



Original Article

The vicious cycle of depressive symptoms and disability in older adults

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ABSTRACT

Background: Unidirectional researches have suggested a correlation between depressive symptoms and disability, but it remains uncertain whether this association is bidirectional.

Methods: The study is based on the 1st-10th waves of the NHATS (National Health and Aging Trends Study). The subset A and subset B included 3,459 and 3,801 samples, respectively. A random-intercept cross-lagged panel model was employed to explore the bidirectional dynamic relationship between depressive symptoms and physical function indicators, including BADL (basic ADL), IADL (instrumental ADL), and ADL (a combination of BADL and IADL).

Results: An increase in depressive symptoms led to a decline in physical function at all levels, and vice versa. When older adults experienced poorer physical function than usual, their subsequent depressive symptoms were stronger (BADL, $\beta = 0.082$, $p < 0.0001$; IADL, $\beta = 0.072$, $p < 0.001$; ADL, $\beta = 0.098$, $p < 0.0001$). Conversely, an increase in earlier depressive symptoms resulted in a future decline in physical function (BADL, $\beta = 0.042$ – 0.057 , $p < 0.05$; IADL, $\beta = 0.048$, $p < 0.05$; ADL, $\beta = 0.061$, $p < 0.01$).

Conclusions: This study is the first to reveal a mutually reinforcing spiral effect between increased depressive symptoms and declines in any level of physical function. These findings highlight the importance of prevention strategies guided by a unity of mental-physical approach, offering a new perspective for the coordinated management of mental and physical health, and providing scientific evidence for policymaking and resource allocation.

Keypoints

- With aging, depressive symptoms increase, and physical function declines in older adults.
- Depressive symptoms and physical decline mutually reinforce each other, forming a vicious cycle.
- Prevention needs to consider integrated management guided by a unity of mental-physical approach.

1. Introduction

Depressive symptoms [1] and disability [2,3] impose significant negative impacts on mental and physical health in later life, respectively, and represent two major challenges in public health. With the aging global population, these issues amplify the severity of mental and physical health consequences, including comorbidities and mortality [4–6], thereby exerting considerable pressure on healthcare systems and social resources.

Epidemiological studies have demonstrated an association between depressive symptoms and disability [7]. Depressive symptoms may lead

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to disability [8–12], while disability, in turn, may increase the risk of depressive symptoms [13–16], indicating that both conditions mutually reinforce one another. Investigating the underlying mechanisms of these interrelationships could inform the development of targeted intervention strategies to mitigate the prevalence of mental and physical health issues.

Despite these two studies have developed in parallel, research on the bidirectional association between elevated depressive symptoms and physical function decline remains limited. One study provides evidence for a bidirectional lagged longitudinal association [17], while the other does not [18]. The limitations of these findings may be influenced by the constraints of the statistical methods used.

Previous studies have explored bidirectional relationships (i.e., the interaction between X and Y) using Structural Equation Modeling (SEM) [18] and Cross-Lagged Panel Model (CLPM) [17] methods. By controlling for autoregressive effects ($X_{t-1} \rightarrow X_t$; $Y_{t-1} \rightarrow Y_t$) to ensure temporal stability, researchers can examine the magnitude and direction of cross-lagged effects to understand the interplay between the two variables and clarify how the influence changes over time [19]. However, these methods are unable to capture within-individual changes, making it impossible to address whether individuals with depressive symptoms above the mean at time t are more likely to have poor activities of daily living (ADL) ability at time $t + 1$, and vice versa. When between-individual stable trait effects and within-individual fluctuating state effects are intertwined, distinguishing within-individual mechanisms from between-individual differences becomes challenging, which may lead to erroneous conclusions. Considering the temporal and situational fluctuations in depressive symptoms and disability, we believe it is necessary to properly decompose the variances of longitudinal effects into stable and changeable parts, to establish genuine lag effects occurring within individuals. Therefore, in this study, we used the Random Intercept Cross-Lagged Panel Model (RI-CLPM) to separate the stable trait-like effects at between-person and the fluctuating state-like effects within-person [20].

It is worth noting that prior studies have often used composite scales of Basic ADL (BADL) and Instrumental ADL (IADL) to assess disability. While the composite scale can enhance the scope and sensitivity of measurement [21], BADL and IADL exhibit inherent heterogeneity [22], underscoring the necessity of separate evaluations. BADL represents foundational functional activities essential for daily living, while IADL encompasses more advanced and context-specific tasks. A key distinction is that BADL involves necessary activities that individuals generally strive to perform independently [7]. In contrast, declines in IADL may be overlooked because elderly people often cease engaging in specific tasks, such as financial management, even before experiencing functional impairment [7]. Furthermore, Hoogendijk et al. argue that IADL reflects early-stage functional decline, whereas BADL signifies advanced-stage decline with greater severity [23]. Declines in BADL also require more intensive healthcare interventions. Understanding the relationship between depressive symptoms and declines in different ADL levels provides critical insights for the development of precise and targeted healthcare policies [24,25].

