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A longitudinal analysis of the relationship between emotional symptoms and cognitive function in patients with major depressive disorder

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Original Article

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Abstract

Background. The relationship between emotional symptoms and cognitive impairments in major depressive disorder (MDD) is key to understanding cognitive dysfunction and optimizing recovery strategies. This study investigates the relationship between subjective and objective cognitive functions and emotional symptoms in MDD and evaluates their contributions to social functioning recovery.

Methods. The Prospective Cohort Study of Depression in China (PROUD) involved 1,376 MDD patients, who underwent 8 weeks of antidepressant monotherapy with assessments at baseline, week 8, and week 52. Measures included the Hamilton Depression Rating Scale (HAMD-17), Quick Inventory of Depressive Symptomatology-Self Report (QIDS-SR16), Chinese Brief Cognitive Test (C-BCT), Perceived Deficits Questionnaire for Depression-5 (PDQ-D5), and Sheehan Disability Scale (SDS). Cross-lagged panel modeling (CLPM) was used to analyze temporal relationships.

Results. Depressive symptoms and cognitive measures demonstrated significant improvement over 8 weeks ($p < 0.001$). Baseline subjective cognitive dysfunction predicted depressive symptoms at week 8 (HAMD-17: $\beta = 0.190$, 95% CI: 0.108–0.271; QIDS-SR16: $\beta = 0.217$, 95% CI: 0.126–0.308). Meanwhile, baseline depressive symptoms (QIDS-SR16) also predicted subsequent subjective cognitive dysfunction ($\beta = 0.090$, 95% CI: 0.003-0.177). Recovery of social functioning was driven by improvements in depressive symptoms ($\beta = 0.384$, $p < 0.0001$) and subjective cognition ($\beta = 0.551$, $p < 0.0001$), with subjective cognition contributing more substantially ($R^2 = 0.196$ vs. 0.075).

Conclusions. Subjective cognitive dysfunction is more strongly associated with depressive symptoms and plays a significant role in social functioning recovery, highlighting the need for targeted interventions addressing subjective cognitive deficits in MDD.

Introduction

Major depressive disorder (MDD) is a prevalent condition that affects approximately 280 million individuals globally (World Health Organization, 2023). It is a leading cause of disability worldwide and significantly contributes to the global burden of disease (GBD 2019 Diseases and Injuries Collaborators, 2020). A study on first-episode drug-naive patients with MDD found that 13.7% of the patients exhibited suicide attempts within 1 month of the study (Li et al., 2024). Annually, more than 700,000 individuals die by suicide due to depression (World Health Organization, 2023).

Cognitive impairment is a significant manifestation of MDD; however, its relationship with depressive symptoms remains unclear. Current diagnostic criteria, as outlined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), recognize cognitive disturbances as associated features of MDD. A meta-analysis (Rock, Roiser, Riedel, & Blackwell, 2014) revealed significant moderate cognitive deficits in executive function, memory, and attention in patients with MDD; it also found that these cognitive impairments are fundamental aspects of the disorder, rather than mere epiphenomena secondary to low mood symptoms. Although cognitive symptoms are traditionally thought to be influenced by the depressive state, research indicates that these deficits can persist even in remitted states (Preiss et al., 2007; Rock et al., 2014), negatively impacting patients' overall functionality (Evans, Iverson, Yatham, & Lam, 2014). Our previous study found that difficulty with concentration and decision-making was the core residual symptom of MDD and was associated with poorer social functioning, increased family

burden, and lower life satisfaction (Zhou et al., 2024). However, to date, the question of whether they represent independent or concomitant symptoms remains unresolved.

Cognitive function can be evaluated through subjective self-assessments or objective tests. However, studies have identified significant discrepancies and weak correlations between subjective and objective cognitive measures in patients with MDD (Miller, 1975; Serra-Blasco et al., 2019). This discrepancy may be partially attributed to findings that emotional symptoms adversely affect subjective cognition but not objective cognition (Srisurapanont, Suttajit, Eurviriyanukul, & Varnado, 2017). This supports the hypothesis that depression severity contributes to negative cognitive bias, and the relationship between emotional symptoms and cognitive function may differ depending on whether it is subjectively or objectively assessed. Restoring social functioning is a key indicator of recovery from depression (Oluboka et al., 2018). While it has been traditionally assumed that alleviating emotional symptoms leads to improved social functioning, emerging evidence suggests otherwise (Iancu et al., 2020; Ojagbemi, Abiona, Luo, & Gureje, 2018). The dynamic interplay between cognition and depression, and their respective contributions to the restoration of social functioning, remains unclear.

This study has two aims: first, to examine the relationship between cognitive functions (both subjective and objective) and emotional symptoms using a robust sample of patients with MDD; second, to assess the individual impacts of cognitive function and depressive symptoms on social functioning. The first hypothesis of this study is that cognitive function is independent of emotional symptoms. The second hypothesis is that subjective and objective cognitive functions exert different effects on social functioning.

