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# Longitudinal bidirectional association between psychosocial function and depression in Chinese patients with clinically remitted depression: a cross-lagged panel model analysis

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## Abstract

**Background** Major depressive disorder (MDD) imposes serious effect on patient's psychosocial function, which hinders the full recovery from the disease and increases the risk of recurrence, although the participants had achieved clinical remission. To understand the relationship between psychosocial function and depressive symptoms could facilitate recurrence prevention. Therefore, the purpose of this study was to analyze the relation among psychosocial function and BDI score in Chinese patients with clinically cured depression within 1 year follow-up.

**Methods** One hundred nineteen valid participants were assessed at baseline(t1), months 6(t2) and months 12(t3). Beck Depression Inventory-II (BDI-II) was used to assess depressive symptoms and indicate the possibility of depression level. Generic Quality of Life Inventory (GQOLI) was used to assessed the participants' psychosocial function(F), including body function (BF), psychological function (PF) and social function (SF). The application of a cross-lagged panel model (CLPM) approach revealed an association between BDI and psychosocial function.

**Result** The CLPM results showed total average score of psychosocial function have reciprocal influence on BDI score. The model analyzed by structural equation modeling satisfied all indices of goodness-of-fit (chi-square = 10.306, TLI = 0.959, CFI = 0.988 RMSEA = 0.115). And body function, psychological function, social function and BDI score also affect each other. Depressive symptoms and psychosocial function could predict scores of each other 6 months later. By comparing standardized cross-lagged path, only social function has a more pronounced impact on depressive symptoms, since the absolute effect of SFt1 → BDI t2 is larger than that of BDI t1 → SFt2 ( $a_1$  vs.  $b_1 = -.267$ ,  $SE = .108$ ,  $P < 0.05$ , 95%CI[-.485, -.063]) and the absolute effect of SFt2 → BDI t3 is larger than that of BDI t2 → SFt3 ( $a_2$  vs.  $b_2 = -.317$ ,  $SE = .096$ ,  $P < 0.01$ , 95%CI[-.508, -.129]).

**Conclusion** The current study showed a significant bidirectional association between depressive level and psychosocial function, and the social function exerted more effect on the depression.

**Keywords** Major depressive disorder, Clinical remission, Psychosocial function, Depression, Cross-lagged panel model

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## Introduction

The social and economic burden of major depressive disorder (MDD) is ever-increasing and it has become a worldwide public health problem [1]. While many individuals with MDD achieved clinically partial or complete remission, they still face some issues [2]. Apart from the typical symptoms of MDD such as negative mood, anhedonia, fatigue, self-dissatisfaction and impaired cognition, they may also experience some anxiety, irritability, pessimism, desperation, and impaired working functioning, etc. [3]. These symptoms would exist for a long time for others despite ongoing treatment [4], especially some mild typical depressive symptoms and few biological symptoms [5].

Apart from some symptoms the remitted individuals with MDD faced, numerous studies have suggested that they also faced persistent poor psychosocial function after remission, such as school, work, family, social interaction and recreation [6–8]. In other words, the recovery of psychosocial function presents a certain lag behind that of symptoms. Although no consensus of domains of psychosocial dysfunction has yet been reached, many recent studies have confirmed functional deficits in physical manifestation, cognition, occupational functioning, autonomy and global functioning [9, 10]. MDD may influence physical manifestation, including sleep difficulty, eating behaviors, sensory functions, etc. Remitted depressed individuals with MDD may continue to have sleep disorder because there are residual disturbances in sleep continuity, increase in REM density, decrease in sleep efficiency, and a shortened REM latency is likely to persist in some [11]. And even though having achieved remission, some eating problems still exist [12]. Significant differences in healthy controls and different depressive course groups were revealed, whereas differences between the depressed groups were found no statistically significant differences [12]. This may indicate that eating styles tend to be trait compared with state variables, and it would not change rapidly with mood. During remission period, improvement in sensory functions is not obvious. Some studies have found that there was no significant difference in scores of odour intensity, odour hedonics and somatic pain, such as musculoskeletal pain, gastro-intestinal pain and cardio respiratory pain, between current and remitted MDD [13, 14]. Moreover, several studies in remitted depression have reported the relationship between expressive suppression and lower affect [15, 16]. In remitted participants with MDD, compared with healthy controls, immediate emotional processing returned to normal levels but remained abnormal emotional regulation [17]. One study investigated implicit and explicit self-esteem

in remitted MDD, indicating no difference in implicit self-esteem but lower explicit self-esteem compared to healthy controls [18]. In addition, it is of no surprise that persons who experience MDD show worse performance in social function than those without MDD [19]. They reported disabilities in many domains, including household activities, interpersonal functioning occupational functioning and participation in society [19]. Individuals with MDD reported that in the past week, due to mental health conditions, there were 35.2% work hours with decreased work efficiency and 8% work hours missed [20]. Some researchers have reported that some individuals with MDD achieved clinical remission after treatment for that acute phase with antidepressants continue to present occupational impairment, for example reduction in hours missed [21]. This suggests that depressive symptoms decreased and patients experienced some improvement in psychosocial function during acute treatment. But it was hard to return premorbid functional levels or levels of normal healthy controls. During the follow-up period when achieving clinical remission, no significant increase in job performance was found [22]. That is to say, MDD reduces work productivity in both acute and remission phases. And residual psychosocial function impairment is associated with increasing risk of MDD recurrence [23]. Therefore, we should understand that participants may consider returning to normal functional level as equally important to relieving depressive symptoms [24].

During the remitted stage, depressive symptoms are associated with persistent problems in psychosocial function [25, 26]. However, the longitudinal relationship between psychosocial function and depressive symptoms is not fully understood. Even though direct evidence is lacking, there is indirect evidence to suggest that various factors of social function may predict depressive symptoms. Some studies confirmed that depressive symptom severity affects psychosocial function [27, 28]. Psychosocial disability is depending on state, which means that disability is manifest when individuals with MDD present depressive symptoms; disability increases when the severity of depressive symptoms increase [29, 30]. Thus, the prerequisite for disability is symptomatic remission [6]. Nevertheless, despite symptoms remission, disability continues to persist. Many studies have shown that although individuals with MDD continue to receive treatment, the rate of improvement in functioning tends to be slower compared to the improvement in depressive symptoms [31–33]. Therefore, some researchers believe that symptom remission fail to predict subsequent psychosocial functional improvement but improvement in functioning could significantly predict subsequent

symptom reduction. In other words, psychosocial function could be treated as a forward prediction in depressive symptoms [34]. These findings imply that the enhancements in functioning cannot be solely attributed to the improvements in depressive symptoms. Finally, some researchers believe that depressive symptoms and psychosocial function affect each other [35–38]. The relationship between depressive symptoms and psychosocial function is bidirectional, and it is mainly reflected in the following three aspects: social, occupational and physical [36]. Most studies have examined associations between depressive symptoms and psychosocial function in patients with acute MDD [33, 35, 39]. Therefore, we aimed to investigate the relationship in remitted MDD. And the hypothesis of this study is that depressive symptom reduction predicted subsequent psychosocial functional improvement, or vice versa.

## Method

### Participants and procedure

This study is a follow-up analysis of participants with clinically cured depression. The first follow-up was just after reaching clinical remission (marked time1, t1), which include 181 participants, with an average age of  $40.93 \pm 9.23$  years, including 71 males and 110 females, 68 first episode and 113 relapse. The second follow-up was conducted in 6 months after clinical remission (marked time 2, t2), which include 156 effective participants and 25 lost, including 56 males and 100 females, 57 first episode and 99 relapse. The third follow-up was conducted in 12 months after clinical remission (marked time 3, t3), there were 119 effective participants with an average age of  $39.50 \pm 9.75$  years, including 42 males and 77 females, 40 first episode and 79 relapse. All study procedures were approved by the Affiliated Brain Hospital of Nanjing Medical University Institutional Review Board, and participants participated with informed, voluntary, written consent.

