Long-term benzodiazepine use is associated with poorer cognitive function in schizophrenia: Findings from the SALT-C cohort

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Abstract

Background

The association between long-term use of benzodiazepines (BDZs) and cognitive function in patients with schizophrenia has not been fully characterized. This study aimed to investigate the relationship between long-term BDZs use and cognitive impairment in schizophrenia.

Methods

Data were derived from an observational study of treatment with atypical antipsychotics in Chinese patients with schizophrenia (SALT-C). Fifty-seven patients with long-term use of BDZs (≥ 60 days) were included, and 57 BDZs non-users were matched using propensity scores for age, sex, disease duration, and atypical antipsychotics received. Cognitive impairment was assessed using the Montreal Cognitive Assessment (MoCA); psychotic symptoms, illness severity, and psychosocial functioning were assessed with the Positive and Negative Syndrome Scale (PANSS), the Clinical Global Impression-Severity (CGI-S) scale, and the Personal and Social Performance (PSP) scale. Between-group comparisons were performed using independent-samples t-tests or Mann–Whitney U tests. Multivariate analysis was performed using binary logistic regression.

Results

The long-term BDZs user group showed a significantly lower total MoCA score [16.02 (6.69)] than the BDZs non-user group [19.33 (7.11)] (P = 0.012). No significant between-group differences were observed in total scores on the PANSS, CGI-S, or PSP (all P > 0.05). Long-term BDZs use was independently associated with cognitive impairment (OR = 7.728, P = 0.017), whereas years of education was negatively associated with cognitive impairment (OR = 0.740, P = 0.007).

Conclusions

Long-term BDZs use is associated with poorer cognitive performance in patients with schizophrenia, suggesting that cognitive function should be monitored during BDZs treatment in this population.

Introduction

Cognitive impairment is recognized as a core feature of schizophrenia, including deficits in working memory, executive function, attention, and processing speed, affecting approximately 75–80% of patients to varying degrees [1–4]. Given that these deficits are linked to impaired social functioning in domains such as community integration, occupational competence, and daily skill acquisition, cognitive dysfunction has been widely recognized as a major determinant of long-term outcome in schizophrenia [5–9].

The pathogenesis of cognitive impairment is complex. Current evidence implicates a combination of genetic vulnerability, cortical excitation-inhibition (E/I) imbalance, and dysregulation of neurotransmitters involving dopamine, glutamate, and gamma-aminobutyric acid (GABA) [10]. Several studies have indicated that pharmacological treatment also plays a crucial role in the progression of cognitive impairment observed in schizophrenia [11, 12]. Given the role of GABAergic dysfunction in cognitive processes, medications targeting the GABA system, particularly benzodiazepines (BDZs), may have clinically relevant effects on cognition in schizophrenia.

The cognitive benefits of currently available psychotropic medications are limited [13]. Moreover, some treatments, such as conventional antipsychotics, may worsen cognitive impairment in schizophrenia [14]. Meanwhile, there is growing concern about the effects of BDZs on cognitive function in schizophrenia. BDZs represent one of the most frequently used adjunctive pharmacotherapies in patients with schizophrenia, and are widely used for various therapeutic purposes, including the management of anxiety and sleep disturbances, the mitigation of antipsychotic side effects, and rapid tranquilization [15]. In the CATIE trial, the co-prescription rate of BDZs was approximately 22%. Notably, another study demonstrated that once initiated, long-term use was highly prevalent, with cumulative treatment duration exceeding one year in up to 62.9% of schizophrenia patients [16].

Although BDZs are considered safe and effective for short-term use, their potential for long-term cognitive impact remains a concern. In non-psychotic patients, long-term BDZs use is often associated with broad cognitive deficits [17]. A recent meta-analysis has confirmed this association, although the severity of impairment and the specific cognitive domains involved vary considerably [18]. The underlying mechanisms for BDZ-induced cognitive impairment may involve the acute enhancement of $GABA_A$ receptor-mediated inhibitory neurotransmission [19]. This disruption of hippocampal and prefrontal cortical function can lead to deficits in working memory, executive function, and attention. Additionally, long-term BDZs use may promote neuroplastic changes or receptor downregulation, further exacerbating cognitive decline [20, 21]. However, it remains uncertain whether these mechanisms operate similarly in schizophrenia, given baseline cognitive impairment and altered GABAergic signaling.