Methods

Study design and participants

The Prospective Cohort Study of Depression in China (PROUD) is a nationally representative, multicenter cohort study. Detailed methods of the PROUD study have been introduced in the published protocol (Zhou et al., 2023). This ongoing study was started in January 2022 and will continue till December 2026. Ethical approvals were obtained from Beijing Anding Hospital, Capital Medical University, Beijing, China, and the independent ethics committee overseeing all participating sites. The study has been registered with the Chinese Clinical Trial Registry (https://www.chictr.org.cn/showproj.html?proj=165790, registration number: ChiCTR2200059053). Written informed consents were obtained from all participants. The current study analyzed the data of the PROUD study collected from June 1, 2022, to June 29, 2024, in 18 qualified tertiary hospitals in China located in 14 provinces and cities. A total of 1,376 eligible patients with MDD were included. Inclusion criteria for participants were: (1) Diagnosed with MDD using the Mini-International Neuropsychiatric Interview (MINI); (2) males and females, aged 18–65 years; (3) having a score of 14 or more on the 17-item Hamilton Depression Rating Scale (HAMD-17); (4) having not taken any antidepressant medications for at least 14 days before screening; and (5) will be treated with antidepressant monotherapy. Patients were excluded if they had psychiatric comorbidities or other medical conditions that might interfere with the completion of the study or require the use of medications prohibited in the protocol.

Clinical and cognitive assessment

Emotional symptoms include both depressive and anxiety symptoms. In this study, depressive symptoms specifically refer to the core features of MDD, characterized by persistent low mood and anhedonia, representing a subset of emotional symptoms (Anderson et al., 2024). A total of seven standardized scales were used in this study to assess depressive and anxiety symptoms, cognitive function, and social functioning.

Hamilton depression rating scale (HAMD-17)

The HAMD-17 is a widely used clinical instrument for assessing the severity of depressive symptoms. This 17-item scale evaluates a range of symptoms, including mood, sleep, appetite, guilt, libido, and loss of interest, with each item scored on a 0–4 scale. The total score ranges from 0 to 52, with higher scores indicating more severe depression (Hamilton, 1960). The HAMD-17 showed good reliability (Cronbach's $\alpha = 0.85$) and validity in depression populations (Zheng et al., 1988).

Quick inventory of depressive symptomatology-self-report (QIDS-SR16)

The QIDS-SR16 is a self-report instrument that evaluates the severity of depressive symptoms. It includes 16 items covering sleep, appetite, interest, energy, physical symptoms, and mood, with items scored according to frequency of occurrence (0–3). The total score varies from 0 to 27, with higher scores reflecting greater severity of symptoms (Hamilton, 1959; Tang & Zhang, 1984). The QIDS-SR16 has shown good reliability (Cronbach's $\alpha = 0.73$–0.82) and validity in depression populations.

Hamilton anxiety rating scale (HAMA)

The HAMA is a 14-item scale rating the severity of anxiety symptoms via two factors: physical and psychological. Each item is rated 0–4 for severity of symptoms. The score range is 0–56; a higher score indicates greater severity of symptoms (Liu et al., 2013; Rush et al., 2003). The HAMA has good internal consistency (Cronbach's $\alpha = 0.74$–0.92) and acceptable test–retest reliability (correlation coefficients 0.74–0.97) (Maier, Buller, Philipp, & Heuser, 1988).

Generalized anxiety disorder-7 (GAD-7)

The GAD-7 is a 7-item self-report scale designed to assess the severity of anxiety symptoms. Items are rated according to symptom frequency (0–3), with total scores ranging from 0 to 21. Higher scores reflect more severe anxiety (Hidalgo & Sheehan, 2012). The GAD-7 exhibits excellent reliability (Cronbach's $\alpha = 0.82$) (Spitzer, Kroenke, Williams, & Löwe, 2006) and has a strong correlation ($r = 0.85$) with the HAMA (Ruiz et al., 2011) in patients with depression or anxiety.

Chinese brief cognitive test (C-BCT)

Cognitive functioning was assessed using the C-BCT, which is based on the MATRICS Consensus Cognitive Battery (MCCB) (Shi et al., 2015). It has been validated in a large-scale study of schizophrenia patients and has shown good internal consistency (Cronbach's $\alpha = 0.75$) and test–retest reliability (ICC = 0.62–0.76) (Ye et al., 2022). Also, additional studies in both the depression (Zhou et al., 2023) and the schizophrenia populations (Du et al., 2024; Zhou et al., 2024; Zhu et al., 2024) provide further evidence of its reliability and validity supporting the robustness of this scale for assessing cognitive function in these clinical groups.

Perceived deficits questionnaire-depression-5 item (PDQ-D5)

The PDQ-D5 assesses patients' self-perceived cognitive deficits. It includes 5 items rated on the severity of cognitive symptoms (0–4),

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with a total score ranging from 0 to 20. Higher scores indicate greater perceived cognitive dysfunction (Sullivan, Edgley, & Dehoux, 1990). The PDQ-D5 has demonstrated good reliability (Cronbach's α = 0.795–0.948) and validity in depressed populations (Shi et al., 2017).

Sheehan disability scale (SDS)

The SDS evaluates the impact of depression on a patient's work, social life, and family responsibilities. The total score ranges from 0 to 30, with higher scores indicating greater functional impairment (Sheehan et al., 2011; Sheehan, Harnett-Sheehan, & Raj, 1996). The SDS has demonstrated strong reliability (Cronbach's α = 0.94) and validity in depressed populations (Leu et al., 2015).

Procedures

Clinic visits for all patients took place at study sites at baseline and Weeks 8 and 52. The interviews were conducted by interviewers with standardized training. The training included practice scoring with feedback from an expert group and one-on-one discussion with the raters who scored very differently from the others. The C-BCT was administered using a tablet at baseline and Weeks 8 and 52. The tasks were administered in the following order: Trail Making Test, Part A (TMT-A), Symbol Coding, Continuous Performance Test (CPT), and Digit Span. The entire assessment would take place in a quiet room free from distractions, with only the researcher and the patient present. All patients were encouraged to make their best effort to complete the tasks and were allowed to take breaks if they felt tired or uncomfortable.