### Measurements

Depressive symptoms were assessed by using Beck Depression Inventory-II (BDI-II). The BDI-II comprises a 21-item checklist designed to evaluate an individual's subjective feelings over the preceding seven days. The scoring range for each item spans from 0 to 3. Higher scores indicate greater severity of depression (<10, normal; 10–18, mild; 19–29, moderate; 30–63, severe) [40]. A sum of all items was computed, with higher score indicating higher levels of depressive symptoms. Good psychometric properties have been reported for the BDI-II as a reliable and valid measure of depressive symptoms among Chinese. The Chinese version of the BDI-II showed good reliability, with a Cronbach's  $\alpha$  of

0.87 [41]. In the present sample, the BDI-II demonstrated good internal consistency across all time points: Time 1 ( $\alpha=0.917$ ), Time 2 ( $\alpha=0.904$ ), and Time 3 ( $\alpha=0.926$ ). We choose BDI because it is self-assessment, which allows participants to assess and respond to their depression overall. It can better reflect overall subjective feelings, including the residual symptoms and patients' overall depression level.

Psychosocial function(F) was assessed by using the Generic Quality of Life Inventory (GQOLI-74). The scale covers three aspects of body function (BF), psychological function (PF), social function (SF), material living condition and overall assessment [32]. The higher the score is, the better the function is. BF (items 11–30) includes sleep and energy, physical discomfort, eating function, sexual function and motor and sensory function; PF (items 31–50) includes mental stress, negative emotions, positive emotions, cognitive function and self-esteem; SF (item 51–70) includes social support, interpersonal communication, work/study, leisure activities and marriage and family [42]. Given their irrelevance to our evaluation, the ten items pertaining to material living conditions have been excluded from the assessment to alleviate respondent burden. Good psychometric properties have been reported as a reliable and valid measure among Chinese. In the present sample, the GQOLI-74 demonstrated good internal consistency across all time points: Time 1 ( $\alpha=0.934$ ), Time 2 ( $\alpha=0.915$ ), and Time 3 ( $\alpha=0.921$ ). The questionnaire has high reliability and validity, and the test–retest reliability of the questionnaire is 0.84–0.93 [32].

### Statistical analyses

SPSS28.0 was used for descriptive statistics of all variables. Descriptive and correlation analyses were conducted in SPSS and  $p < 0.05$  was considered to be statistically significant. The  $\chi^2$  and T tests showed that the missing participants were compared with those who completed 3 follow-up visits: there was no significant differences among gender( $\chi^2(1) = .084, p = .772$ ), relapses( $\chi^2(1) = 3.154, p = .076$ ), age( $t(179) = 1.278, p = 0.203$ ), BDI t1 score( $t(179) = -0.186, p = 0.853$ ), body function t1 score ( $t(179) = 0.580, p = 0.563$ ), psychological function t1 score ( $t(179) = -0.534, p = 0.594$ ), social function t1 score ( $t(179) = 0.135, p = 0.893$ ). And total average t1 score of psychosocial function( $t(179) = 0.067, p = 0.947$ ). It indicates that there is no structural loss in participants. To avoid possible errors caused by data interpolation [43], 119 subjects who completed three follow-up visits were included for latent growth model and cross-lagged panel model [44, 45]. Mplus8.8 was used to conduct cross-lagged panel model [45, 46] of maximum likelihood method to examine the relationship between

BDI score and psychosocial function. The CLPM for BDI score and three kinds of functions (included total average score of psychosocial function) were established respectively, and standardized cross-lagged coefficients were compared.

### Common method bias

Firstly, on the advice of the relevant research, mainly from the aspects of procedure control, including the high reliability and validity of maturity scale as measuring tools, protect the anonymity of participants, appropriate transform different questionnaire instructions and scoring methods, part of the entries using reverse score, etc., and to strictly control the source of the common method bias. Secondly, Harman single-factor test was used: in the 3 measurements, the total number of factors with eigenvalues greater than 1 was 21, 22, and 21, respectively, and the variation explained by the first factor was 16.56%, 16.98%, and 17.74%, respectively, all lower than the critical standard of 40%, indicating that the common method bias was not obvious.

## Results

### Descriptive statistics

All descriptive statistics across 3 follow-up period are summarized in Table 1. BDI score decreased from t1 to t3, three kinds of functions and total average score of psychosocial function risen from t1 to t3. There was a

moderate correlation between BDI score, three kinds of functions and total average score of psychosocial function at three time point.

### Cross-lagged panel models

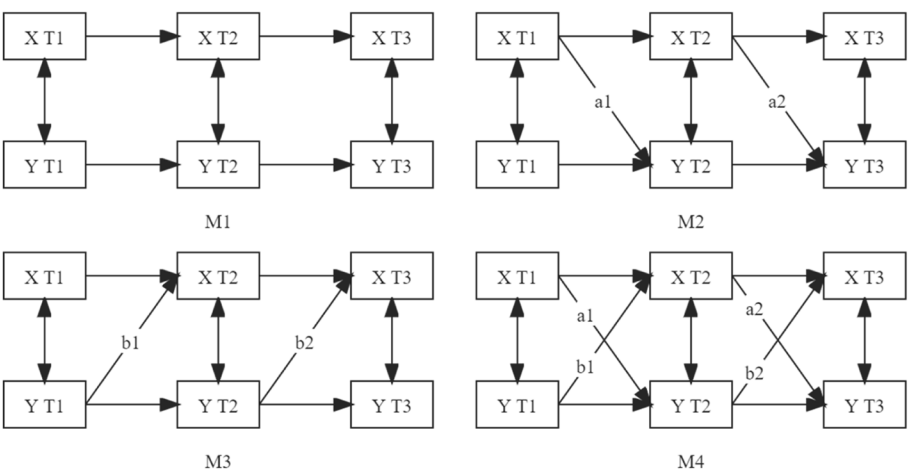
Firstly, the relationship between total average score of psychosocial function and BDI score was analyzed. CLPM controls the autoregressive effect of each variable by setting stability coefficient and is considered the best method to test the direction of "pure" effect between variables [43, 46, 47], which can be used to understand the overall degree of prediction of one variable on another. When CLPM is used to explore the relationship between variables, four models need to be tested (Fig. 1): (1) Baseline model M1 including only autoregressive effects; (2) On the basis of M1, construct model M2, in which X points to Y; (3) Add competition model M3 where Y points to X path on the basis of M1; (4) The full model M4 including all paths of M1, M2 and M3. The model M4 can be expressed as Eq. (1) and (2), of which X represent functions, Y represent BDI score. Based on the principles and practice of structure equation modeling (Fifth Edition), the  $\chi^2/DF$  of the model fitting index is less than 5, the CFI and TLI are greater than 0.9, the larger the better, and the RMSEA and SRMR are less than 0.08, the smaller the better, which belongs to the situation of good fitting [43].