The cognitive effects of BDZs in schizophrenia remain debated. A cross-sectional study reported an association between BDZs use and poorer cognitive and social function in older adults (≥ 55 years) with schizophrenia, along with increased hospitalization duration and readmission rates [22]. Research on community-dwelling stabilized patients has shown that daily use of BDZs is associated with reduced working memory and attentional performance [23]. In contrast, a few studies have found no significant association between benzodiazepine use and cognitive function [24]. Moreover, research suggests that cognitive deficits associated with BDZs may be at least partially reversible. In a Japanese cohort of patients with schizophrenia, significant improvement in verbal memory was observed following gradual BDZs discontinuation [25]. Longitudinal studies have not consistently demonstrated progressive decline over time; some evidence suggests that cognitive deficits appear to stabilize after a certain duration of exposure [26]. These conflicting findings may be attributed to variations in study design, BDZs dosage, exposure duration, or the cognitive domains assessed. Thus, the real-world association between long-term BDZs use and cognitive function in schizophrenia has yet to be clearly characterized.

Therefore, this study aimed to examine the association between long-term BDZs use and cognitive function in patients with schizophrenia using real-world data. Propensity score matching was applied to balance key clinical characteristics, allowing for a more robust assessment of the cognitive impact of long-term BDZs use in routine clinical practice.

Methods

Participants

The data for this study were obtained from the SALT-C study, an observational study on safety and related factors of treatment with atypical antipsychotics in Chinese patients with schizophrenia [27]. Participants were recruited from the Shanghai Mental Health Center between 2016 and 2019. All participants were outpatients or inpatients aged 18 years or older with a diagnosis of schizophrenia according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV), confirmed independently by at least two attending psychiatrists. All participants were either receiving or planning to initiate atypical antipsychotic (AAP) treatment. The study protocol was approved by the Ethics Committee of the Shanghai Mental Health Center (2010-35).

Based on this cohort, this analysis aimed to evaluate the association between long-term BDZs use and cognitive function. Participants were eligible for the long-term BDZs user group if they had been using BDZs at a stable dosage for at least 60 consecutive days prior to enrollment. Participants were eligible for the BDZs non-user group if they had no prior history of BDZs use.

Patients were excluded if they had comorbid psychiatric disorders (including bipolar disorder, major depressive disorder, or substance-induced psychotic disorders); neurological diseases (such as epilepsy, Parkinson's disease, stroke, or traumatic brain injury); severe physical illnesses; a history of substance abuse or dependence (except nicotine) within the preceding three months; intellectual disability or severe cognitive impairment that would preclude completion of cognitive assessments; refusal or inability to provide informed consent; concurrent participation in another clinical study; were deemed unsuitable by the investigator; were currently receiving electroconvulsive therapy (ECT) or modified ECT without convulsion; or were currently using nootropics or potent anticholinergic agents.

Procedures

Demographic and clinical data were collected, including age, gender, years of education, episode and duration of illness. Detailed medication information was recorded for all prescribed AAPs, and for concomitant medications such as antidepressants. Information on BDZs was recorded, including the specific type, initial prescription date, and duration of use. Long-term use was defined as continuous use exceeding 60 days.

Cognitive function was evaluated using the Montreal Cognitive Assessment (MoCA) [28]. The MoCA assesses multiple cognitive domains, including attention and concentration, executive function, memory, language, visuospatial ability, abstract thinking, and orientation. The total score ranges from 0 to 30, with higher scores indicating better cognitive performance; a cutoff score of 26 was used, with scores ≥ 26 indicating no cognitive impairment and scores < 26 indicating cognitive impairment.