Statistical analysis

The prospective relationships between depressive symptoms and cognitive function were analyzed using cross-lagged panel modeling (CLPM). This method was chosen to systematically examine the directional effects and to offer insights into the temporal associations between these two variables. The analysis specifically aimed to address two key questions: (1) whether depressive symptoms and cognitive function mutually influence each other over time, and (2) the direction of these influences – whether cognitive function predicts subsequent depressive symptoms or vice versa. No constraints were applied to the CLPM in this study. To handle the missing data, we employed full information maximum likelihood (FIML) estimation, which directly fits the models to the raw data. This approach is recognized for producing less biased and more reliable results compared with traditional methods such as listwise deletion (Orth, Clark, Donnellan, & Robins, 2021). Standardized estimates and 95% confidence intervals were reported for the paths of interest. Model fit was evaluated using the comparative fit index (CFI) and root mean square error of approximation (RMSEA), where a CFI near one and an RMSEA close to zero indicated a good model fit. In all models, benchmark values for interpreting the size of the CLPM cross-lag effect, standardized betas, may be interpreted similarly to correlation coefficients wherein small = 0.1, medium = 0.3, and large = 0.5 (Bredemeier et al., 2023; Cohen, 1992).

To ensure the robustness of our findings, we conducted a series of sensitivity analyses. First, to strengthen the findings, we employed both self-report and clinician-rated tools for evaluations in the current study (HAMD-17 and QIDS-SR16 for depressive symptoms, HAMA and GAD-7 for anxiety symptoms, and C-BCT and PDQ-D5 for cognitive function). Second, we applied a multiple-group CLPM, stratified by episode status (first episode vs. relapse) and treatment type (SSRIs vs. other medications). Third, to explore the contribution of key predictors to changes in symptoms and cognitive function, we included variables such as age, total illness duration, education level, and number of episodes based on a literature review and clinical experience (Hasselbalch, Knorr, & Kessing, 2011; Sachs-Ericsson et al., 2013; Zaninotto et al., 2016), to assess their impact on both domains. Finally, data from three follow-up time points were included to further validate the relationship between symptoms and cognitive function in the model. The 52-week follow-up data included all participants who successfully completed the follow-up, without any specific inclusion or exclusion criteria.

Linear mixed models (LMM) were constructed using all available data without imputation. The models included random intercepts, with visit time modeled as fixed effects and participants as random effects, to estimate linear changes in cognitive function and depressive symptoms over the assessment waves. This approach was chosen due to the limitations of the cross-lagged panel model (CLPM) in explicitly modeling longitudinal trends in outcome measures. Consequently, assessing the extent of longitudinal change is essential for determining the appropriateness of applying these models in this context (Best & Cosco, 2022).

A multiple linear regression analysis was conducted to evaluate the contribution of changes in depressive symptoms and cognitive function to the improvement in social functioning. The model's covariates included the number of depressive episodes, total illness duration (in months), and age. To assess multicollinearity among the variables, the variance inflation factor (VIF) was calculated, with a threshold of 2 applied to identify potential collinearity issues.

All CLPM were conducted in Mplus version 8.1, and other analyses were performed using SAS for Windows, version 9.4 (SAS Institute, Cary, NC) or R version 4.3.2 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Demographic and clinical characteristics

Among the 1,376 patients included in the study, 900 (65.41%) were female and 252 (18.37%) had a family history of mental disorders. A total of 820 patients (59.85%) were experiencing their first episode, and 722 (52.47%) were treated with SSRIs. The median duration of illness was 12.00 months (interquartile range: 2.00–49.00 months), and the median age was 27.86 years (interquartile range: 22.88–35.86 years) (Table 1). Further stratified analyses of basic demographic and clinical characteristics by episode status (first episode vs. relapse) and treatment type (SSRIs vs. other medications) are presented in Supplementary eTable 1 and Supplementary eTable 2, respectively.

Trends in symptom changes over time

Supplementary eFigure 1 in the supplement summarizes the trend of changes in total scores across different measurements. LMM revealed that the changes from baseline to Week 8 were statistically significant. Specifically, the C-BCT total score increased from 48.82 at baseline to 53.85 at Week 8 ($F = 94.68$, $P < 0.001$); the HAMD-17 total score decreased from 20.72 at baseline to 10.53 at Week 8 ($F = 1625.93$, $P < 0.001$); the PDQ-D5 total score decreased from 11.30 at baseline to 6.97 at Week 8 ($F = 252.52$, $P < 0.001$); the SDS total score decreased from 15.31 at baseline to 8.52 at Week

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Table 1. Demographic and clinical characteristics of the participants