$$X_{t+1} = \beta_x X_t + \beta_y Y_t + \varepsilon_{x(t+1)} \quad (1)$$

**Table 1** Descriptive statistics

Variables	1.BDI t1	2.BDI t2	3.BDI t3	4.BF t1	5.BF t2	6.BF t3	7.PF t1	8.PF t2	9.PF t3	10.SF t1	11.SF t2	12.SF t3	13.Ft t1	14.Ft t2	15.Ft t3
2	.672**	1													
3	.529**	.601**	1												
4	-.533**	-.522**	-.578**	1											
5	-.523**	-.525**	-.571**	.727**	1										
6	-.482**	-.532**	-.574**	.579**	.802**	1									
7	-.588**	-.564**	-.543**	.549**	.549**	.551**	1								
8	-.627**	-.548**	-.640**	.595**	.581**	.523**	.570**	1							
9	-.548**	-.594**	-.689**	.581**	.627**	.618**	.633**	.661**	1						
10	-.528**	-.709**	-.596**	.753**	.734**	.687**	.576**	.600**	.636**	1					
11	-.536**	-.634**	-.640**	.732**	.750**	.700**	.575**	.612**	.628**	.710**	1				
12	-.540**	-.629**	-.654**	.786**	.776**	.751**	.621**	.581**	.682**	.694**	.883**	1			
13	-.637**	-.684**	-.660**	.887**	.770**	.695**	.829**	.680**	.711**	.882**	.774**	.810**	1		
14	-.644**	-.648**	-.705**	.782**	.891**	.772**	.646**	.841**	.731**	.779**	.892**	.849**	.847**	1	
15	-.588**	-.657**	-.719**	.726**	.828**	.893**	.676**	.662**	.864**	.757**	.825**	.907**	.829**	.881**	1
M	4.00	3.02	2.45	66.01	70.38	71.31	65.53	68.80	70.38	66.27	69.63	69.90	65.94	69.60	70.53
SD	2.84	3.02	2.67	12.44	10.96	12.49	12.52	10.87	12.09	10.41	9.76	11.17	10.20	9.20	10.58
Skew	-0.070	0.395	1.326	-0.043	0.106	-0.178	-0.212	-0.542	-0.470	-0.410	-0.643	-0.267	-0.091	-0.374	-0.313
Kurtosis	-1.329	-1.357	0.621	-0.666	-0.646	-0.093	-1.085	-0.270	-0.726	-0.249	0.189	0.221	-0.654	-0.537	-0.429

\* $p < .05$ , \*\* $p < .01$ ; t1, assessed at just after reaching clinical remission; t2, assessed in 6 months after clinical remission; t3, assessed in 12 months after clinical remission. BDI, average score of Beck Depression Inventory-II; BF, average score of body function; PF, average score of psychological function; SF, average score of social function; F, average score of psychosocial function



**Fig. 1** Cross-lagged panel models

$$Y_{t+1} = \beta_y Y_t + \beta_x X_t + \varepsilon_{y(t+1)}$$

(2)

**CLPM- psychosocial function**

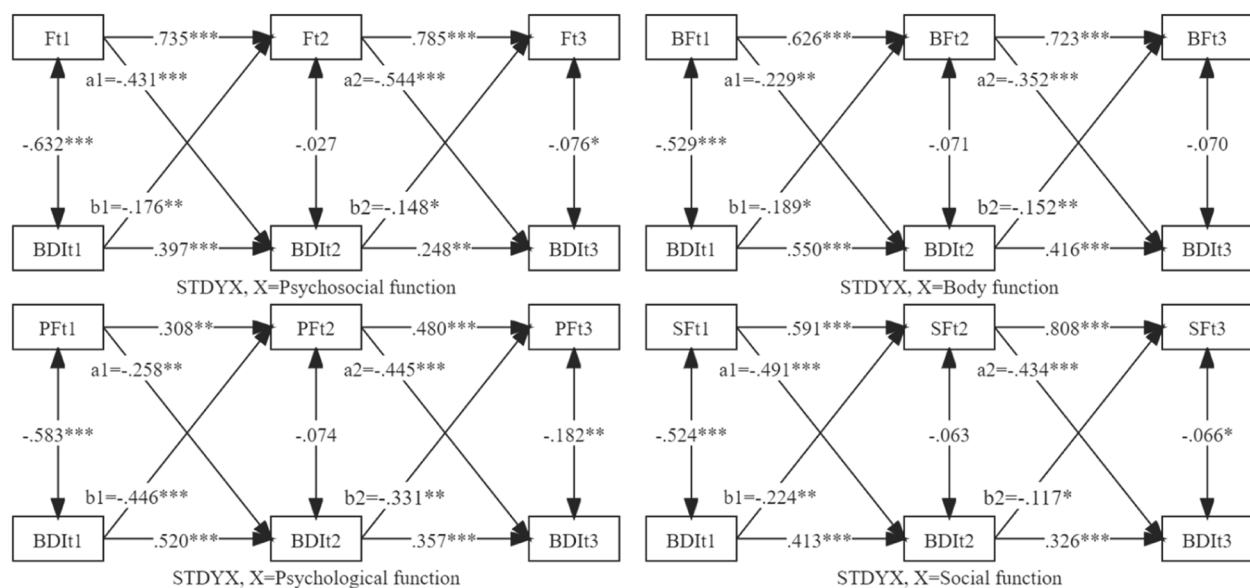
The model fit is shown in Table 2. M4 was selected as the optimal model according to model fit (Fig. 2). Ft1 significantly predicted(a1=−0.431, SE=0.092, *P*<0.001) BDIt2, and Ft2 significantly predicted(a2=−0.544, SE=0.091, *P*<0.001) BDIt3. BDIt1 significantly predicted (b1=−0.176, SE=0.066, *P*<0.01) Ft2, and BDIt2 significantly predicted

(b2=−0.148, SE=0.067, *P*<0.05) Ft3. Comparison of standardized cross-lagged path with bias-corrected bootstrap 5000 samples shows that: a1 vs. b1=−0.256, SE=0.104, *P*<0.05, 95%CI [−0.470, −0.067], indicating that the absolute effect of Ft1→BDIt2 is larger than that of BDIt1→Ft2; a2 vs. b2=−0.396, SE=0.121, *P*<0.01,95%CI [−0.624, −0.149], indicating that the absolute effect of Ft2→BDIt3 is larger than that of BDIt2→Ft3. In conclusion, psychosocial function and BDI score have reciprocal influence, and function has larger influence on BDI score.

**Table 2** Model fit of CLPMs

Model	$\chi^2$	df	$\chi^2/\text{df}$	CFI	TLI	RMSEA	SRMR
X=Psychosocial function							
M1	94.978	8	11.872	.838	.716	.302	.188
M2	25.237	6	4.206	.964	.916	.164	.046
M3	74.310	6	12.385	.873	.703	.309	.144
M4	10.306	4	2.577	.988	.959	.115	.014
X=Body function							
M1	49.344	8	6.168	.890	.807	.208	.156
M2	20.105	6	3.351	.962	.912	.141	.064
M3	34.167	6	5.695	.925	.825	.199	.110
M4	7.739	4	1.935	.990	.965	.089	.024
X=Psychological function							
M1	107.180	8	13.398	.709	.491	.323	.212
M2	56.334	6	9.389	.853	.656	.266	.129
M3	52.364	6	8.727	.864	.683	.255	.115
M4	13.444	4	3.361	.972	.903	.141	.038
X= Social function							
M1	93.355	8	11.669	.818	.682	.299	.202
M2	19.784	6	3.297	.971	.932	.139	.068
M3	74.730	6	12.455	.854	.659	.310	.143
M4	6.292	4	1.573	.995	.983	.069	.020





**Fig. 2** M4 models

#### CLPM- body function

Model M1-M4 was analyzed successively, and the model fit showed that M4 was the optimal model (Table 2; Fig. 2). BFt1 significantly predicted ( $a1 = -0.229$ ,  $SE = 0.074$ ,  $P < 0.01$ ) BDI2t, and BFT2 significantly predicted ( $a2 = -0.352$ ,  $SE = 0.081$ ,  $P < 0.001$ ) BDI3t. BDI1t significantly predicted ( $b1 = -0.189$ ,  $SE = 0.079$ ,  $P < 0.05$ ) BFT2, and BDI2t significantly predicted ( $b2 = -0.152$ ,  $SE = 0.057$ ,  $P < 0.01$ ) BFT3. Comparison of standardized cross-lagged path with bias-corrected bootstrap 5000 samples shows that:  $a1$  vs.  $b1 = -0.040$ ,  $SE = 0.106$ ,  $P > 0.05$ , 95%CI  $[-0.254, 0.165]$ , indicating that there is no significant differences between effect of BFt1  $\rightarrow$  BDI2t and BDI1t  $\rightarrow$  BFT2;  $a2$  vs.  $b2 = -0.200$ ,  $SE = 0.102$ ,  $P = 0.051$ , 95%CI  $[-0.405, -0.004]$ , indicating that there is no significant differences between effect of BFT2  $\rightarrow$  BDI3t and BDI2t  $\rightarrow$  BFT3. In conclusion, body function and BDI score have reciprocal influence.