Thus, this study utilizes RI-CLPM along with detailed disability assessment indicators BADL, IADL and a composite indicator ADL (BADL + IADL) to explore the true bidirectional dynamic relationship between depressive symptoms and various levels of physical function decline. The findings aim to provide more precise insights for health policy formulation and contribute to improving the mental and physical health of older adults.

2. Methods

2.1. Study population

The National Health and Aging Trends Study (NHATS) is a nationally representative cohort study designed to investigate multiple aspects of

function and disability in later life [26]. Funded by the National Institute on Aging (NIA) of the National Institutes of Health (NIH), the study collects comprehensive data through face-to-face interviews with Medicare beneficiaries aged 65 and older residing in the United States. NHATS began in 2011 and follows a longitudinal design with annual follow-up waves. In 2011, the study recruited 8,245 participants using a stratified multistage sampling design, achieving a baseline response rate of 71% (8,245/11,637) [27]. The study protocol was approved by the Institutional Review Board at Johns Hopkins University. Trained interviewers conducted interviews in participants' homes. To assess the temporal consistency of our findings across different follow-up periods, we divided the follow-up waves into two alternating subsets: waves 1, 2, 3, 4 and 5 were grouped as subset A, while waves 6, 7, 8, 9 and 10 were grouped as subset B. Further information about NHATS can be found at (<http://www.nhats.org/>).

2.2. Measurement of depressive symptoms

Depressive symptoms was measured using the Patient Health Questionnaire-2 (PHQ-2). Higher scores on the PHQ-2 indicate more pronounced depressive symptoms.

2.3. Measurement of ADL

BADL includes five essential activities: eating, bathing, toileting, dressing, and transferring. IADL encompasses cooking, shopping, laundry, managing finances, and medication management. For each item, the response is coded based on whether the participant performs the task independently without difficulty: “no difficulty” (0) or “difficulty” (1). The total scores for BADL and IADL range from 0 to 5, with higher scores indicating greater difficulty in performing these tasks and worse physical function.

2.4. Covariates

Covariates in this study included demographic characteristics and health conditions. Demographic characteristics included age and gender (male or female). Health status was assessed based on the presence of the following comorbidities: self-reported health conditions, heart disease, hypertension, arthritis, osteoporosis, diabetes, lung disease, stroke, dementia or Alzheimer's disease, and cancer. Participants were classified into two categories according to their health status: no comorbidities, comorbidities.

2.5. Statistical analysis

All descriptive statistics were implemented using R 4.2.0. The Spearman correlation was used to assess the relationship between the depressive symptoms and ADLs. The RI-CLPM was constructed to separate between-person and within-person effects (Fig. 1). Intra-class correlation (ICC) was calculated to quantify the proportion of variance in depressive symptoms that could be attributed to within-person and between-person variations across the three ADLs. The root mean square error of approximation (RMSEA) and the comparative fit index (CFI) were used to assess the fit of the RI-CLPM model.

We apply the latent factor model to construct the latent variables of the random intercept term for depressive symptoms and each ADL measurement, respectively. Each loading was constrained to 1 to ensure proper scaling of the latent variables. The correlation between random intercepts was used to reflect the stable difference at between-person level. The first-order autoregressive models were used to explain the effect of each latent variable at one wave on its corresponding latent variable at the subsequent wave. The cross-lagged effects were used to characterize the bidirectional relationships between latent variables, accounting for both immediate and lagged effects.

