Longitudinal relationship between cognition and depressive symptoms in older adults from the
Virginia Cognitive Aging Project: a latent dual change score analysis

Mariana Teles¹ & Dingjing Shi²

¹ Department of Psychology, University of Virginia

² Department of Psychology, University of Oklahoma

Author notes

The authors made the following contributions. Mariana Teles Santos Golino:

Conceptualization, Writing - Original Draft Preparation, Writing - Review & Editing; Dingjing

Shi: Writing - Review & Editing.

Correspondence concerning this article should be addressed to Mariana Teles. E-mail:

mt2yq@virginia.edu

Abstract

Background: the direction of the longitudinal association between depression and cognition remains a topic of intense debate. A unidirectional association where depression impacts the change in cognition (or vice-versa) and a bidirectional association where the trajectories of both dimensions affect each other lead to different clinical implications. **Method:** this study aimed to investigate the directionality of the depression-cognition association in a sample of 2,057 older adults aged between 60 to 99 years old from the Virginia Cognitive Aging Project (VCAP). We used the bivariate dual change score model to investigate the association between five cognitive domains (memory, speed, reasoning, space, and vocabulary) and three dimensions of depression (somatic, depressed affect, and positive affect) throughout five measurement points with an average interval of 2.5 years between the assessments. Three directions were tested for each pair of variables: two unidirectional models and one bidirectional model. **Results:** a unidirectional effect in which depression at time t predicts change in cognition presented the best data fit for most of the cognitive domains. One significant causal effect was found: depressed affect (DA) at time t significantly predicted the change in memory with a negative association, that is, higher levels of DA at baseline predicted a worse performance on memory over time ($\gamma_{\text{Dep}} = -0.32881$; $\text{SE} = 0.17$; $p < 0.05$). **Conclusions:** our findings support a unidirectional association with depression predicting change for most of the cognitive domains tested. Higher levels of DA in older adults predicted an accelerated decline in memory.

Keywords: cognition, depression, association, directionality

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Introduction

How our mood is affected by the aging process and whether cognitive decline is influenced by depressive symptoms have been a topic of intense debate due to the clinical implications. Since a variability in the trajectory of depression is expected around the age of 60, when a steeper decline in the cognitive performance is also expected (Salthouse, 2004, 2019), the investigation of a possible relationship between depression and cognition is justified. A negative relationship between depression and cognition has been consistently reported by longitudinal studies, with depressive symptoms and cognitive decline identified in the literature as coexistent phenomena in older adulthood (e.g., Bielak, Gerstorf, Kiely, Anstey, & Luszcz, 2011; Dong et.al., 2016; Oi, 2017). In general, cross-sectional and longitudinal studies have demonstrated that higher scores in self-reported depressive symptoms are associated with lower scores in multiple cognitive measures and with an accelerated cognitive decline (Gale, Allert, & Deary, 2012; van den Kommer et. al., 2013; Wang, Yip, Lu, & Yeh, 2017). It is, therefore, central to the area of aging to investigate whether the trajectories of depression and cognition are strongly associated and the direction of this association.

Over the last decades, accumulated evidence have allowed the formulation of three hypotheses. The first two call for a unidirectional relationship between depression and cognition, while in the third hypothesis the relationship is bidirectional. In the first hypothesis, depression is a predictor of the rate of change in cognition ($D \rightarrow C$) and is considered either as a risk factor and as a prodromal stage of dementia (Butters et. al., 2008). Depression has been reported as an

important predictor of different cognitive abilities, like processing speed/attention (Bielak et al., 2011; Dong et. el., 2016; van den Kommer et al., 2013), memory (e.g.: Gale et al., 2012; Oi, 2017; Kohler, Thomas, Barnett, & O'Brien, 2010) and executive functioning (Kohler et. al., 2010). Wilson, Arnold, Beck, Bienias and Bennett (2008) also reported that, from a total of 917 participants, those who subsequently developed mild cognitive impairment (MCI; N = 319) had a significantly higher baseline level of depressive symptoms than those who did not develop MCI (N = 368).