Psychopathology was assessed using the total score and the positive, negative, and general psychopathology subscale scores of the Positive and Negative Syndrome Scale (PANSS) [29]. Illness severity and psychosocial functioning were evaluated using the Clinical Global Impression-Severity (CGI-S) scale and the Personal and Social Performance (PSP) scale [30, 31].

All psychological assessments were conducted by trained psychiatrists, having completed standardized training for each scale and demonstrated inter-rater reliability prior to data collection.

Statistical analysis

Propensity score matching (PSM) was performed to reduce selection bias between the study groups. A 1:1 matching protocol was applied based on age, gender, illness duration, and type of AAPs.

All statistical analyses were conducted using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables following normal distribution were expressed as mean and standard deviation [M (SD)]; non-normally distributed data were expressed as median and interquartile range [M (P25, P75)]. Categorical and ordinal variables were summarized as frequencies and percentages. Between-group comparisons were performed using independent-samples t-tests or Mann–Whitney U tests. Multivariate analysis was performed using binary logistic regression. A two-tailed P-value < 0.05 was considered statistically significant.

Results

Demographic characteristics and propensity score matching

Of 842 patients initially screened, 690 were included in the analysis, comprising 62 long-term BDZs users and 628 non-users (Fig. 1). Among patients treated with BDZs, 66.0% were long-term users. The most commonly prescribed BDZs were alprazolam, followed by clonazepam and lorazepam; other agents included oxazepam, estazolam, and diazepam.

Before propensity score matching, significant differences were observed between the two groups in age, episode status (first episode vs. relapse), illness duration, and type of AAPs prescribed (all P < 0.05).

After propensity score matching, 57 patients were retained in the long-term BDZs user group and the BDZs non-user group, respectively. There were no significant differences between the two groups in age, gender, years of education, patient type (outpatient vs. inpatient), schizophrenia subtype, episode status, illness duration, or type of AAPs (all P > 0.05), indicating adequate covariate balance. In the long-term BDZs user group, the commonly prescribed AAP was olanzapine (43.9%), followed by aripiprazole (17.5%). Similarly, in the BDZs non-user group, olanzapine was the most commonly prescribed AAP (45.6%), followed by aripiprazole (17.5%) (Table 1).

Figure 1 Trial profile.

Note

BDZs: benzodiazepines; PSM: propensity score matching; MoCA: Montreal Cognitive Assessment.

Table 1
Demographic characteristics.