Variables	*n* (%) or median (interquartile range)
Sex	
Male	476 (34.59)
Female	900 (65.41)
Family history of mental disorder	252 (18.37)
Body mass index category	
Normal or healthy weight	673 (48.91)
Overweight	361 (26.24)
Underweight	342 (24.85)
Ethnicity	
Han	1279 (93.91)
Hui	26 (1.91)
Manchu	12 (0.88)
Mongolian	9 (0.66)
Other	36 (2.64)
Education level	
Primary school	26 (1.91)
Middle school	121 (8.90)
High school	177 (13.02)
University	866 (63.72)
Postgraduate	169 (12.44)
Geographic location	
Urban	1112 (84.76)
Rural	200 (15.24)
Monthly income range (RMB)	
Below 1,000	35 (2.78)
1,001–5,000	365 (29.04)
5,001–10,000	466 (37.07)
Above 10,000	391 (31.11)
Type of health insurance	
Urban resident basic medical insurance	298 (23.28)
Urban employee basic medical insurance	586 (45.78)
Public healthcare	68 (5.31)
Full self-pay	123 (9.61)
New rural cooperative medical insurance	180 (14.06)
Other	25 (1.95)
Marital status	
Single	828 (61.06)
Married	448 (33.04)
Divorced/widowed	80 (5.90)
Employment status	
Part-time job	44 (3.27)
Full-time job	662 (49.18)
Student	350 (26.00)
Retired/unemployed	290 (21.55)
First episode	820 (59.85)
Type of medication	
SSRIs	722 (52.47)
Other	513 (47.53)
Age (years)	27.86 (22.88–35.86)
Overall duration of illness (months)	12.00 (2.00–49.00)
Duration of the current episode (months)	2.00 (1.00–8.00)
Onset age (year)	27.00 (21.00–35.00)
Number of episodes	1.00 (1.00–2.00)

Note: RMB, Renminbi; SSRIs, Selective Serotonin Reuptake Inhibitors. Percentages are calculated from the total sample size (n = 1376). Data presented as n (%) or median (interquartile range).

8 (F = 287.52, $P < 0.001$). See the Supplementary Materials for details.

Relationships between cognitive function, depressive symptoms, and anxiety symptoms

Results of the cross-lagged panel models depicting the relationships of objective cognition with depressive symptoms and with anxiety symptoms are displayed in Figure 1. As expected, all cross-sectional relationships between the variables were statistically significant at both time points ($P < 0.05$). In the analysis of prospective associations, statistically significant positive relationships were observed between the variables at baseline and Week 8 ($P < 0.05$). However, no significant association was found between depressive symptoms at baseline (measured by HAMD-17 or QIDS-SR16) and cognitive function at Week 8, as measured by C-BCT ($P > 0.05$). Similarly, anxiety symptoms at baseline were not statistically associated with cognitive function at Week 8 measured by C-BCT ($P > 0.05$). All model fit indices were consistent and satisfactory, with RMSEA = 0, CFI = 1.0.

Further sensitivity analyses exploring the relationship between depressive symptoms and objective cognition were stratified by episode status (first episode versus relapse) and treatment type (SSRIs vs. other medications), as illustrated in Figure 2.

Consistent with prior results, no significant association was observed in any stratification between baseline depressive symptoms (measured by the HAMD-17) and cognitive function at Week 8, as assessed by the C-BCT ($P > 0.05$). Additionally, both the CLPM model with additional variables, including age, total illness duration, education level, and number of episodes (see Supplementary eFigure 2 and Supplementary eFigure3 in the supplement) and the model built using data from three follow-up time points supported these results (see Supplementary eFigure 4 in the supplement). These findings were further validated with analyses of the results of different C-BCT subtests (see Supplementary eFigure 5 in the supplement). In contrast to these findings, the longitudinal analysis of the relationship between depressive symptoms and

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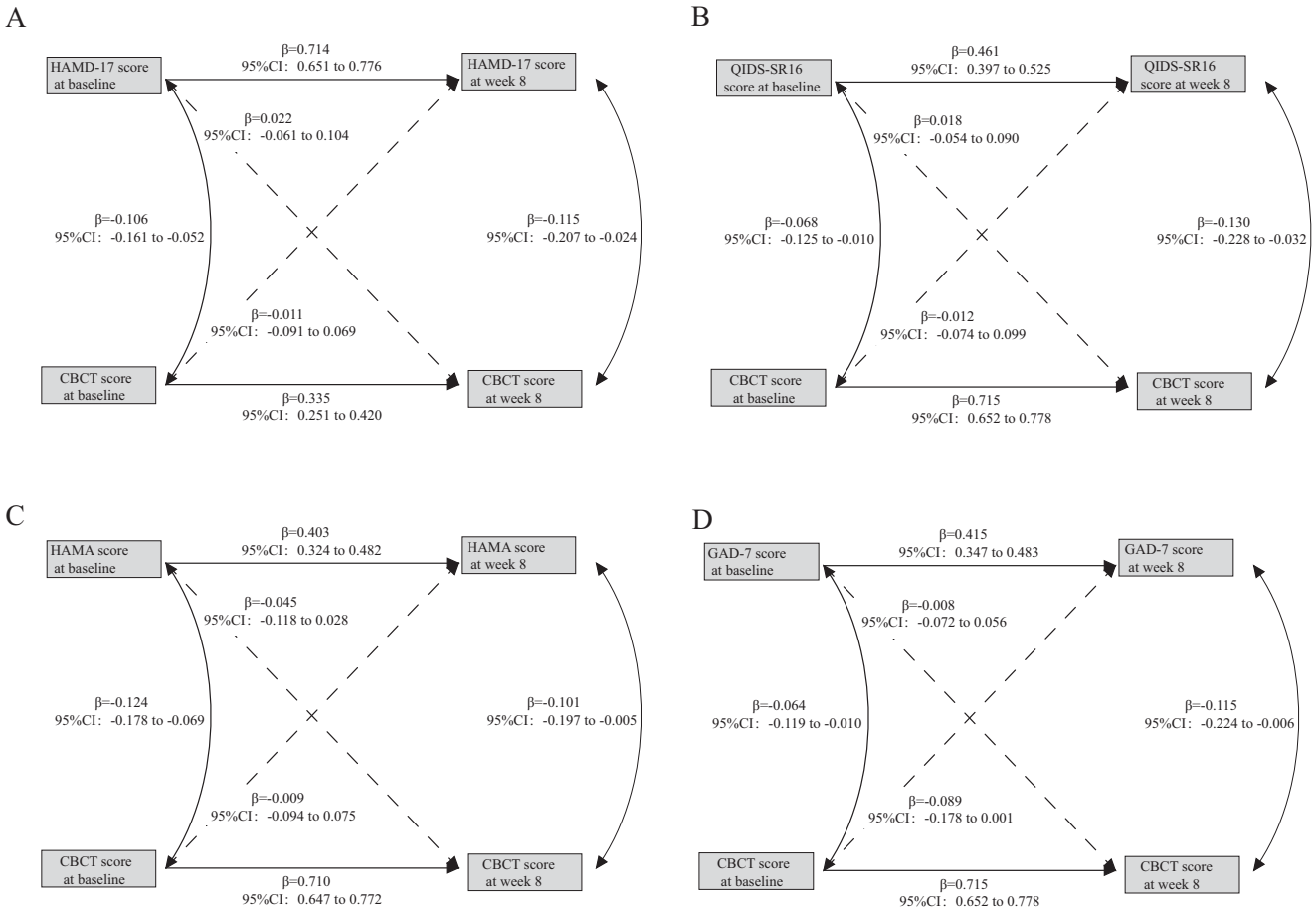