Three RI-CLPMs were constructed to test the robustness of the

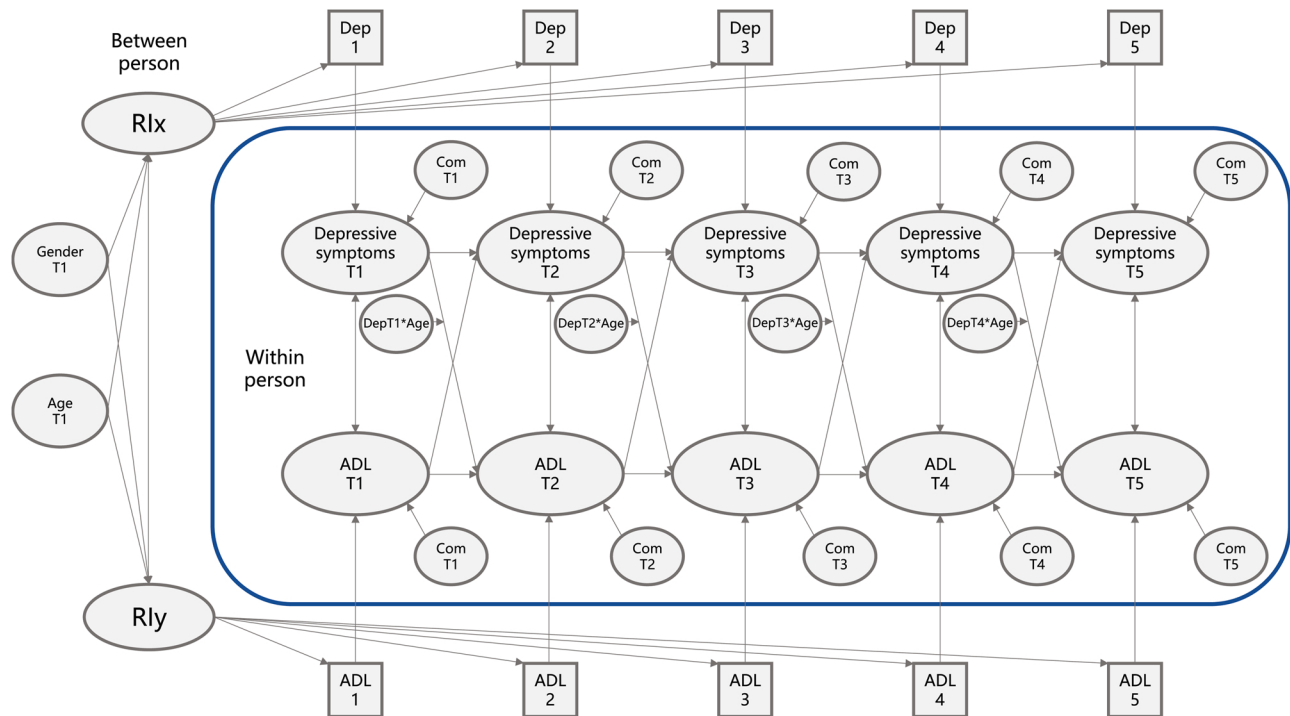


Fig. 1. Random intercept cross-lagged panel model (Model 3) of depressive symptoms and ADL across five time points.
Note: Model 3 = Model with Age Interaction + Covariates (Gender, Age, Comorbidities); Dep = depressive symptoms; ADL = activities of daily living; bp = between-person; wp = within-person; Com = Comorbidity; Rlx = person-specific trait factor of depressive symptoms; Rly = person-specific trait factor of disability.

findings. Model 1 was the baseline model without adjustments (Fig. S1). Model 2 extended Model 1 by including interaction terms between age and depressive symptoms at T_n to test whether age moderated the cross-lagged effects from T_n to T_{n+1} (Fig. S2). Model 3 further adjusted for covariates, including sex and age at the between-person level, and time-varying comorbidities at the within-person level (Fig. 1).

3. Results

3.1. Descriptive statistics and model fitting results

The baseline characteristics of the participants are summarized in Table 1 and further illustrated in Supplementary Figures S3 and S4. The subset A included 3,459 participants (age = 76.12 ± 7.17 years, female = 59.32% at the baseline) and the subset B included 3,801 participants (age = 77.26 ± 6.76 years, female = 58.51% at the second wave of follow-up). As the follow-up period progressed, participants experienced increased depressive symptoms, declining physical function, and a rise in comorbidities.

Fig. 2 illustrated the Spearman correlation. A significant autocorrelation was observed in depressive symptoms. The results revealed significant pairwise correlations between BADL, IADL, and ADL at different follow-up periods, with the strongest autocorrelation observed for ADLs and the weakest for BADLs. All three levels of ADLs were significantly negatively correlated with depressive symptoms, with BADL showing the strongest correlation and IADL showing the weakest. More detailed correlation coefficient results are provided in Table S1.

3.2. The association between depressive symptoms and ADLs

The association between depressive symptoms and ADLs is presented in Fig. 3, Supplementary Figures S5 and S6, and Supplementary Tables S1-S8.

Table 1

Sample characteristics and functional measures at two-time segments (T1-T5 and T6-T10).