#### CLPM- psychological function

Model M1-M4 was analyzed successively, and the model fit showed that M4 was the optimal model (Table 2; Fig. 2). PFt1 significantly predicted ( $a1 = -0.258$ ,  $SE = 0.097$ ,  $P < 0.01$ ) BDI2t, and PFT2 significantly predicted ( $a2 = -0.445$ ,  $SE = 0.083$ ,  $P < 0.001$ ) BDI3t. BDI1t significantly predicted ( $b1 = -0.446$ ,  $SE = 0.086$ ,  $P < 0.001$ ) PFT2, and BDI2t significantly predicted ( $b2 = -0.331$ ,  $SE = 0.095$ ,  $P < 0.01$ ) PFT3. Comparison of standardized cross-lagged path with bias-corrected bootstrap 5000 samples shows that:  $a1$  vs.  $b1 = 0.188$ ,  $SE = 0.115$ ,  $P > 0.05$ , 95%CI  $[-0.039, 0.411]$ , indicating that there is

no significant differences between effect of PFt1  $\rightarrow$  BDI2t and BDI1t  $\rightarrow$  PFT2;  $a2$  vs.  $b2 = -0.114$ ,  $SE = 0.139$ ,  $P > 0.05$ , 95%CI  $[-0.393, 0.145]$ , indicating that there is no significant differences between effect of PFT2  $\rightarrow$  BDI3t and BDI2t  $\rightarrow$  PFT3. In conclusion, psychological function and BDI score have reciprocal influence.

#### CLPM- social function

Model M1-M4 was analyzed successively, and the model fit showed that M4 was the optimal model (Table 2; Fig. 2). SFt1 significantly predicted ( $a1 = -0.491$ ,  $SE = 0.080$ ,  $P < 0.001$ ) BDI2t, and SFT2 significantly predicted ( $a2 = -0.434$ ,  $SE = 0.086$ ,  $P < 0.001$ ) BDI3t. BDI1t significantly predicted ( $b1 = -0.224$ ,  $SE = 0.079$ ,  $P < 0.01$ ) SFT2, and BDI2t significantly predicted ( $b2 = -0.117$ ,  $SE = 0.055$ ,  $P < 0.05$ ) SFT3. Comparison of standardized cross-lagged path with bias-corrected bootstrap 5000 samples shows that:  $a1$  vs.  $b1 = -0.267$ ,  $SE = 0.108$ ,  $P < 0.05$ , 95%CI  $[-0.485, -0.063]$ , indicating that the absolute effect of SFt1  $\rightarrow$  BDI2t is larger than that of BDI1t  $\rightarrow$  SFT2;  $a2$  vs.  $b2 = -0.317$ ,  $SE = 0.096$ ,  $P < 0.01$ , 95%CI  $[-0.508, -0.129]$ , indicating that the absolute effect of SFT2  $\rightarrow$  BDI3t is larger than that of BDI2t  $\rightarrow$  SFT3. In conclusion, social function and BDI score have reciprocal influence, and social function has larger influence on BDI score.

#### Discussion

The aim of this study is to examine the possible longitudinal association between depressive level and psychosocial function in remitted MDD, including respective

relationships between depressive symptoms and total psychosocial function, body function, psychological function, social function. We found that overall psychosocial function affect each other. In our study, depressive symptom at baseline could predict psychosocial function in 6 months, and depressive symptom at 6 months could predict psychosocial function at 12 months. In this longitudinal one-year follow-up study, it implies that depressive symptoms and psychosocial function could predict scores of each other 6 months later. Consistent with previous research results, some researchers found association between depression severity and psychosocial functional impairment at two years of follow-up visits [48, 49]. It implies that depressive symptom reduction contributes to improvement in psychosocial function, and vice versa.

This finding correlates with previous studies, which found regardless of treatment (pharmacotherapy or psychotherapy), improvement in depressive symptoms is reported to contribute to functional restoration [33, 35]. Likewise, the severity of depressive symptoms directly correlates with the impairment of functional capacity in individuals diagnosed with major depressive disorder, potentially severely limiting the daily activities of individuals, causing a decline in self-care abilities and diminished productivity in work and study endeavors [50]. A bidirectional relationship exists between social-interpersonal functioning and depressive disorders. Depressive symptoms may lead to functional impairment through various mechanisms. For instance, neurocognitive dysfunction and the decrement in self-esteem resulting from interpersonal rejection exhibit significant correlations with depressive disorders [51]. Also, we confirm that functional improvement predicts symptom relief [35]. Additionally, some studies have determined some individuals with MDD experienced functional deficits before a depressive episode and these individuals are susceptible to MDD [52, 53]. Because they are vulnerable to psychosocial deficits, it is difficult to turn to normal level of psychosocial function. Therefore, lower psychosocial function has a potential influence on symptom improvement and residual symptom would cause functional impairment in turn.

Body functioning and depressive symptoms affect each other. We found that depressive symptoms and psychosocial function predicted scores of each other 6 months later in 1 year. This finding is consistent with past research [54, 55]. Improvement in depressive symptoms is positively correlated with physical condition and perceived overall health [56, 57]. Still not clear how well the depressive severity of various depression subtypes

predicts postmorbid functioning. However, to a certain degree, improvement in depressive symptoms is associated with better physical function [58]. After remission, depressive symptoms exhibited adverse effects on physical functioning, causing a functional recovery lag [29]. Recent research shows that among studied physical conditions, heart disease and chronic pain conditions (backache, neck pain, chronic headache and multiple pains) are mostly associated with depressive symptoms [59]. Depressive symptoms might alter the level of social and physical functioning and cause a lagging behind in recovery of functioning [60]. And depressive symptoms may affect the level of body functioning, causing a lag in functional recovery [60].

We also found the relationship of mutual influence of psychological functioning and depressive symptoms. This suggests that depression symptoms improvement can promote psychological functioning improvement, and vice versa. Psychological functioning includes cognitive functioning, negative emotions, mental stress, self-esteem, etc. Firstly, cognitive impairments had a significant positive correlation with depressive symptoms [57, 61]. Several researchers have found severe depression was associated with poor neurocognitive performance, such as executive function and memory [62]. Secondly, MDD is characterized by depressed mood and negative emotions [61]. Studies have found reductions in negative emotions precedes and predicts reduction in depression severity [63]. Additionally, severity of depression has a strong association with rumination [64]. And individuals with MDD reported disrupted emotional regulation and processing. When faced sad, scary and entertained situations, their emotional responses tend to be less sensitive and flat [65]. Thirdly, in remitted MDD, if they experience major events, such as divorce or financially threatening job loss, indicate shorter time and greater possibility to relapse or recurrence [66]. Therefore, even controlling for chronic stress (e.g., chronic financial difficulties, ongoing family conflict), depressive symptoms bring about increased risk of recurrence [66]. Finally, decreased self-esteem is also one of symptoms of MDD [61]. Many studies show low self-esteem has a negative correlation with high depression scores, and low self-esteem could prospectively predict depression [67, 68].