Figure 1. Cross-lagged panel models depicting the associations of objective cognition with depressive symptoms and with anxiety symptoms. *Note:* Standardized estimates with 95% confidence intervals are presented. Solid lines in the Cross-Lagged Panel Models indicate statistically significant standardized estimates, while dashed lines represent estimates that are not statistically significant. A total of 504 patients were followed at Week 8.

subjective cognitive function, as presented in Figure 3, revealed that baseline subjective cognitive function significantly predicted depressive symptoms at Week 8. For instance, PDQ-D5 scores at baseline were significantly associated with HAMD-17 scores at Week 8 (β = 0.190, 95% CI: 0.108–0.271). Similarly, PDQ-D5 scores at baseline were associated with QIDS-SR16 scores at Week 8 (β = 0.217, 95% CI: 0.126–0.308), and QIDS-SR16 scores at baseline were also significantly associated with PDQ-D5 scores at Week 8. Additionally, PDQ-D5 scores at baseline were significantly associated with HAMA scores at Week 8 (β = 0.227, 95% CI: 0.150–0.304), as were GAD-7 scores at baseline (β = 0.179, 95% CI: 0.089–0.269). All model fit indices were consistent and satisfactory, with RMSEA = 0, CFI = 1.0.

Effects of cognitive function and depressive symptoms on social functioning

Separate multiple linear regression models were constructed to assess the improvement in social functioning based on changes in objective and subjective cognitive function. In Model 1, the restoration of social functioning was found to depend solely on changes in depressive symptoms (β = 0.567, $P < 0.001$), with no significant contribution of objective cognitive function. However, in Model 2, the restoration of social functioning was influenced by both changes in depressive symptoms (β = 0.384, $P < 0.001$) and subjective cognitive function (β = 0.551, $P < 0.001$). Additionally, in Model 2, the R^2 for the decrease in the PDQ-D5 score was 0.196, higher than the R^2 for the decrease in the HAMD-17 score, which was 0.075 (Table 2).

Discussion

This study performed a comprehensive, large-scale longitudinal analysis using cross-lagged panel models to examine the temporal relationships between cognitive function and emotional symptoms in patients with MDD, clarifying directional relationships among the symptoms and these symptoms' impacts on the restoration of social functioning. A key finding was that there were significant directional relationships of both depressive and anxiety symptoms with subjective cognitive function but not with objective cognitive function. Furthermore, improvements in subjective cognitive function significantly contributed to the restoration of social functioning, exceeding the effects of improvements in depressive symptoms, whereas objective cognitive function showed no contribution.

The finding that subjective cognitive function can predict depressive and anxiety symptoms during follow-up was consistent with previous findings. For instance, a 2019 study on Intensive Care Unit (ICU) survivors revealed significant associations between subjective cognitive function and psychological symptoms,