Catagoties	T1	T2	T3	T4	T5
Gender (Female, n%)	59.32				
Age (mean ± SD)	76.12 (7.17)				
Comorbidity (mean ± SD)	2.83 (1.74)	2.6 (1.56)	2.75 (1.59)	2.87 (1.58)	3 (1.58)
Depressive Symptoms (mean ± SD)	0.88 (1.31)	0.85 (1.27)	0.89 (1.35)	0.91 (1.32)	0.96 (1.35)
IADL (mean ± SD)	1.81 (1.63)	1.84 (1.68)	1.88 (1.7)	2 (1.74)	2.13 (1.77)
BADL (mean ± SD)	0.51 (1.04)	0.51 (1.05)	0.57 (1.13)	0.68 (1.23)	0.8 (1.37)
ADL (mean ± SD)	2.32 (2.29)	2.35 (2.34)	2.45 (2.47)	2.68 (2.62)	2.93 (2.8)
Catagoties	T6	T7	T8	T9	T10
Gender (Female, n%)	58.51				
Age (mean ± SD)	77.26 (6.76)				
Comorbidity (mean ± SD)	2.22 (1.51)	2.35 (1.55)	2.44 (1.53)	2.55 (1.56)	2.64 (1.56)
Depressive Symptoms (mean ± SD)	0.77 (1.21)	0.82 (1.25)	0.83 (1.22)	0.86 (1.27)	1.06 (1.42)
IADL (mean ± SD)	1.67 (1.58)	1.75 (1.62)	1.85 (1.68)	1.96 (1.73)	2.16 (1.77)
BADL (mean ± SD)	0.49 (1.02)	0.55 (1.08)	0.62 (1.16)	0.73 (1.26)	0.91 (1.47)
ADL (mean ± SD)	2.16 (2.25)	2.3 (2.34)	2.47 (2.5)	2.69 (2.67)	3.07 (2.92)

3.2.1. The association between depressive symptoms and BADL

In the model 1 of subset A, the RI-CLPM model demonstrated good fit (RMSEA = 0.036, 95% CI, 0.030–0.043; CFI = 0.994, Table S2). A total of 19.7% of the BADL effect variance was explained by between-person differences, while 80.3% was explained by within-person variations (Table S2). After accounting for the interaction with age (Model 2) and