	Before PSM				After PSM			
	Long-term BDZs users	BDZs non-users	Z/χ^2	*P*	Long-term BDZs users	BDZs non-users	Z/χ^2	*P*
	(n = 62)	(n = 628)			(n = 57)	(n = 57)		
Age	57.00(46.75,63.00)	53.00(37.00,60.00)	-2.897	0.004*	57.00(49.00,64.00)	57.00(48.00,63.00)	-0.139	0.889
Gender			1.588	0.208			0.039	0.843
Male	40 (64.5%)	353 (56.2%)			37(64.9%)	38(66.7%)		
Female	22 (35.5%)	275 (43.8%)			20(35.1%)	19(33.3%)		
Years of education	9.00(8.00,12.00)	9.00(9.00,12.00)	-1.253	0.201	9.00(8.00,12.00)	9.00(8.00,12.00)	-0.382	0.703
Patient type			5.449	0.066			1.036	0.309
Outpatient	2 (3.2%)	84 (13.4%)			1 (1.8%)	3 (5.3%)		
Inpatient	60 (96.8%)	544 (86.6%)			56 (98.2%)	54 (94.7%)		
Schizophrenia subtype			7.877	0.096			1.345	0.718
Hebephrenic schizophrenia	0 (0.0%)	6 (0.9%)			0 (0.0%)	0 (0.0%)		
Catatonic schizophrenia	0 (0.0%)	6 (0.9%)			0 (0.0%)	0 (0.0%)		
Paranoid schizophrenia	20 (32.3%)	227 (36.1%)			18 (31.6%)	20 (35.1%)		
Undifferentiated schizophrenia	10 (16.1%)	43 (6.8%)			10 (17.5%)	8 (14.0%)		
Residual schizophrenia	32 (51.6%)	346 (55.1%)			29 (50.9%)	29 (50.9%)		
Episode			8.220	0.004*			2.151	0.142
First-episode status	2 (3.2%)	108 (17.2%)			2 (3.5%)	6 (10.5%)		
Relapse status	60 (96.8%)	520 (82.8%)			55 (96.5%)	51 (89.5%)		
Illness duration	28.00(18.50,35.50)	22.50(10.00,30.00)	-3.255	0.001*	28.00(19.00,37.50)	28.00(17.00,35.50)	-0.397	0.691
AAP			24.758	0.001*			0.353	1.000
Quetiapine	1 (1.6%)	37 (5.9%)			1 (1.8%)	1 (1.8%)		
Olanzapine	28 (45.2%)	180 (28.7%)			25 (43.9%)	26 (45.6%)		
Risperidone	9 (14.5%)	143 (22.8%)			9 (15.8%)	9 (15.8%)		
Aripiprazole	10 (16.1%)	122 (19.4%)			10 (17.5%)	10 (17.5%)		
Ziprasidone	2 (3.2%)	8 (1.3%)			2 (3.5%)	1 (1.8%)		
Paliperidone	3 (9.4%)	14 (2.2%)			3 (5.3%)	3 (5.3%)		
Amisulpride	1 (1.6%)	30 (4.8%)			1 (1.8%)	1 (1.8%)		
Perospirone	0 (0.0%)	0 (0.0%)			0 (0.0%)	0 (0.0%)		
Clozapine	8 (12.9%)	94 (15.0%)			6 (10.5%)	6 (10.5%)		
BDZ Treatment duration (months)	31.00(10.70,48.00)				31.00(14.50,50.00)			

Note: Data are presented as number (percentage%) or median (interquartile range). PSM: propensity score matching; BDZs: benzodiazepines; AAP: atypical antipsychotic. ** P < 0.05.*

Comparison of clinical symptoms and severity between groups

The total PANSS score was 57.50 (49.00, 75.75) in the long-term BDZs user group and 55.00 (46.75, 70.25) in the BDZs non-user group, with no significant difference observed between the groups (P > 0.05). Similarly, no significant differences were observed between the groups in the PANSS positive, negative, or general psychopathology subscales (all P > 0.05). The CGI-S and PSP scores also showed no significant differences between groups (all P > 0.05), indicating comparable severity of clinical symptoms and social functioning (Table 2).

Table 2
Comparison of clinical symptoms, severity, and social performance.

	Long-term BDZs users	BDZs non-users	*Z*	*P*
PANSS total score	57.50 (49.00,75.75)	55.00 (46.75,70.25)	-0.969	0.333
PANSS-positive score	8.00 (7.00,16.00)	8.50 (7.00,12.00)	-0.814	0.415
PANSS-negative score	19.00 (16.25,25.75)	20.00 (15.75,24.00)	-0.509	0.610
PANSS-general psychopathology score	28.00 (24.00,36.00)	27.00 (22.00,35.00)	-0.832	0.405
CGI-S score	4.00 (3.00,4.00)	3.00 (3.00,4.50)	-0.571	0.568
PSP score	60.00 (40.00,70.50)	60.00(45.00,65.00)	-0.216	0.829

Note: Data are presented as median (interquartile range). BDZs: benzodiazepines; PANSS: Positive and Negative Syndrome Scale; CGI-S: Clinical Global Impression-Severity Scale; PSP: Personal and Social Performance Scale.

Comparison of cognitive function between groups

The mean total MoCA score was significantly lower in the long-term BDZs user group [16.02 (6.69)] compared with the BDZs non-user group [19.33 (7.11); P = 0.012, t = 2.566]. Both groups scored below the cutoff of 26, indicating the presence of overall cognitive impairment.