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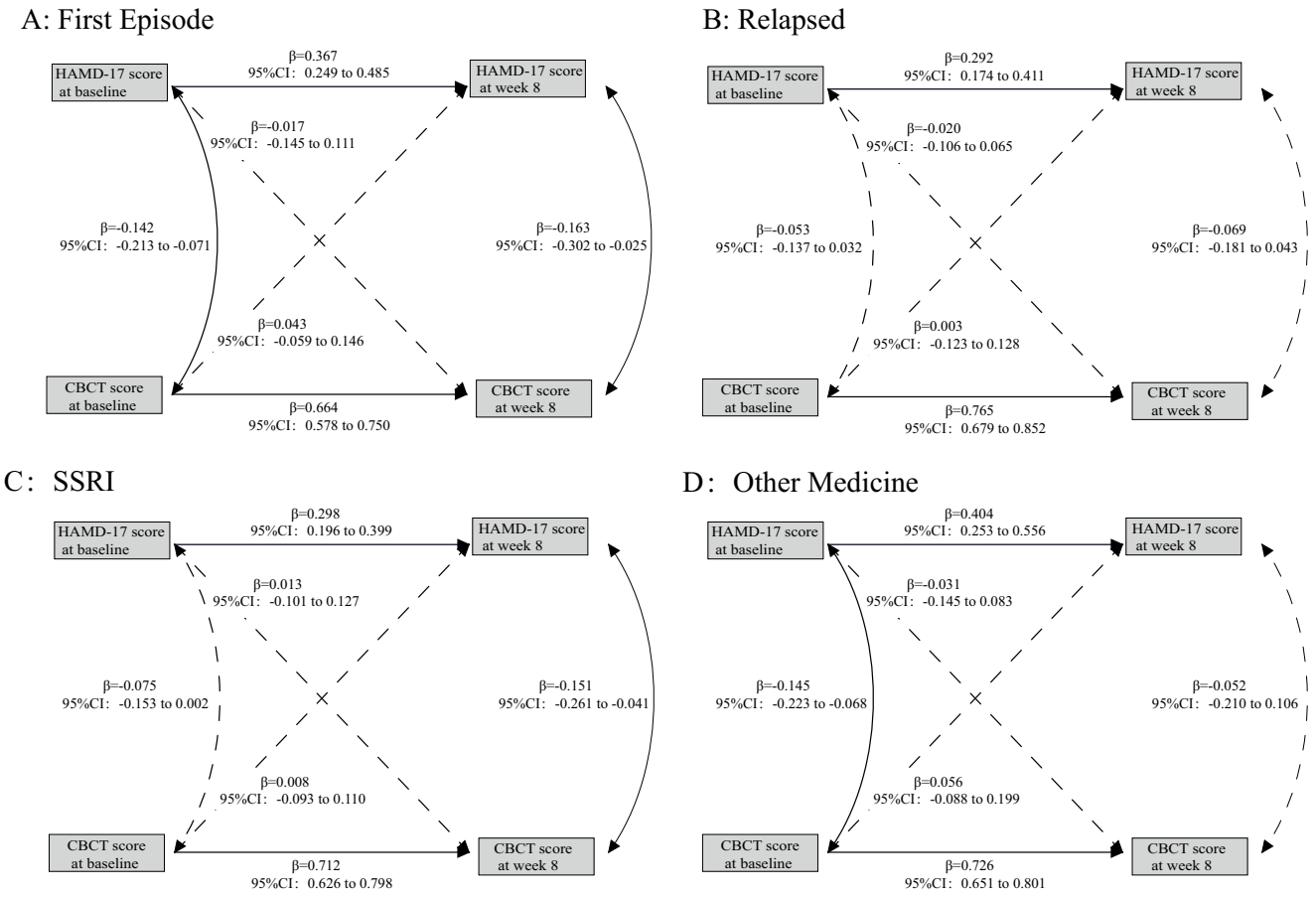

Figure 2. Cross-lagged panel models associations between depressive symptoms and objective cognition stratified by episode groups and treatments. *Note:* Standardized estimates with 95% confidence intervals are presented. Solid lines in the Cross-Lagged Panel Models indicate statistically significant standardized estimates, while dashed lines represent estimates that are not statistically significant. Patients taking medications other than SSRIs included 141 cases on SNRIs (Serotonin-Norepinephrine Reuptake Inhibitors), 23 cases on Mirtazapine, and 318 cases on other antidepressants, such as Bupropion and Trazodone. Additionally, 518 patients were on other psychiatric medications, including 191 on sedative-hypnotics, 348 on benzodiazepines, and 132 on non-benzodiazepines. Some patients were concurrently taking multiple medications.

including anxiety and depression, at various intervals (Brück et al., 2019). Similarly, a 2020 study on adolescents identified a strong relationship between improvements in subjective cognitive function and alleviation in depressive and anxiety symptoms over 3 months of treatment; also, the worsening of subjective cognitive function was associated with symptom exacerbation (Allott et al., 2020). Additionally, an analysis of data from 800 older adults from the 2021 Whitehall II study revealed a significant association between subjective cognitive complaints and depressive symptoms, both cross-sectionally and longitudinally (Topiwala et al., 2021).

These findings may be explained by cognitive models, particularly Beck's cognitive theory of depression (Beck, 1967). This theory suggests that cognitive biases, including negative self-assessment and attentional bias, contribute to the cognitive distortions prevalent in depression. Impaired cognitive control in the prefrontal cortex diminishes patients' ability to inhibit negative emotions, resulting in the sustained activation of negative self-referential schemas. This cycle perpetuates depressive symptoms and exacerbates anxiety through rumination (Disner, Beevers, Haigh, & Beck, 2011). Consequently, subjective cognitive distortions – especially negative self-assessments – may reflect these biased cognitive processes and predict the onset and progression of emotional symptoms (Kube et al., 2020). Therefore, the findings highlight the importance of subjective assessments of cognitive function in clinical practice. Moreover, interventions aimed at enhancing subjective cognitive function, such as cognitive–behavioral therapy (CBT) (Ng et al., 2023) and mindfulness training (van der Velden et al., 2023), may effectively address negative cognitive biases and improve emotional regulation. Cognitive control training, which focuses on tasks designed to enhance cognitive flexibility and executive function, may further assist in managing emotional symptoms (Koster et al., 2017).

At the same time, the finding that there was not a significant relationship between objective cognitive function and emotional symptoms was also consistent with previous findings. For instance, a randomized longitudinal study found that despite significant improvements in depressive symptoms following antidepressant treatment, deficits in key cognitive domains such as attention and verbal memory showed minimal improvement (Shilyansky et al., 2016). Additionally, a meta-analysis demonstrated that 73% of objective cognitive variables – particularly those related to processing speed, selective attention, working memory, verbal learning, and executive function – remained impaired in patients with remitted depression (Semkovska et al., 2019).