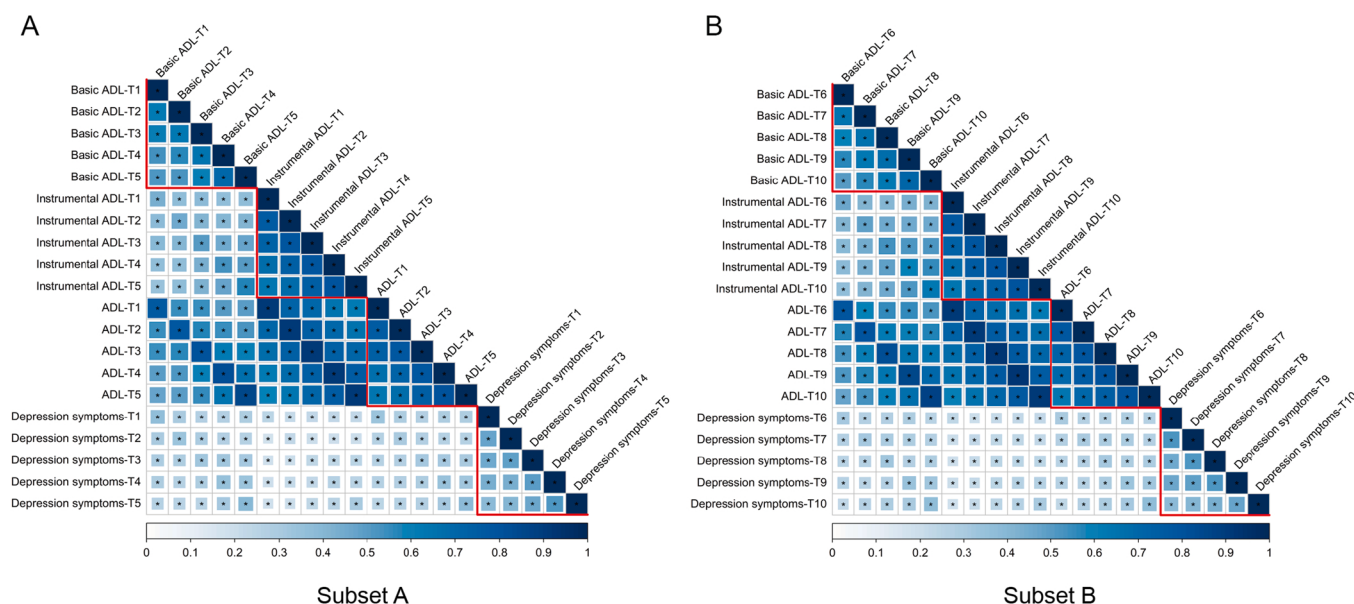


Fig. 2. The correlation among depressive symptoms and disability (BADL, IADL, and ADL) in the subset A (A, T1, T2, T3, T4, T5) and subset B (B, T6, T7, T8, T9, T10). The areas of the squares represent the absolute value of corresponding Spearman's correlations. Correlation estimates significantly different from zero at a 0.001 significance level (two stars) and a 0.05 significance level (one star) are shown.

Note: BADL = basic activities of daily living; IADL = instrumental activities of daily living; ADL = BADL + IADL.

additional covariates (Model 3), the model fit remained similar to that of Model 1, with good overall fit indices (Table S2). There was a significant correlation between depressive symptoms and BADL at the between-person level ($\beta = 0.499-0.548$, $p < 0.0001$, Fig. 3, Table S3) in three models. In model 3, the autoregressive effect of depressive symptoms was significantly positive across the five follow-up waves since 2011 (T1, T2, T3, T4, T5; the five effects β s range from 0.064 to 0.151, $p < 0.01$, Fig. 3, Table S3). Similarly, BADL exhibited significant positive autoregressive effects (β s range from 0.235 to 0.407, $p < 0.01$, Fig. 3, S3) across these waves. After adjusting for covariates, a significant cross-lag effect was observed between BADL at T4 and depressive symptoms at T5. The lower the level of BADL, the stronger the depressive symptoms in the subsequent measurement. For each 1-point decrease in BADL, depressive symptoms increased by 0.082 points (Table S3). Conversely, a significant cross-lag effect was observed from depressive symptoms to BADL, where a decrease in depressive symptoms at T1 increased BADL difficulty at T2 ($\beta = 0.051$, $p < 0.05$, Fig. 3, Table S3). Similar significant effects were also found from T2 to T3, T3 to T4, and T4 to T5.

In the subset B, model 3 also demonstrated good fit (Table S2). The cross-lag effects of BADL on depressive symptoms were observed in the five follow-up waves (T9 and T10: $\beta = 0.092$, $p < 0.0001$, Table S4). Conversely, the lagged effects of depressive symptoms on BADL were observed in T9 and T10 ($\beta = 0.066$, $p < 0.0001$, Table S4).

Gender had a significant impact on both depressive symptoms and disability. Compared to men, women were more prone to depression and had poorer BADL. Older age was associated with worse BADL at between-person. While baseline age did not significantly predict the between-person component (random intercept) of depressive symptoms, the average level of depressive symptoms increased over time (Fig. S4), suggesting a possible within-person aging effect or period-related trend. Individuals with comorbidities are more likely to experience depressive symptoms and had worse BADL compared to healthy individuals (Table S3 and S4).

3.2.2. The association between depressive symptoms and IADL

The model demonstrated good fit in the subset A (RMSEA = 0.040, 95% CI, 0.037–0.043, CFI = 0.970, Table S2) and the subset B (RMSEA = 0.038, 95% CI, 0.035–0.040, CFI = 0.974, Table S2) in the adjusted models. Differences in IADL were attributed 29.8% to between-person

variation and 70.2% to within-person variation (Table S2). At the between-person level, depressive symptoms were significantly positively correlated with IADL ($\beta = 0.395$, $p < 0.0001$, Fig. 3, Table S5). At the within-person level, depressive symptoms during follow-up waves exhibited significant positive autoregressive effects ($\beta = 0.064-0.148$, $p < 0.01$, Fig. 3, Table S5). Similarly, IADL demonstrated significant positive autoregressive effects at the follow-up waves ($\beta = 0.164-0.368$, $p < 0.0001$, Fig. 3, Table S5). Significant cross-lagged effects were observed between IADL and depressive symptoms. Worse IADL at T4 predicted stronger depressive symptoms at T5 ($\beta = 0.072$, $p < 0.001$, Fig. 3, Table S5). Conversely, depressive symptoms at T3 significantly predicted worse IADL at T4 ($\beta = 0.048$, $p < 0.05$, Fig. 3, Table S5).

Bidirectional cross-lagged effects were also observed in the subset B (Table S6). Notably, the impact of IADL on depressive symptoms was stronger than the reverse effect of depressive symptoms on IADL.