Analysis of MoCA subdomains showed significantly poorer performance in the long-term BDZs user group compared with the BDZs non-user group on visuospatial/executive function (P = 0.012, Z = -2.501), naming (P = 0.025, Z = -2.236), language (P = 0.032, Z = -2.139), and abstraction (P = 0.032, Z = -2.142). No significant differences were observed in attention, delayed recall, or orientation (all P > 0.05) (Table 3).

Table 3
Comparison of cognitive function.

	Long-term BDZs users	BDZs non-users	*t/Z*	*P*
MoCA total score	16.02 (6.69)	19.33 (7.11)	2.566	0.012*
Visuospatial/executive	2.00 (1.00,3.00)	3.00 (1.50,4.00)	-2.501	0.012*
Naming	2.00 (2.00,3.00)	3.00 (2.00,3.00)	-2.236	0.025*
Attention	4.00 (3.00,5.00)	5.00 (3.00,6,00)	-1.891	0.059
Language	0.00 (0.00,1.00)	1.00 (0.00,2.00)	-2.139	0.032*
Abstraction	0.82 (0.83)	1.18 (0.87)	-2.142	0.032*
Delayed recall	1.00 (0.00,2.00)	2.00 (0.00,2.50)	-1.856	0.063
Orientation	6.00 (4.00,6.00)	6.00 (5.00,6.00)	-1.547	0.122

Note: Data are presented as mean (SD) or median (interquartile range). BDZs: benzodiazepines; MoCA: Montreal Cognitive Assessment. ** P < 0.05.*

Multivariate Regression Analysis on correlates of cognitive impairment

A binary logistic regression model was constructed to assess factors associated with cognitive impairment. Normal cognition (MoCA ≥ 26) served as the reference category for the dependent variable. The model included the following independent variables: age, gender, years of education, illness duration, and long-term BDZs use, and showed good model fit (χ^2 = 3.088, P = 0.929).

Among these variables, long-term BDZs use was independently associated with cognitive impairment (OR = 7.728, P = 0.017), whereas years of education was associated with cognitive impairment (OR = 0.740, P = 0.007), while age, gender, and duration of illness were not significantly

associated with cognitive impairment (Fig. 2, Table 4).

Figure 2 Correlates of cognitive impairment

Note

BDZs: benzodiazepines; OR: odds ratio.

Table 4
Logistic Regression analysis of factors associated with cognitive function

	B	SE	Wald χ^2	P	OR	95%CI	
						C_{min}	C_{max}
Long-term BDZs use	2.045	0.853	5.745	0.017*	7.728	1.452	41.135
Years of education	-0.301	0.111	7.355	0.007*	0.740	0.595	0.920
Age	-0.014	0.036	0.148	0.700	0.986	0.920	1.058
Gender	0.509	0.794	0.410	0.522	1.663	0.351	7.892
Illness duration	0.059	0.041	2.013	0.156	1.061	0.978	1.150
Note: ** P < 0.05.*							

Discussion

This real-world study explored the association between long-term BDZs use and cognitive function in patients with schizophrenia. The primary finding was that patients with long-term BDZs use demonstrated significantly poorer overall cognitive performance than BDZs non-users. Moreover, long-term BDZs use was independently associated with cognitive impairment, while years of education was negatively associated with cognitive impairment. Additionally, it was observed that the proportion of long-term users among BDZ users was relatively high.

