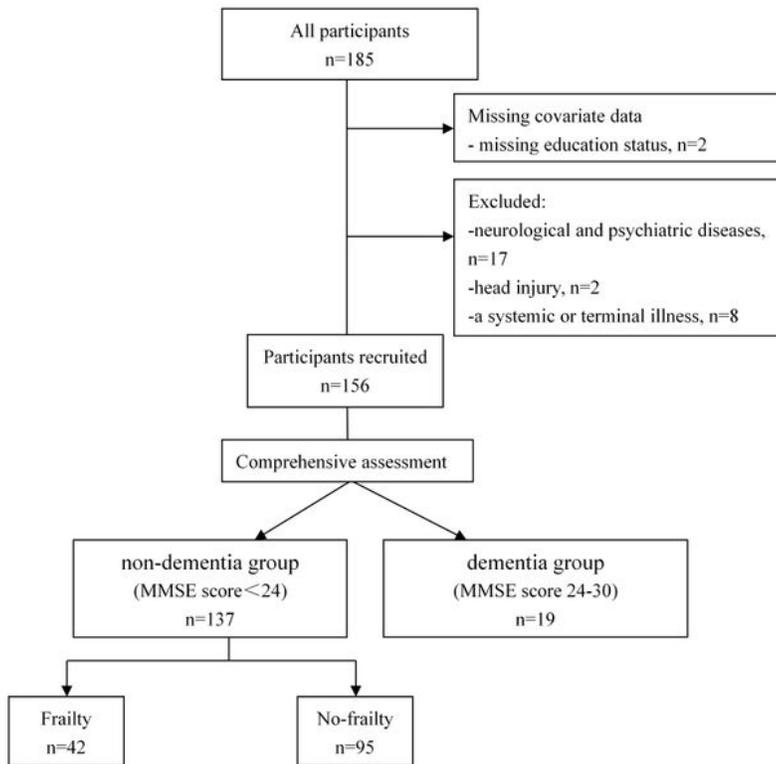


Figure 1 Participant flow chart

Participant inclusion/exclusion criteria. Missing data categories are mutually exclusive. *One participant with missing frailty component (gait speed) and frailty categorical definition not possible.

1/1

Figure 1

Participant flow chart. Participant inclusion/exclusion criteria. Missing data categories are mutually exclusive.