Gender significantly influenced both depressive symptoms and IADL. Although women were more susceptible to depressive symptoms, they tended to have better IADL functioning than men. While advancing age was associated with a decline in IADL, it did not appear to affect depressive symptoms at between-person. Additionally, individuals with comorbidities were more likely to experience depression and had worse IADL than those without comorbid conditions (Table S5 and S6).

3.2.3. The association between depressive symptoms and ADL

The adjusted models fitted well in both the subset A (RMSEA = 0.042, 95% CI, 0.040–0.045, CFI = 0.967, Table S2) and the subset B (RMSEA = 0.041, 95% CI, 0.038–0.044, CFI = 0.970, Table S2). 28.7% of the ADL differences were explained by between-person, and 71.3% were explained by within-person. Depressive symptoms and ADL were significantly positively correlated at the between-person ($\beta = 0.475$, $p < 0.0001$, Fig. 3, Table S7). At the within-person level, depressive symptoms during the follow-up waves (T1, T2, T3, T4, and T5) had significant positive autoregressive effects ($\beta = 0.066-0.146$, $p < 0.01$, Fig. 3, Table S7). Similarly, ADL at four waves (T2, T3, T4, and T5) showed significant positive autoregressive effects ($\beta = 0.105-0.457$, $p < 0.001$, Fig. 3, Table S7). There was a significant cross-lagged effect between ADL at T4 and depressive symptoms at T5 ($\beta = 0.098$, $p < 0.0001$, Fig. 3, Table S7). Lower level of ADL function was associated with stronger depressive symptoms in the subsequent measurement (Fig. 3, Table S7).