In this study, cognitive function was assessed using the MoCA, a widely used and sensitive screening tool for cognitive impairment that has been validated for detecting cognitive deficits in patients with schizophrenia [28, 32]. Propensity score matching was applied to balance baseline characteristics and account for potential confounding factors between long-term BDZs users and non-users [33]. Patients with long-term BDZs use had lower total MoCA scores, with more pronounced deficits observed in naming, language, abstraction, and visuospatial/executive subdomains compared with non-users, indicating an association between long-term BDZs use and poorer performance in these cognitive domains. Similar associations have also been reported in the non-psychiatric population [34]. This finding aligns with the multicenter FACE-SZ study, which reported that among 407 clinically stable patients with schizophrenia, long-term BDZs use was significantly associated with impairments in verbal memory and working memory [23]. Similarly, previous research has reported lower scores on the Global Assessment of Functioning–Cognition in Schizophrenia (GAF-CogS) scale in patients with schizophrenia spectrum disorders receiving long-term BDZ therapy[35]. Despite methodological differences, impairments across multiple cognitive domains were identified, particularly in language and executive function. Moreover, the present study found that patients with long-term BDZs use performed worse in visuospatial and abstraction domains, which is consistent with previous findings [36]. Overall, these observations suggest an association between long-term BDZs use and deficits across multiple cognitive domains in patients with schizophrenia.

Although some studies have suggested that long-term BDZs users present with more severe clinical symptoms [35], no significant between-group differences in clinical symptom severity or social functioning were observed in our sample.

In the present study, multivariable logistic regression analysis indicated that long-term BDZs use was positively associated with cognitive impairment in patients with schizophrenia, whereas years of education were inversely associated with cognitive impairment. These findings further suggest long-term BDZs use independently associated with an increased cognitive impairment after adjustment for relevant covariates. Nevertheless, although long-term benzodiazepine use was associated with greater cognitive impairment (OR = 7.728), the relatively wide confidence interval indicates limited precision of the effect estimate. The limited sample size and heterogeneity in duration of BDZs use may contribute to this uncertainty. These results point to a possible risk trend, with the clinical significance requiring further investigation in larger, prospective studies. The association between years of education and cognitive impairment is consistent with the cognitive reserve hypothesis, whereby educational attainment could increase the tolerance to disease-related brain dysfunction [37].

Among all initially recruited patients prescribed BDZs, approximately two-thirds were identified as long-term users. The mean duration of BDZs use was 31 months, exceeding two years. Although the overall rate of BDZs co-prescription in the baseline dataset was lower than that reported in some previous studies, the proportion of long-term users was relatively high. This observation might be partially attributable to real-world prescribing practices, as suggested in previous study, whereby BDZ treatment, once initiated, tends to be maintained over extended periods in certain clinical

settings [16]. Similar findings have been reported, with up to 62.9% of patients with schizophrenia exposed to BDZs for more than one year [38]. This interpretation remains uncertain and needs to be evaluated in longitudinal studies focusing on long-term BDZs prescribing practices and related clinical factors.

Definitions of *long-term use* vary across studies, ranging from several months to years; the most common thresholds are continuous use for 3–12 months [23, 35, 39, 40]. In the present study, long-term use was defined as continuous exposure for more than two months. This operational definition was selected based on clinical practice considerations and data availability in a real-world setting.

Several limitations of this study should be acknowledged. First, residual confounding cannot be fully excluded despite the use of propensity score matching. Second, cognitive function was assessed at a single time point using the MoCA, which precludes evaluation of longitudinal changes and may limit precision in domain-specific cognitive assessment. Third, potentially relevant factors such as illness duration, episode status, and concomitant medications were not formally analyzed, and the relatively high proportion of long-term BDZ use was identified as an exploratory observation. Finally, the cognitive effects of short-term BDZs use were not evaluated in the present study. Future studies using multicenter, longitudinal designs and controlled interventional approaches are needed to further characterize the clinical factors associated with the relationship between BDZs use and cognitive function.

The findings of the present study may have clinical implications for the use of BDZs in patients with schizophrenia. Clinicians may consider paying attention to treatment duration and cognitive status during BDZ therapy, while carefully weighing potential anxiolytic or sedative benefits against possible associations with cognitive impairment. Decisions regarding dose reduction, discontinuation, or the use of alternative interventions should be individualized and supported by further empirical evidence.