We found that the improvement of subjective cognitive function contributed more significantly to social functioning recovery

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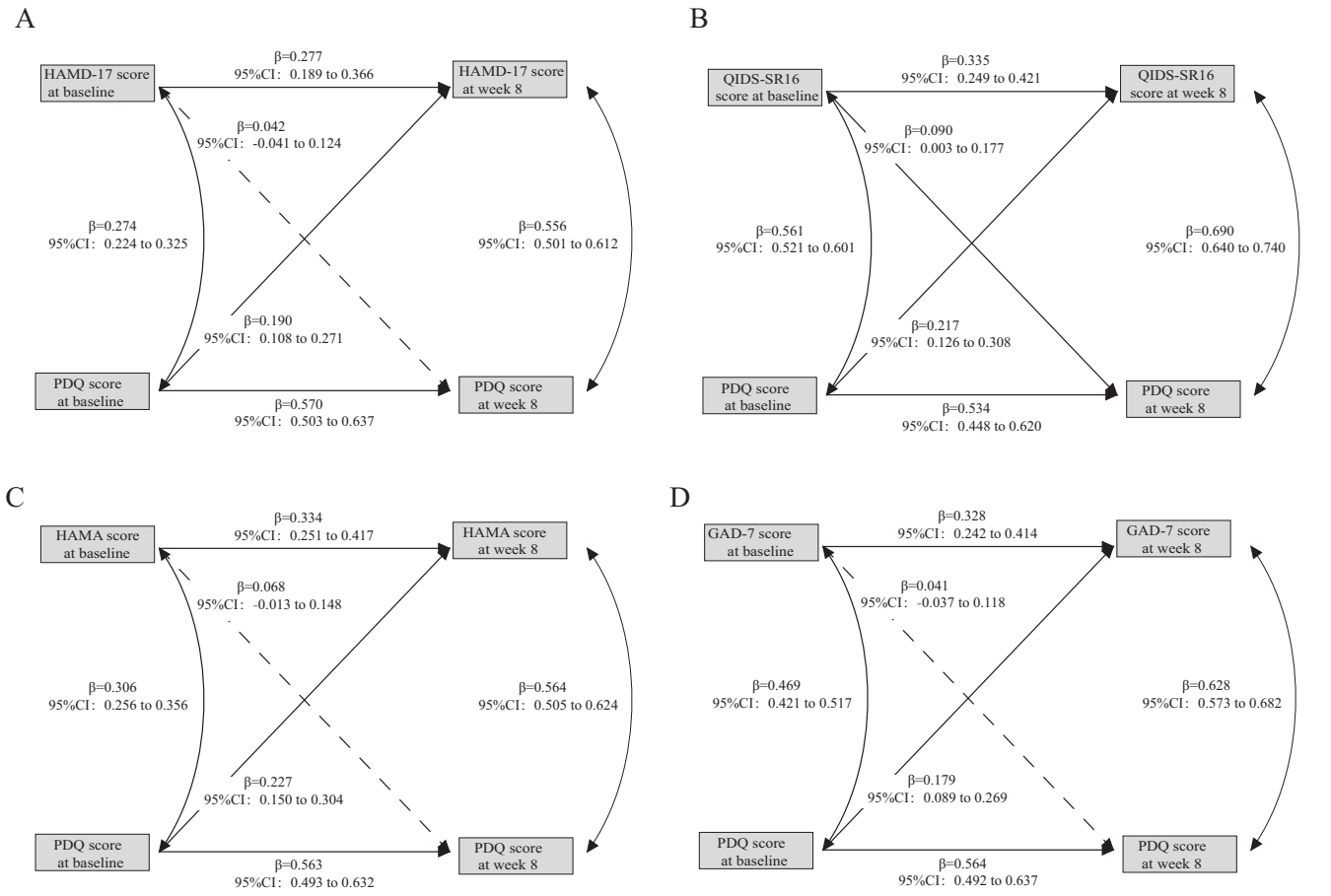

Figure 3. Cross-lagged panel models associations between depressive symptoms and subjective cognition stratified by episode groups and treatment types. *Note:* Standardized estimates with 95% confidence intervals are presented. Solid lines in the Cross-Lagged Panel Models indicate statistically significant standardized estimates, while dashed lines represent estimates that are not statistically significant. Patients taking medications other than SSRIs included 141 cases on SNRIs (Serotonin-Norepinephrine Reuptake Inhibitors), 23 cases on Mirtazapine, and 318 cases on other antidepressants, such as Bupropion and Trazodone. Additionally, 518 patients were on other psychiatric medications, including 191 on sedative-hypnotics, 348 on benzodiazepines, and 132 on non-benzodiazepines. Some patients were concurrently taking multiple medications.

than improvements in depressive symptoms. This finding was also consistent with the findings of recent studies. For instance, a 2020 study found that subjective cognitive impairments had a greater incremental effect on depressive symptoms and functional disability than objective cognitive function (Dhillon et al., 2020). Similarly, the 2021 PERFORM-J study found that subjective cognitive impairments were closely related to poorer psychosocial functioning and quality of life among patients with MDD, whereas objective cognitive function showed no significant correlation with psychosocial functioning or quality of life (Sumiyoshi et al., 2021). Additionally, the 2021 CAN-BIND-1 study revealed that the cognitive self-appraisals were strongly associated with the alleviation of depressive symptoms, better recovery of functioning, and enhancements in quality of life (Rnic et al., 2021). From the perspective of self-efficacy theory, enhancements in subjective cognitive function may boost patients' self-efficacy, leading to increased engagement in work, family, and social activities, thereby facilitating the restoration of social functioning (Ryan & Deci, 2000; Stanley & Maddux, 1986). Additionally, from a psychosocial perspective, improvements in subjective cognitive function may strengthen patients' resilience, enhancing their ability to regulate responses to stress and environmental challenges (Stover, Shulkin, Lac, & Rapp, 2024). In contrast, improvements in objective cognitive function may exert less influence on social functioning due to a disconnect between objective assessments and real-world demands (Howieson, 2019).