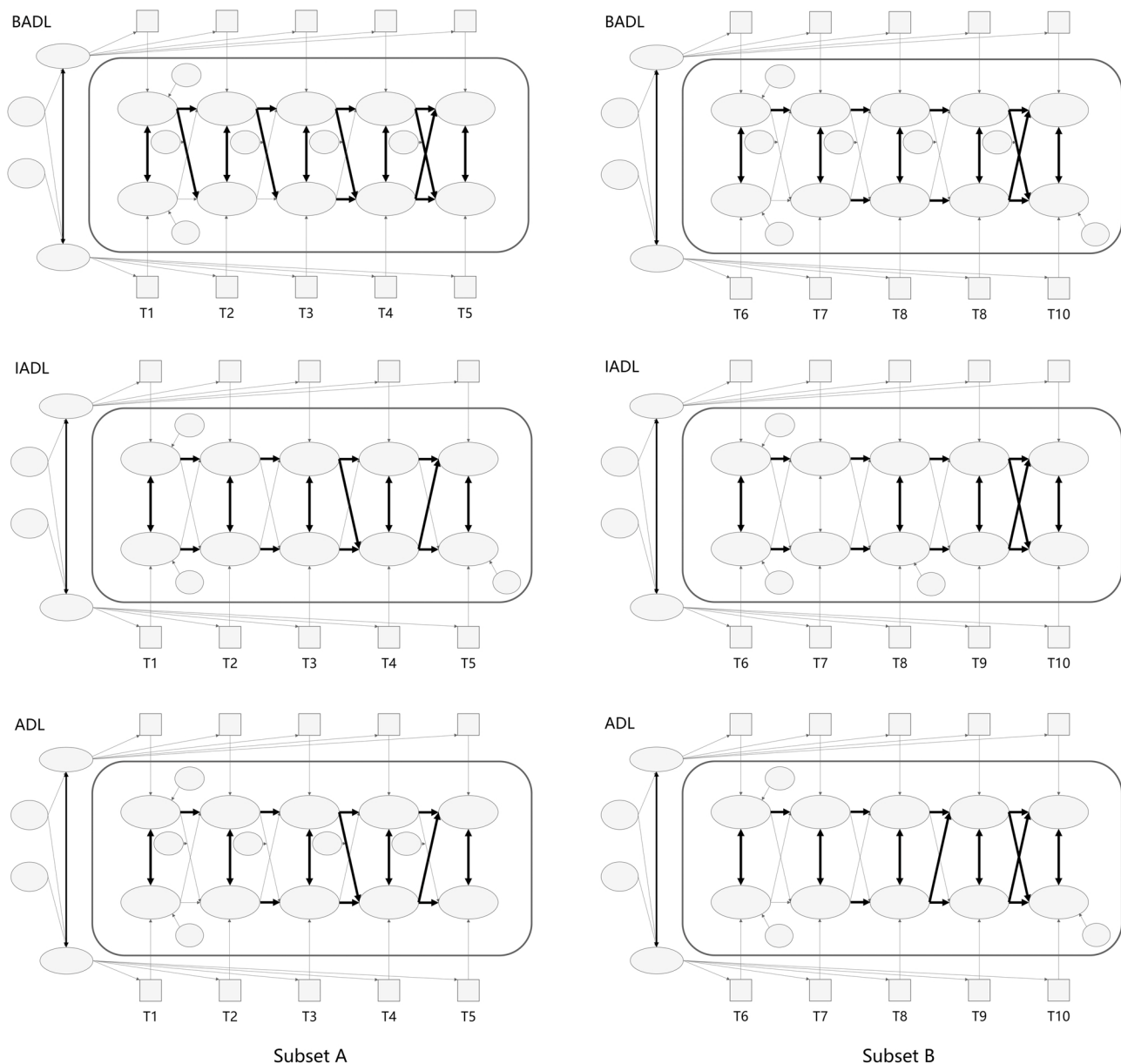


Fig. 3. Comparison of random intercept cross-lagged panel models (Model 3) of three ADL levels—including BADL, IADL, and ADL—across five time points in subset A (T1-T5) and subset B (T6-T10). Model 3 includes age interaction terms and adjusts for covariates (gender, age, and comorbidities).

Note: BADL = basic activities of daily living; IADL = instrumental activities of daily living; ADL = BADL + IADL.

A significant cross-lagged effect showed between depressive symptoms at T3 and ADL at T4 ($\beta = 0.061$, $p < 0.01$, Fig. 3, Table S7), indicating that stronger depressive symptoms resulted in worse levels of ADL in the next measurement.

Bidirectional cross-lagged effects were observed in the subset B as well (Table S8).

These results indicated a significant bidirectional correlation between ADL and depressive symptoms. The impact of ADL on depressive symptoms was stronger than the reverse effect.

Gender significantly impacted both depressive symptoms and ADL. Women were more prone to depressive symptoms, while men had better ADL. The older the age, the worse the ADL, though age did not significantly affect depressive symptoms at between-person. Compared to healthy individuals, those with comorbidities were more likely to experience depression and had poorer ADL.

4. Discussion

The study identified a mutually exacerbating spiral progression between the development of disability and the increase in depressive symptoms. When older adults experienced worse physical function than usual, their depressive symptoms became more pronounced in the subsequent periods. Conversely, an increase in depressive symptoms in the earlier periods contributed to declines in physical function in the future. This vicious cycle provides new perspectives for managing both mental and physical health in older adults.

Previous studies have reported inconsistent conclusions regarding the bidirectional effects of depressive symptoms and disability. An early study from the Groningen Ageing Study (GLAS) suggested that depressive symptoms had a lagged effect on disability, while disability influenced depressive symptoms only immediately, without a lagged effect [18]. In contrast, a recent study from the China Health and Retirement Longitudinal Study (CHARLS) found bidirectional lagged associations

between depressive symptoms and disability [17]. These discrepancies may be attributed to differences in statistical methodologies. The GLAS study applied SEM, whereas the CHARLS study used a CLPM. While both methods can examine reciprocal relationships, they may conflate within- and between-individual effects. Moreover, both studies utilized a composite ADL measure for disability assessment. Although this approach increases statistical power, it fails to capture nuanced functional distinctions, which may hinder the development of precise healthcare policies.

By investigating the relationship between depressive symptoms and disability within the same population, our study enriches existing knowledge. For the first time, we differentiated among various levels of ADL, addressing the issue of insufficient sensitivity caused by the use of comprehensive ADL indicators in previous studies. This differentiation allows for more targeted prevention strategies. Methodologically, we applied the RI-CLPM, which enhanced our ability to assess temporal and causal relationships by distinguishing between within-individual fluctuations and stable between-individual traits. Moreover, we accounted for the impact of covariates, ensuring the robustness of our findings.

A decline in ADLs leads to an increase in depressive symptoms. When older adults are unable to independently perform basic daily activities, they often feel helpless, lonely, and lost [28,29]. This functional loss not only impacts their sense of self-efficacy but also diminishes their social interactions, which in turn exacerbates depressive symptoms [30–32]. Numerous longitudinal studies have shown that sustained declines in ADLs are often associated with the onset of depressive symptoms [13–16]. Therefore, changes in ADLs can serve as important early warning signs for the emergence of depressive symptoms, underscoring the importance of maintaining ADL abilities in older adults.

Depressive symptoms are a critical predictor of disability. Previous research shows that individuals experiencing depressive symptoms are more likely to report difficulties in ADL functioning. Depressive symptoms can affect disability through various mechanisms. Cognitive symptoms of depression may impair an individual's ability to comprehend the tasks that need to be completed or how to perform them [33]. Physical symptoms, such as fatigue and pain, may further exacerbate physical decline, thereby reducing ADL capabilities [34]. Additionally, depressive symptoms can accelerate the progression of ADL disability through both social and psychological mechanisms [35].