Conclusions

Long-term use of BDZs was associated with lower global cognitive performance in patients with schizophrenia receiving atypical antipsychotics, with pronounced deficits in visuospatial/executive function, naming, language, and abstraction. Long-term BDZs use was independently associated with cognitive impairment, whereas years of education was associated with cognitive impairment. These findings underscore the importance of monitoring cognitive function in patients with schizophrenia treated with BDZs.

Abbreviations

BDZs benzodiazepines

E/I excitation-inhibition

GABA gamma-aminobutyric acid

AAP atypical antipsychotic

MoCA the Montreal Cognitive Assessment

PANSS the Positive and Negative Syndrome Scale

CGI-S the Clinical Global Impression-Severity Scale

PSP the Personal and Social Performance Scale

PSM propensity score matching

SD standard deviation

OR odds ratio

95% CI 95% confidence interval

Declarations

Ethics approval and consent to participate

The study was approved by the Ethics Committee of the Shanghai Mental Health Center (2010-35). All participants provided written informed consent.

Consent for publication

Not applicable.

Declaration of competing interest:

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Author Contribution

Caiping Liu: Conceptualization, Formal analysis, Writing – original draft. Lei Zhang: Investigation, Methodology, Data curation. Qiyang Pan: Investigation, Formal analysis. Yue Shi: Data curation, Visualization. Wenjuan Yu:Investigation, Writing – review ; editing. Guanjun Li:Methodology. Writing – review ; editing. Huafang Li:Writing – original draft, Writing – review ; editing, supervision, Funding acquisition.

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Data Availability

The datasets generated and analysed during the current study are not publicly available due to the ethical and institutional restrictions. The informed consent and ethics approval did not include public data sharing.