Strengths and limitations

This study's strength lies in its large, multicenter, and representative sample. First, as sex differences have been found in many aspects of MDD (Li et al., 2024), the composition of the study population might influence the study results. One strength of the current study was that the male-to-female ratio was ~1:2, consistent with patterns observed in psychiatric epidemiology studies (Bone, Lewis, & Lewis, 2020). Second, the participants exhibited diverse demographic and clinical characteristics, including varying education levels, employment statuses, marital statuses, illness episodes, and durations, which enhanced the generalizability of the findings. Third, cross-lagged panel models were employed to examine the temporal relationships between cognitive function and emotional symptoms in patients with MDD, providing insights into their directional influences and their impact on the restoration of social functioning. Fourth, cognitive evaluations were conducted in controlled laboratory or standardized settings, enhancing the accuracy and reliability of the evaluations. Nevertheless, several limitations must be considered when interpreting

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Table 2. Multiple linear regression analysis for improvement of social function

Variables	Estimation	Standard error	t	*P*	VIF
Model 1					
Intercept	1.445	1.32637	1.09	0.2765	0
Increase in the C-BCT score	−0.013	0.03875	−0.34	0.7364	1.02
Decrease in the HAMD–17 score*	0.567	0.05361	10.57	<.0001	1.02
Number of episodes	−0.094	0.21748	−0.43	0.6650	1.28
Overall duration of illness (month)	0.006	0.00670	0.82	0.4113	1.36
Age	−0.057	0.04006	−1.42	0.1561	1.08
Model 2					
Intercept	0.836	1.15325	0.72	0.4690	0
Decrease in the PDQ-D5 score*	0.551	0.07053	7.81	<.0001	1.21
Decrease in the HAMD–17 score*	0.384	0.05161	7.44	<.0001	1.22
Number of episodes	−0.111	0.20364	−0.54	0.5862	1.33
Overall duration of illness (month)	0.003	0.00582	0.52	0.6039	1.44
Age	−0.031	0.03493	−0.89	0.3765	1.09

Note: C-BCT, The Chinese Brief Cognitive Test; PDQ-D5, Perceived Deficits Questionnaire-Depression 5-item; HAMD-17, the 17-item Hamilton Depression Rating Scale; VIF, Variance Inflation Factor.
*Statistically significant variables ($P < 0.05$).
In Model 2, the R^2 for the decrease in the PDQ-D5 score was 0.196, and the R^2 for the decrease in the HAMD-17 score was 0.075.

these findings. First, although the study included follow-up data, most were collected over an 8-week period, and there was less data from week 52 available. This might have limited the analysis of long-term changes in cognitive, emotional, and functional outcomes. Second, PDQ-5 and C-BCT differ in their cognitive assessment dimensions. PDQ-5 focuses on functional cognitive experiences, including attention/concentration, prospective/retrospective memory, and planning ability ('Perceived Deficits Questionnaire – Depression, 5-item (PDQ-D-5)', 2016; Shi et al., 2017). Its items (e.g. 'difficulty organizing tasks') directly reflect subjective cognitive impairments in daily life, which are susceptible to core depressive symptoms such as motivation and negative cognitive bias. In contrast, C-BCT assesses fundamental neurocognitive functions, including information processing speed, working memory, and reasoning/problem-solving (Ye et al., 2022), which are primarily regulated by neurocircuitry efficiency and exhibit weaker dynamic associations with emotional states. Third, although CLPM allows for the examination of temporal predictive relationships between variables, its observational nature inherently limits causal inference. Unmeasured confounders, such as genetic predisposition and heterogeneity in environmental exposures, may still influence the observed associations. Therefore, our findings should be interpreted as reflecting the dynamic interplay between symptoms and cognition rather than definitive causal pathways. Future studies should employ prospective randomized controlled trials (RCTs) to further elucidate the causal relationship between depressive symptoms and cognitive function. Finally, the relatively young median age of our cohort (27.86 years) may limit the generalizability of our findings to older patients with MDD. Cognitive decline in older adults is often intertwined with neurodegenerative processes, potentially altering the relationship between subjective cognitive complaints and emotional symptoms compared with younger individuals. Future research will incorporate age-stratified analyses to assess the generalizability of our conclusions.

Conclusion

This study revealed that cognitive function predicted reductions in depressive symptoms. Also, subjective cognitive function contributed more significantly to the restoration of social functioning than objective cognitive function, even exceeding the influence of depressive symptoms. These findings highlighted the importance of improving subjective cognitive function to enhance the outcomes of patients with MDD. It is important to develop interventions aimed at achieving full recovery, which are based not only on reducing emotional symptoms but also on improving cognition, especially including patients' appraisal of their own cognitive functioning.

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