There is a mutual influence of social function and depressive symptoms, and social function has a more pronounced impact on depressive symptoms. Researchers have found depressive symptoms can not only directly predict productive activities, but also could predict through the mediating role of dysfunctional attitudes [69]. Some studies confirmed reduction of depressive symptoms is particularly associated with improvements

in social function [59]. Several researchers have found that improvement in social-interpersonal functioning from acute phase cognitive therapy could be maintained 32 months [51, 70]. And consistent with our study, they also found social-interpersonal dysfunction could predict depressive symptoms and MDD relapse or recurrence [51, 70]. In previous research, after acute phase treatment, functioning got improved and lasted for 2 years [38]. However, continuation treatment in remitted phase cannot bring additional improvement and they found social interpersonal functioning and deteriorated before depression relapses/recurrence [51]. According to Lewinsohn's behavioral theory of depression, social skill deficits were considered as one of possible causes of MDD [71]. And a dyadic analysis showed poor marital adjustment could predict more severe depressive symptoms [72]. Therefore, social function is a risk of importance for severity of depressive symptoms. On the other hand, restoration of social function would reduce depressive symptoms and prevent relapse, which has a protective effect on mental health [73–75]. A review has shown that social contacts was found to be a predictor of remission in MDD. As a result, preventing loneliness and social isolation becomes a common and growing problem in individuals with MDD [73]. Moreover, social support has a greatly positive impact on reducing depressive symptoms and suicidal ideation. Several researches have reported when individuals are faced stress, social support, especially the number of support providers, may help enhance resilience [74]. Tariq et al. confirmed that perceived social support significantly predicted depressive symptoms and higher levels of social support relate to lower depression scores [75]. Additionally, they also found that friends' support and family's support have the most significant association with the level of depression [75]. Hence, medical staff could help remitted MDD improve personal and interpersonal functioning, which provides strong protection just in case increase in depressive symptoms and relapse or recurrence [34].

Psychosocial function may provide substantial protection for MDD participants against relapse/recurrence [76]. Moreover, when predicting recurrence, psychosocial function, especially social function, seems to be more essential. Therefore, complete remission of MDD should not totally equal to clinical symptom remission. When MDD participants achieve clinical remission standard, psychosocial function, especially social function, should be one of important focuses of therapeutic intervention, including pharmacotherapy and psychotherapy [30]. As a result, in settings with limited resources, interventions targeting to improve social function should be considered to relieve depressive symptoms and restore functioning [76].

The improvement of psychosocial function has significant clinical significance for individuals with MDD. Researches indicate that individuals with MDD who experience early enhancements in psychosocial functioning demonstrate increased rates of long-term symptomatic remission when compared to those undergoing gradual improvements [38, 77]. Such early enhancement not only assists in forecasting individuals' long-term symptomatic remission but may also serve to efficiently guard against MDD relapse and recurrence by bolstering their social adaptability [77]. Additionally, variations in psychosocial functioning have been established as a critical predictor of long-term symptomatic remission in individuals with MDD, notably, individuals with minimal and gradual improvements in social and interpersonal functioning demonstrate slower rates of depressive improvement [51].

Effective therapeutic strategies, including psychotherapy and pharmacotherapy, have been shown to enhance psychosocial functioning, which in turn promotes the rehabilitation of participants with depression. Studies have indicated that effective antidepressant therapy can markedly enhance the psychosocial functioning of individuals with depression [38, 77]. Administration of noradrenergic antidepressants, such as nortriptyline, and selective serotonin reuptake inhibitors (SSRIs), like escitalopram, has been demonstrated to enhance psychosocial functioning [77]. Both cognitive therapy (CT) and mindfulness-based cognitive therapy (MBCT) have been empirically validated to enhance psychosocial functioning [38, 78]. Additionally, in clinical settings, it is imperative to tailor treatment strategies to the individual needs of participants to optimize psychosocial functioning and prevent relapse.

A strength of this article is the longitudinal repeated measure design. We obtained 3 data from the same patient at baseline, 6 and 12 months after clinical remission. Cross-lagged models examined bidirectional causality between symptoms and psychosocial function, which presented symptoms and psychosocial function at one time point would predict subsequent symptoms and psychosocial function at the next time point.

This study also has some limitations. First, it is uncertain whether these results generalize to other individuals and settings. Because there may exist certain limitations in the process of sample selection, encompassing the extensive age span as well as the authentic educational backgrounds. Therefore, further studies with a larger sample of representative individuals with MDD are necessary to confirm our present findings.

Second, the statistical test power of the employed methodology could be constrained. Given that the study population consists of clinically remitted MDD,



the dropout rates are relatively high. Furthermore, the research mainly examines the correlation between the severity of depressive symptoms and psychosocial function. Consequently, the CLPM is deemed more appropriate for our analytical purposes. Alternatively, other methodological approaches, like random intercept cross-lagged modeling (RI-CLPM), could potentially provide higher statistical test power and incorporate both between-individual and within-individual variability. In future studies, we need to gather more data, especially from large, multi-center studies, to better understand these factors.

Third, there is no unified tool to evaluate psychosocial function and GQOLI-74 is greatly onerous for patients due to many entries [32]. This emphasizes the necessity of promoting unified standardized assessment tools in this field. Fourth, there remain no gold standard of recovery of psychosocial function so that we fail to identify the normal range of functioning recovery [32]. Future studies ought to prioritize the development and dissemination of a consensus-based gold-standard assessment instrument. Future research needs to put more emphasis on creating and spreading a standardized, top-tier assessment tool. Finally, the present research only assessed psychosocial function through self-report. Consequently, it is subjective evaluation results rather than objective performance. Therefore, other objective methods to assess psychosocial function, such as behavioral observations, should be used in future studies.

To conclude, in this study, we found psychosocial function and depressive symptoms affect each other. Particularly, social function has stronger impact on symptoms. Increased severity of depressive symptoms predicts greater psychosocial dysfunction and lower levels of functioning. In turn, impaired social function heralds increased depressive symptoms and recurrence. It is helpful to prevent recurrence and reduce disease burden to explore the relationship between psychosocial function and depressive symptoms in remitted MDD. In particular, attending to the restoration of social function among individuals with MDD can yield significant dividends in terms of preventing relapse and recurrence of depressive symptoms.

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#### Authors' contributions

Zhang Ning, and Ma Hui conceptualized and designed the study. Teng Changjun, Ma Hui, and Qiao Huifen, Yang Hua collected and provided patients and kept follow-up. Teng Changjun, Hao Yang contributed to the data analysis. Huang Wenyan, Teng Changjun, and Ma Hui wrote and revised the manuscript. All authors contributed to the interpretation of the results and revised the important intellectual content. All authors read and approved the final manuscript.

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#### Data availability

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

#### Declarations

##### Ethics approval and consent to participate

This research was approved by the Medical Research Ethics Committee of Nanjing Brain Hospital affiliated to Nanjing Medical University (No. 2015-KY002) in accordance with the World Medical Association Declaration of Helsinki. All participants signed informed consent after receiving a full description of the study, an explanation of its purpose, and information about the confidentiality of the data.

##### Consent for publication

Not applicable.

##### Competing interests

The authors declare no competing interests.