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Figures

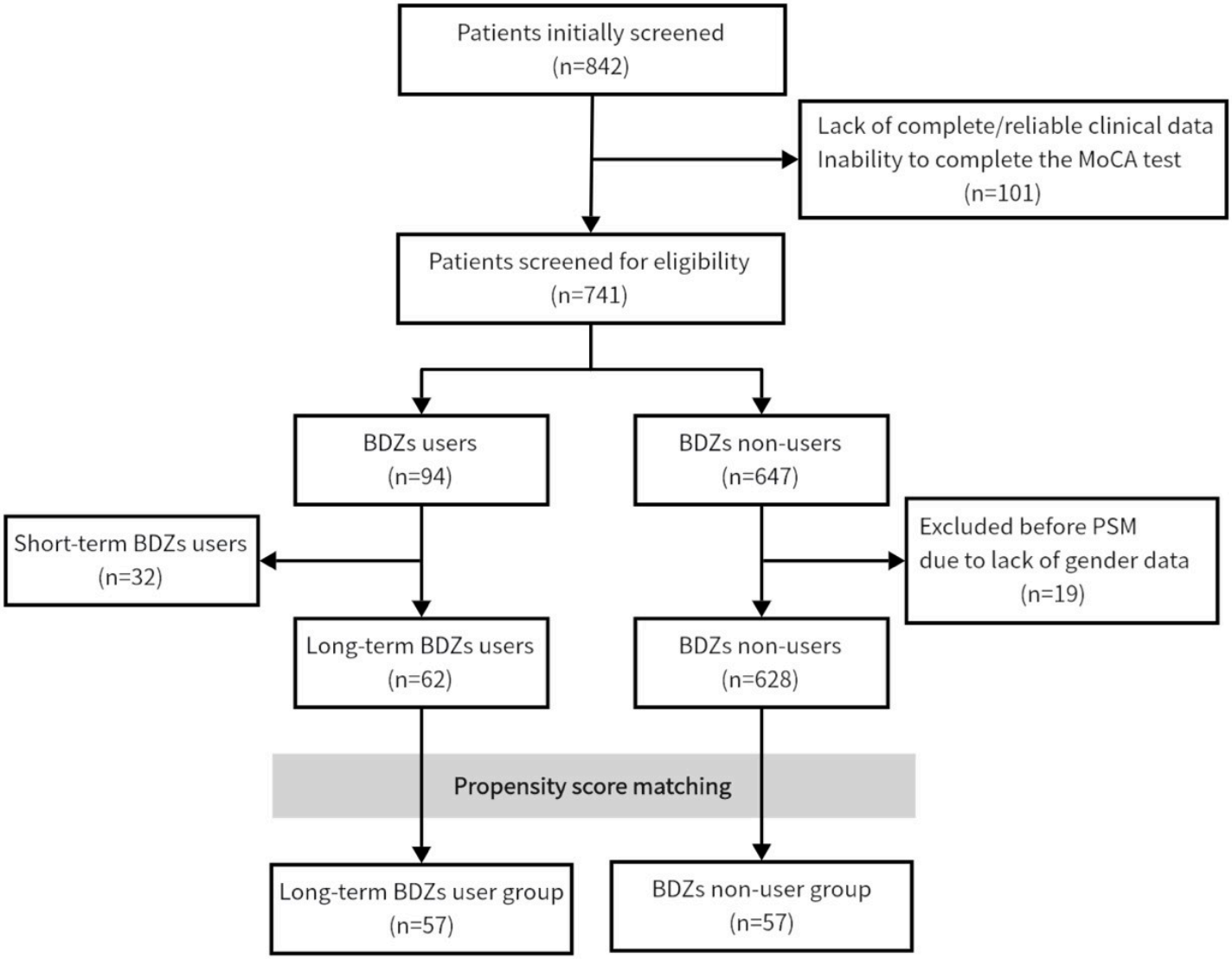

Figure 1

Trial profile.

Note: BDZs: benzodiazepines; PSM: propensity score matching; MoCA: Montreal Cognitive Assessment.

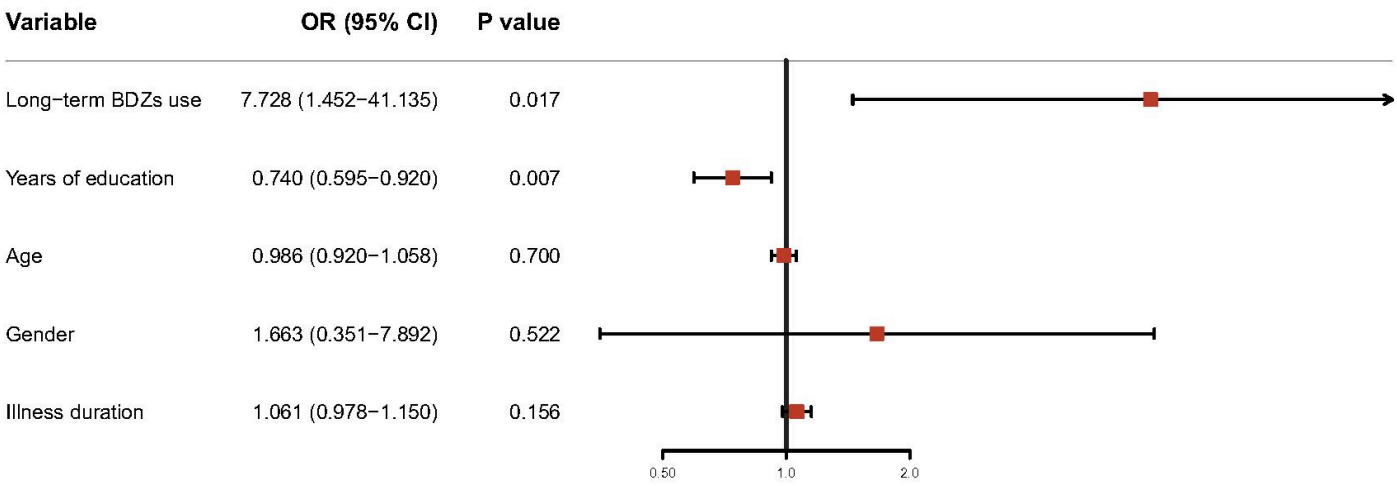

Figure 2

Correlates of cognitive impairment

Note: BDZs: benzodiazepines; OR: odds ratio.