Our findings indicate differential roles of IADLs and BADLs in relation to depressive symptoms and overall functional status. BADLs showed stronger bidirectional within-person associations with depressive symptoms across the five waves. For example, depressive symptoms predicted worse BADL ($\beta = 0.057$), and conversely, impaired BADL in wave 1 predicted higher depressive symptoms in wave 3 ($\beta = 0.082$). In contrast, the corresponding cross-lagged coefficients for IADLs were lower (depressive symptoms $\rightarrow$ IADL: $\beta = 0.048$; IADL $\rightarrow$ depressive symptoms: $\beta = 0.072$). This suggests that BADLs are more sensitive to short-term fluctuations in mood and vice versa. On the other hand, IADLs had the highest intraclass correlation coefficient (ICC = 0.298, based on between-person variance), compared to BADLs (ICC = 0.174) and total ADLs (ICC = 0.287), indicating that IADLs account for a larger proportion of stable, trait-like functional differences across individuals. Taken together, IADLs contribute more to the long-term structure of disability, whereas BADLs are more reactive and dynamically linked to mental health.

Extensive research has explored various prevention strategies for depressive symptoms and disability, with interventions such as improved sleep and dietary adjustments proven effective for preventing depressive symptoms [36–38], and physical exercise widely used to prevent disability [39]. However, achieving simultaneous prevention typically requires large, interdisciplinary, and cross-sectoral teams, posing significant demands on societal resources and healthcare systems.

This study offers a new perspective, advocating for an integrated approach that considers depressive symptoms and disability as

interconnected aspects of overall health, rather than isolated conditions. The mutually reinforcing spiral process suggests that a single preventive strategy could effectively address both issues, breaking the vicious cycle between them. For instance, targeting social factors could provide a promising entry point. Previous research has identified social interaction as a key mediator of mental and physical health [40,41]. Thus, strategies aimed at promoting social interaction could serve as preventive measures, offering dual benefits for both mental and physical health while mitigating the negative effects of their interaction.

Our study has important public health implications. Firstly, it uncovers the vicious cycle between depressive symptoms and disability, providing valuable insights into holistic pathways for mental-physical health management. Secondly, the bidirectional influence between depressive symptoms and declines in physical function at different levels informs directional recommendations for healthcare resource allocation, avoiding issues related to both over-treatment and under-treatment. Finally, viewing depressive symptoms and disability as interconnected aspects of health offers a theoretical foundation for developing more efficient prevention strategies, aiding in breaking the vicious cycle and achieving comprehensive health management goals.

Patients with depressive symptoms may exaggerate their self-assessment of ADLs due to personality traits such as pessimism or high neuroticism related to emotions, which could lead individuals to perceive their functional state as worse than it is [42,43]. Additionally, our evaluation of functional ability is based on subjective assessments and should ideally be complemented by objective measurements to improve the accuracy of evaluations [44]. Finally, the associations between depressive symptoms and ADLs need to be validated in additional populations to confirm their generalizability.

In summary, this study highlights the vicious cycle between depressive symptoms and disability, revealing its profound implications for health management in older adults. By proposing prevention strategies guided by an integrated health perspective, the research not only offers new insights into coordinated mental-physical health management but also provides a scientific basis for policymaking and resource allocation.

CRediT authorship contribution statement

Mengzhen Sun: Writing – review & editing, Writing – original draft, Methodology, Investigation, Data curation, Conceptualization. **Xiaoru Sun:** Writing – review & editing, Writing – original draft, Methodology. **Hui Zhang:** Investigation, Data curation. **Xiaoyan Jiang:** Investigation, Data curation, Conceptualization. **Xiaofeng Wang:** Writing – review & editing, Investigation, Data curation, Conceptualization. **Qi Zhang:** Writing – review & editing, Methodology.

Consent for publication

Not applicable.

Ethical approval and consent to participate

The study protocol for the NHATS was approved by the Johns Hopkins University Institutional Review Board. All participants provided written informed consent. Institutional review board approval was exempted for this study because of the publicly available and de-identified data.

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Data availability

The dataset used in this study is publicly available from the National Health and Aging Trends Study (NHATS) repository [<https://www.nhats.org/researcher/nhats>].

Declaration of competing interest

The authors declare no competing interests.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:<https://doi.org/10.1016/j.jnha.2025.100649>.

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