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#### References

- Lafleur MF, Baruch P, Grondin S, Lafleur MF. Psychosocial and neurocognitive functioning in unipolar and bipolar depression: A 12-month prospective study. *Psychiat Res*. 2012;196(1):145–53. <https://doi.org/10.1016/j.psychres.2011.09.013>.
- Kupka RW, Altshuler LL, Nolen WA. Three times more days depressed than manic or hypomanic in both bipolar I and bipolar II disorder. *Bipolar Disord*. 2007;5(9):531–5.
- Fava GA, Fabbri S, Sonino N. Residual symptoms in depression: an emerging therapeutic target. *Prog Neuro-Psychoph*. 2002;26(6):1019–27. [https://doi.org/10.1016/s0278-5846\(02\)00226-9](https://doi.org/10.1016/s0278-5846(02)00226-9).
- Nierenberg AA, Husain MM, Trivedi MH, Fava M, Warden D, Wisniewski SR, Miyahara S, Rush AJ. Residual symptoms after remission of major depressive disorder with citalopram and risk of relapse: a STARD report. *Psychol Med*. 2010;40(1):41–50. <https://doi.org/10.1017/S0033291709006011>.
- Paykel ES. Remission and residual symptomatology in major depression. *Psychopathology*. 1998;31(1):5–14. <https://doi.org/10.1159/000029018>.
- Iancu SC, Wong YM, Rhebergen D, van Balkom AJLM, Batelaan NM. Long-term disability in major depressive disorder: a 6-year follow-up study. *Psychol Med*. 2020;50(10):1644–52. <https://doi.org/10.1017/S0033291719001612>.
- Knight MJ, Air T, Baune BT. The role of cognitive impairment in psychosocial functioning in remitted depression. *J Affect Disorders*. 2018;235:129–34. <https://doi.org/10.1016/j.jad.2018.04.051>.
- Baune BT, Miller R, McAfoose J, Johnson M, Quirk F, Mitchell D. The role of cognitive impairment in general functioning in major depression. *Psychiat Res*. 2010;176(2–3):183–9. <https://doi.org/10.1016/j.psychres.2008.12.001>.
- Cambridge OR, Knight MJ, Mills N, Baune BT. The clinical relationship between cognitive impairment and psychosocial functioning in major depressive disorder: A systematic review. *Psychiat Res*. 2018;269:157–71. <https://doi.org/10.1016/j.psychres.2018.08.033>.
- McIntyre RS, Lee Y. Cognition in major depressive disorder: a "Systemically Important Functional Index" (SIFI). *Curr Opin Psychiatr*. 2016;29(1):48–55. <https://doi.org/10.1097/YCO.0000000000000221>.
- Rush AJ, Erman MK, Giles DE, Schlessner MA, Carpenter G, Vasavada N, Roffwarg HP. Polysomnographic Findings in Recently Drug-Free and Clinically Remitted Depressed Patients. *Arch Gen Psychiatry*.