The second hypothesis states that lower initial levels of cognitive performance are associated with an increase in depression over time, with the depressive symptoms being a mood reaction caused by one's awareness of cognitive loss (e.g.: Jajodia & Borders, 2011; Kommer et al., 2013; Perrino et al., 2008). And the third hypothesis points to a bidirectional relationship, in which depression and cognitive decline are co-existent phenomena with a co-morbid manifestation that possibly reflects either a causal effect (reciprocal or bidirectional association between the trajectories of depression and cognition) or a common cause related to physical processes, such as cardiovascular disease, reduced frontal lobe activity and neurodegenerative changes in limbic regions (MacQueen et al., 2003; Panza et al., 2010; Wilson et al., 2008).

The literature has accumulated evidences supporting all these hypotheses and the mixed results might be explained by some methodological limitations. The first limitation is the low quality of cognitive assessments since some studies used screening scales, like the MMSE (Kraemer, Moritz, & Yesavage, 1998) as a cognitive measure (e.g., Bassuk, Berkman, & Wypij, 1998; Ganguli, Du, Dodge, Ratcliff, & Chang, 2006; Geerlings et al., 2000; van Kommer et al., 2013; Ng, Niti, Zaw, & Kua, 2009) or the use of a single cognitive test (e.g., delay recall test) to assess a broad cognitive domain (e.g., memory; Bielak et al., 2011; Jajodia & Borders, 2011; Oi,

2017). Screening scales are not reliable measures for an early detection of cognitive decline and dementia (Salthouse, 2010) and the use of single tests as a proxy for the performance on broad cognitive domains affects the robustness of the assessment (i.e., does not enable the adequate identification of broad latent variables). Compelling evidence against reliance on single tests was presented by Salthouse (2012), who investigated in which level of a cognitive hierarchical structure depressive symptoms presented the highest impact and showed that depressive symptoms were significantly related only with the highest levels of the model (g-factor and five cognitive domains), rather than the level of observable variables (sixteen cognitive tests).

As a second methodological limitation of the field, many studies relied on longitudinal growth curve (LGC) models (e.g., Oi, 2017; Brailean et al., 2017; van Kommer et al., 2013); regression models (e.g., Chodosh, Kado, Seeman, & Karlamangla, 2007; Gale et al., 2012); or structural equation models (e.g., Perrino, Mason, Brown, Spokane, & Szapocznik, 2008) to investigate the directionality of the relationship between depression and cognition. Few studies used the dual change score (DCS) model (e.g., Bielak et al., 2011; Jajodia & Borders, 2011), which is a more appropriate approach to investigate developmental questions regarding causal effects and the directionality of the association between two variables in a latent variable modeling framework. Grimm (2007) points out that questions which could be answered by LGC models can also be answered using the DCS models, with the latter presenting the advantage of “dealing with multiple processes over time. This additional benefit is that the model directly examines time-dependent change as the latent differences are the outcome of interest and can be predicted by previous scores.” (p. 333) Therefore, the DCS approach, in particular the bivariate dual change score model (BDCS; McArdle, & Hamagami, 2001) allows the investigation of an important developmental question: whether a latent variable X at time t is a predictor of the changes in a latent variable Y and vice-versa. This can be accomplished by investigating the fit of

competing models, each with a different directionality represented by the coupling parameter (that, if set to zero, indicates that a given latent variable does not predict changes in a second latent variable). The time lagged predictions are unique to the BDCS model and cannot be estimated using LGC models.

Present Study

The current study investigates the directionality of the association between three dimensions of depression (somatic symptoms, depressed affect and positive affect) and five cognitive domains (vocabulary, reasoning, space, memory, and speed) measured with 16 cognitive tests, using data from the VCAP. Given the mixed results about the directionality of this association, the present study will analyze which of the two dimensions (cognition and depression) best predicts the other over time, using the BDSC model. This study tests the three hypotheses presented earlier in the introduction: 1) cognitive performance at time t predicts the change of depression (between time t and $t+1$); 2) depression at time t predicts the change in cognition (between time t and $t+1$); and 3) both dimensions predict each other. Additionally, we investigate whether higher levels of depression at baseline predict the rate of change of cognition, if lower cognitive performance at baseline predicts the rate of change of depressive symptoms, and if a parallel association exists between the trajectories of depressive symptoms and cognition with the rate of change of one varying according to the other.