- 1986;43(9):878–84. <https://doi.org/10.1001/archpsyc.1986.01800090068009>.
12. Nadine PGP, Mariska B, van Tatjana S. Eating styles in major depressive disorder: Results from a large-scale study. *J Psychiatr Res.* 2018;97:38–46. <https://doi.org/10.1016/j.jpsychires.2017.11.003>.
  13. Clepce M, Gossler A, Reich K. The relation between depression, anhedonia and olfactory hedonic estimates—A pilot study in major depression. *Neurosci Lett.* 2010;471(3):139–43. <https://doi.org/10.1016/j.neulet.2010.01.027>.
  14. Jaracz J, Karolina G, Krystyna J. Unexplained Painful Physical Symptoms in Patients with Major Depressive Disorder: Prevalence, Pathophysiology and Management *Cns Drugs.* 2016;30(4):293–304. <https://doi.org/10.1007/s40263-016-0328-5>.
  15. Visted E, Birkeland JVM. Emotion Regulation in Current and Remitted Depression: A Systematic Review and Meta-Analysis. *Front Psychol.* 2018;9:756. <https://doi.org/10.3389/fpsyg.2018.00756>.
  16. Liu DY, Thompson RJ. Selection and implementation of emotion regulation strategies in major depressive disorder: an integrative review. *Clin Psychol Rev.* 2017;57:183–94. <https://doi.org/10.1016/j.cpr.2017.07.004>.
  17. Rozemarijn SVK, Jan-Bernard CME. Neural basis of positive and negative emotion regulation in remitted depression. *Randomized Controlled Trial.* 2022;34: 102988. <https://doi.org/10.1016/j.nicl.2022.102988>.
  18. Danique S, Janna NV, van Iris O. Implicit and explicit self-esteem in remitted depressed patients. *J Behav Ther Exp Psy.* 2016;54:301–6. <https://doi.org/10.1016/j.jbtep.2016.10.006>.
  19. Greden JF. Workplace depression: personalize, partner, or pay the price. *Am J Psychiatry.* 2013;170(6):578–81. <https://doi.org/10.1176/appi.Ajp.2012.13030382>.
  20. Beck A, Crain AL, Solberg LI, Unutzer J, Glasgow RE, Maciosek MV, Whitebird R. Severity of depression and magnitude of productivity loss. *Ann Fam Med.* 2011;9(4):305–11. <https://doi.org/10.1370/afm.1260>.
  21. Trivedi MH, Morris DW, Wisniewski SR, Lesser I, Nierenberg AA, Daly E, Kurian BT, Gaynes BN, Balasubramani GK, Rush AJ. Increase in work productivity of depressed individuals with improvement in depressive symptom severity. *Am J Psychiatry.* 2013;170(6):633–41. <https://doi.org/10.1176/appi.Ajp.2012.12020250>.
  22. Adler DA, McLaughlin TJ, Rogers WH, Chang H, Lapitsky L, Lerner D. Job performance deficits due to depression. *Am J Psychiatry.* 2006;163(9):1569–76. <https://doi.org/10.1176/ajp.2006.163.9.1569>.
  23. Hardeveld F, Spijker J, De Graaf R, Nolen WA, Beekman ATF. Prevalence and predictors of recurrence of major depressive disorder in the adult population. *Acta Psychiatr Scand.* 2010;122(3):184–91. <https://doi.org/10.1111/j.1600-0447.2009.01519.x>.
  24. Zimmerman M, McGlinchey JB, Posternak MA, Friedman M, Attiullah N, Boerscu D. How should remission from depression be defined? The depressed patient's perspective. *Am J Psychiatry.* 2006;163(1):148–50. <https://doi.org/10.1176/appi.Ajp.163.1.148>.
  25. Fava Giovanni A, Ruini Chiara, Carlotta B. The concept of recovery in major depression. *Psychol Med.* 2007;37(03):307–17. <https://doi.org/10.1017/S0033291706008981>.
  26. Zimmerman M, Posternak MA, Chelminski I. Heterogeneity among depressed outpatients considered to be in remission. *Compr Psychiatr.* 2007;48(2):113–7. <https://doi.org/10.1016/j.comppsy.2006.10.005>.
  27. IsHak WW, Balayan K, Bresee C, Greenberg JM, Fakhry H, Christensen S, Rapaport MH. A descriptive analysis of quality of life using patient-reported measures in major depressive disorder in a naturalistic outpatient setting. *Qual Life Res.* 2013;22(3):585–96. <https://doi.org/10.1007/s11136-012-0187-6>.
  28. Engel L, Chen G, Richardson J, Mihalopoulos C. The impact of depression on health-related quality of life and wellbeing: identifying important dimensions and assessing their inclusion in multi-attribute utility instruments. *Qual Life Res.* 2018;27(11):2873–84. <https://doi.org/10.1007/s11136-018-1936-y>.
  29. Judd LL, Akiskal HS, Zeller PJ, Paulus M, Leon AC, Maser JD, Endicott J, Coryell W, Kunovac JL, Mueller TI, Rice JP, Keller MB. Psychosocial disability during the long-term course of unipolar major depressive disorder. *Arch Gen Psychiatry.* 2000;57(4):375–80. <https://doi.org/10.1001/archpsyc.57.4.375>.
  30. Hirschfeld RM, Dunner DL, Keitner G, Klein DN, Koran LM, Kornstein SG, Markowitz JC, Miller I, Nemeroff CB, Ninan PT, Rush AJ, Schatzberg AF, Thase ME, Trivedi MH, Borian FE, Crits-Christoph P, Keller MB. Does psychosocial functioning improve independent of depressive symptoms? a comparison of nefazodone, psychotherapy, and their combination. *Biol Psychiatr.* 2002;51(2):123–33. [https://doi.org/10.1016/S0006-3223\(01\)01291-4](https://doi.org/10.1016/S0006-3223(01)01291-4).
  31. Renner F, Cuijpers P, Huibers M. The effect of psychotherapy for depression on improvements in social functioning: a meta-analysis. *Psychol Med.* 2014;44(14):2913–26. <https://doi.org/10.1017/S0033291713003152>.
  32. Wang Z, Qu H, Zhong J, Han Y, Wan C, Wang H, Yang H, Lu S, Diao K, Zhang N, Ma H. Restoration of psychosocial functioning in remitted major depressive disorder patients: A 1-year longitudinal study. *Compr Psychiatr.* 2020;102: 152204. <https://doi.org/10.1016/j.comppsy.2020.152204>.
  33. Lin C, Chou L, Chen M, Chen C. The relationship between symptom relief and functional improvement during acute fluoxetine treatment for patients with major depressive disorder. *J Affect Disorders.* 2015;182:115–20. <https://doi.org/10.1016/j.jad.2015.04.022>.
  34. Vittengl JR, Clark LA, Thase ME. Relations of Shared and Unique Components of Personality and Psychosocial Functioning to Depressive Symptoms. *J Pers Disord.* 2017;5(32):577–602. <https://doi.org/10.1521/pedi.2017.31.313>.
  35. Dunn TW, Vittengl JR, Clark LA, Carmody T, Thase ME, Jarrett RB. Change in psychosocial functioning and depressive symptoms during acute-phase cognitive therapy for depression. *Psychol Med.* 2012;42(2):317–26. <https://doi.org/10.1017/S0033291711001279>.
  36. Patrick E, McKnight TB. The importance of functional impairment to mental health outcomes: a case for reassessing our goals in depression treatment research. *Clin Psychol Rev.* 2009;3(29):243–59. <https://doi.org/10.1016/j.cpr.2009.01.005>.
  37. Hammer-Helmich L, Haro JM, Jönsson B, Tanguy Melac A, Di Nicola S, Chollet J, Milea D, Rive B, Saragoussi D. Functional impairment in patients with major depressive disorder: the 2-year PERFORM study. 2018;14:239–49. <https://doi.org/10.2147/NDT.S146098>.
  38. Vittengl JR, Clark LA, Jarrett RB. Deterioration in psychosocial functioning predicts relapse/recurrence after cognitive therapy for depression. *J Affect Disorders.* 2009;112(1):135–43. <https://doi.org/10.1016/j.jad.2008.04.004>.
  39. Lin CH, Yang WC. The Relationship between Symptom Relief and Psychosocial Functional Improvement during Acute Electroconvulsive Therapy for Patients with Major Depressive Disorder. *Int J Neuropsychoph.* 2017;7(20):538–45. <https://doi.org/10.1093/ijnp/pyx022>.
  40. Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An Inventory for Measuring Depression wide variety of psychiatric rating scales.4,15. *Archives of General Psychiatry.* 1961;4:561–71. <https://doi.org/10.1001/archpsyc.1961.01710120031004>.
  41. Lu ML, Che HH, Chang SW. Reliability and Validity of the Chinese Version of the Beck Depression Inventory-II. *Taiwanese J Psychiatry.* 2002;16(4):301–9.
  42. Zhou Y, Zhou R, Li W, Lin Y, Yao J, Chen J, Shen T. Controlled trial of the effectiveness of community rehabilitation for patients with schizophrenia in Shanghai. *China Shanghai Arch Psychiatry.* 2015;27(3):167–74. <https://doi.org/10.11919/j.issn.1002-0829.215026>.
  43. Kline RB. Principles and practice of structural equation modeling. 4th ed. New York: Guilford Press; 2016.
  44. Curran PJ, Obeidat K, Losardo D. Twelve frequently asked questions about growth curve modeling. *J Cogn Dev.* 2010;11(2):121–36. <https://doi.org/10.1080/15248371003699969>.
  45. Fitzmaurice GM, Laird NM, Ware JH. Applied longitudinal analysis. Hoboken, NJ: John Wiley & Sons; 2012.
  46. Wang J, Wang X. Structural equation modeling: Applications using Mplus. Hoboken, NJ: John Wiley & Sons; 2019.
  47. Preacher KJ. Advances in mediation analysis: a survey and synthesis of new developments. *Annu Rev Psychol.* 2015;66:825–52. <https://doi.org/10.1146/annurev-psych-010814-015258>.
  48. Kim JM, Chalem Y, Nicola DS. A crosssectional study of functional disabilities and perceived cognitive dysfunction in patients with major depressive disorder in South Korea: the PERFORM-K study. *Psychiat Res.* 2016;30:353–61. <https://doi.org/10.1016/j.psychres.2016.01.022>.
  49. Iosifescu DV. The relation between mood, cognition and psychosocial functioning in psychiatric disorders. *Eur Neuropsychopharm.* 2012;22:S499–504. <https://doi.org/10.1016/j.euroneuro.2012.08.002>.

50. Lin CH, Yen YC, Chen MC, Chen CC. Depression and Pain Impair Daily Functioning and Quality of Life in. *J Affect Disord*. 2014;166:173–8. <https://doi.org/10.1016/j.jad.2014.03.039>.
51. Vittengl JR, Clark LA, Thase ME, Jarrett RB. Longitudinal social-interpersonal functioning among higher-risk responders to acute-phase cognitive therapy for recurrent major depressive disorder. *J Affect Disorders*. 2016;199:148–56. <https://doi.org/10.1016/j.jad.2016.04.017>.
52. Ormel J, Oldehinkel AJ, Nolen WA. Psychosocial disability before, during, and after a major depressive episode: a 3-wave population-based study of state, scar, and trait effects. *Arch Gen Psychiatry*. 2004;61(4):387–92. <https://doi.org/10.1001/archpsyc.61.4.387>.
53. Buist-Bouwman MA, Ormel J, Graaf RD. Functioning after a major depressive episode: complete or incomplete recovery. 2004;82(3):363–71. <https://doi.org/10.1016/j.jad.2004.02.007>.
54. Rhebergen D, Beekman AT, Graaf RD, Nolen WA, Spijker J, Hoogendijk WJ, Penninx BW. The three-year naturalistic course of major depressive disorder, dysthymic disorder and double depression. *J Affect Disorders*. 2009;115(3):450–9. <https://doi.org/10.1016/j.jad.2008.10.018>.
55. Scott KM, Bruffaerts R, Tsang A, Ormel J, Alonso J, Angermeyer MC, Benjet C, Bromet E, de Girolamo G, de Graaf R, Gasquet I, Gureje O, Haro JM, He Y, Kessler RC, Levinson D, Mneimneh ZN, Oakley Browne MA, Posada-Villa J, Stein DJ, Takeshima T, Von Korff M. Depression–anxiety relationships with chronic physical conditions: Results from the World Mental Health surveys. *J Affect Disorders*. 2007;103(1–3):113–20. <https://doi.org/10.1016/j.jad.2007.01.015>.
56. Rhebergen D, Beekman AT, de Graaf R, Nolen WA, Spijker J, Hoogendijk WJ, Penninx BW. Trajectories of recovery of social and physical functioning in major depression, dysthymic disorder and double depression: A 3-year follow-up. *J Affect Disorders*. 2010;124(1–2):148–56. <https://doi.org/10.1016/j.jad.2009.10.029>.
57. Toyoshima K, Inoue T, Baba T, Masuya J, Ichiki M, Fujimura Y, Kusumi I. Associations of Cognitive Complaints and Depressive Symptoms with Health-Related Quality of Life and Perceived Overall Health in Japanese Adult Volunteers. *Int J Environ Res Public Health*. 2021;18(18):9647. <https://doi.org/10.3390/ijerph18189647>.
58. Simon GE, Von Korff M, Elizabeth L. Clinical and functional outcomes of depression treatment in patients with and without chronic medical illness. *Psychol Med*. 2005;35(2):271–9. <https://doi.org/10.1017/S0033291704003071>.
59. Evans DL, Charney DS, Lewis L, Golden RN, Gorman JM, Krishnan KRR, Nemeroff CB, Bremner JD, Carney RM, Coyne JC, Delong MR, Frasurre-Smith N, Glassman AH, Gold PW, Grant I, Gwyther L, Ironson G, Johnson RL, Kanner AM, Katon WJ, Kaufmann PG, Keefe FJ, Ketter T, Laughren TP, Leserman J, Lyketsos CG, McDonald WM, McEwen BS, Miller AH, Muselman D, O'Connor C, Petitto JM, Pollock BG, Robinson RG, Roose SP, Rowland J, Sheline Y, Sheps DS, Simon G, Spiegel D, Stunkard A, Sunderland T, Tibbits P, Valvo WJ. Mood Disorders in the Medically Ill: Scientific Review and Recommendations. *Biol Psychiat*. 2005;58(3):175–89. <https://doi.org/10.1016/j.biopsych.2005.05.001>.
60. Judd LL, Akiskal HS. Delineating the Longitudinal Structure of Depressive Illness: Beyond Clinical Subtypes and Duration Thresholds I. *Pharmacopsychiatry*. 2000;33(1):3–7. <https://doi.org/10.1055/s-2000-7967>.
61. Otte C, Gold SM, Penninx BW, Pariante CM, Etkin A, Fava M, Mohr DC, Schatzberg AF. Major depressive disorder. *Nat Rev Dis Primers*. 2016;2(1):16065. <https://doi.org/10.1038/nrdp.2016.65>.
62. Tam CW, Lam LC. Cognitive Function, Functional Performance and Severity of Depression in Chinese Older Persons with Late-onset Depression. *East Asian Arch Psychiatry*. 2012;22(1):12–7. <https://doi.org/10.3969/j.issn.1020-7639.2012.01.003>.
63. Arslanoglou E, Banerjee S, Pantelides J, Evans L, Kiosses DN. Negative Emotions and the Course of Depression During Psychotherapy in Suicidal Older Adults With Depression and Cognitive Impairment. *Am J Geriatr Psychiatry*. 2019;27(12):1287–95. <https://doi.org/10.1016/j.jagp.2019.08.018>.
64. Kang Y, Gruber J. Harnessing happiness? Uncontrollable positive emotion in bipolar disorder, major depression, and healthy adults. *Emotion*. 2013;13(2):290–301. <https://doi.org/10.1037/a0030780>.
65. Rottenberg J, Kasch KL, Gross JJ. Sadness and Amusement Reactivity Differentially Predict Concurrent and Prospective Functioning in Major Depressive Disorder. *Emotion*. 2002;2(2):135–46. <https://doi.org/10.1037/1528-3542.2.2.135>.
66. Harkness KL, Theriault JE, Stewart JG, Bagby RM. Acute and chronic stress exposure predicts 1-year recurrence in adult outpatients with residual depression symptoms following response to treatment. *Depress Anxiety*. 2014;31(1):1–8. <https://doi.org/10.1002/da.22177>.
67. Sowislo JF, Orth U. Does low self-esteem predict depression and anxiety? A meta-analysis of longitudinal studies. *Psychol Bull*. 2013;139(1):213–40. <https://doi.org/10.1037/a0028931>.
68. Orth U, Robins RW, Roberts BW. Low self-esteem prospectively predicts depression in adolescence and young adulthood. *J Pers Soc Psychol*. 2008;95(3):695–708. <https://doi.org/10.1037/0022-3514.95.3.695>.
69. Wood-Ross C, Tran T, Milanovic M, Jokic R, Milev R, Bowie CR. Neurocognition and Depressive Symptoms have Unique Pathways to Predicting Different Domains of Functioning in Major Depressive Disorder. *The Canadian Journal of Psychiatry*. 2023;68(4):241–8. <https://doi.org/10.1177/07067437221133375>.
70. Vittengl JR, Clark LA, Jarrett RB. Improvement in social-interpersonal functioning after cognitive therapy for recurrent depression. *Psychol Med*. 2004;34(4):643–58. <https://doi.org/10.1017/S0033291703001478>.
71. Segrin CG. Depressive disorders and interpersonal processes. In: Horowitz LM, Strack S, editors. *Handbook of interpersonal psychology: Theory, research, assessment, and therapeutic interventions*. Hoboken, NJ: John Wiley & Sons; 2010. p. 425–448.
72. Locke TF, Newcomb MD. Psychosocial outcomes of alcohol involvement and dysphoria in women: a 16-year prospective community study. *J Stud Alcohol*. 2003;64(4):531–46. <https://doi.org/10.15288/jsa.2003.64.531>.
73. Solmi M, Cortese S, Vita G, De Prisco M, Radua J, Dragioti E, Kohler-Forsberg O, Madsen NM, Rohde C, Eudave L, Aymerich C, Pedruzo B, Rodriguez V, Rosson S, Sabe M, Hojlund M, Catalan A, de Luca B, Fornaro M, Ostuzzi G, Barbui C, Salazar-de-Pablo G, Fusar-Poli P, Correll CU. An umbrella review of candidate predictors of response, remission, recovery, and relapse across mental disorders. *Mol Psychiatry*. 2023;28(9):3671–87. <https://doi.org/10.1038/s41380-023-02298-3>.
74. Chang YH, Yang CT, Hsieh S. Social support enhances the mediating effect of psychological resilience on the relationship between life satisfaction and depressive symptom severity. *Sci Rep-Uk*. 2023;13(1):4818. <https://doi.org/10.1038/s41598-023-31863-7>.
75. Tariq A, Beihai T, Abbas N, Ali S, Yao W, Imran M. Role of Perceived Social Support on the Association between Physical Disability and Symptoms of Depression in Senior Citizens of Pakistan. *Int J Env Res Pub He*. 2020;17(5):1485. <https://doi.org/10.3390/ijerph17051485>.
76. Vittengl JR, Jha MK, Minhajuddin A. Quality of life after response to acute-phase cognitive therapy for recurrent depression. *J Affect Disord*. 2021;278:218–25. <https://doi.org/10.1016/j.jad.2020.09.059>.
77. Jha MK, Minhajuddin A, Greer TL, Carmody T, Rush AJ, MH T. Early Improvement in Psychosocial Function Predicts Longer-Term Symptomatic Remission in Depressed Patients. *Plos One*. 2016;11(12):e167901. <https://doi.org/10.1371/journal.pone.0167901>.
78. Segal ZV, Dimidjian S, Beck A, Boggs JM, Vanderkruik R, Metcalf CA, Gallop A, Felder JN, Levy J. Outcomes of Online Mindfulness-Based Cognitive Therapy for Patients With Residual Depressive Symptoms. *Jama Psychiatry*. 2020;77(6):563. <https://doi.org/10.1001/jamapsychiatry.2019.4693>.

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