Methods

Participants

The sample was composed of 2,057 participants ranging from 60 to 99 years old at the first measurement point (Mean = 70.25; SD = 7.74), being 62.23% female, with 16.23 years of education in average (SD = 2.85). For a summary of the recruitment strategies and data collection, see Salthouse (2019). About the selectivity of attrition for this sample, Salthouse (2014) reported that returning participants at initial occasion had higher cognitive performance among adults older than 50 years old.

Measures

Cognitive functioning was evaluated using sixteen tests, representing five cognitive domains: (1) Memory: Paired Association test (Salthouse, Fristoe, & Rhee, 1996) and two subtests of the Wechsler Memory Scale (Wechsler & others, 1997): logical memory and word recall; (2) Reasoning: Raven's Progressive Matrices (Raven, 1962), letter sets (Ekstrom, Dermen, & Harman, 1976), and the Shipley abstraction test (Zachary & Shipley, 1986); (3) Processing speed: digit symbol test (Wechsler, 1997), the letter comparison and pattern comparison tests (Salthouse & Babcock, 1991); (4) Vocabulary: Vocabulary test (Wechsler, 1997), Woodcock–Johnson picture vocabulary test (Woodcock, Johnson, & Mather, 1990), and the Synonym and Antonym Vocabulary tests (Salthouse, 1993); (5) Spatial visualization: spatial relations (Bennett, Seashore, & Wesman, 1947), paper folding and form boards (Ekstrom et al., 1976). For each time point, sum scores of each cognitive domain were calculated by summing the scores of the individual tests (e.g., total score of vocabulary at time t = score in the synonym test at time t + score in the antonym tests at time t).

Depression was assessed using the full version (20-items) of the Center for Epidemiological Studies-Depression scale (CES-D) (Radloff, 1977). Studies have reported a four-factor structure for this scale: depressed affect, positive affect, somatic symptoms, and interpersonal difficulties (Brailean et al., 2017; Radloff, 1977). The interpersonal difficulties factor has been recognized as a poor measure (Carleton et al., 2013) and for this reason it was excluded from our analyses.

Data analysis

In this study, the reciprocal and directional relationships between cognition and depression are addressed using the longitudinal structural equation modeling framework via the bivariate dual change score model (BDCS; McArdle, J. J., & Hamagami, F. (2001). The BDCS model can capture developmental processes, time-sequential associations within each developmental process and the directional relations between the developmental processes. The BDCS model combines methodological aspects of growth curve models and autoregressive cross-lagged models. The next paragraphs summarize how BDCS can be used to investigate the longitudinal relationship between depression and cognition.

The BDCS model focuses on the changes in trajectory and, therefore, the latent changes are typically the outcomes of the interest in BDCS. Based on the dual change score model (e.g., Grimm, 2017), one typical form of the bivariate change equations can be represented as:

$$\begin{aligned}\Delta d_{it} &= \eta_{d0} \times S_{di} + \beta_{d1} \times d_{i(t-1)} + \gamma_{d2} \times c_{i(t-1)}, \\ \Delta c_{it} &= \eta_{c0} \times S_{ci} + \beta_{c1} \times c_{i(t-1)} + \gamma_{c2} \times d_{i(t-1)},\end{aligned}\quad (3)$$

Where Δd_{it} and Δc_{it} represent the latent change and S_{di} and S_{ci} represent the rates of change. These terms are constant across time and are usually allowed to covary. The second sources of

latent difference come from the variables of their own previous states, which are summarized in the autoregressive parameters β_{d1} and β_{c1} . The second sources show that cognitive scores at the previous time point $c_{i(t-1)}$ affect the cognitive scores at its next state. Similarly, the current scores for the depressive symptoms are partially affected by the depressive symptoms from its previous time point $d_{i(t-1)}$. Additionally, there is a third piece that contributes to the latent changes in cognition and depressive symptoms, which makes the BDCS advantageous to determine the directional relationships between cognition and depressive symptoms. These unique sources of latent difference are captured in the coupling parameters γ_{d2} and γ_{c2} , demonstrating the amounts of latent difference that are explained by the previous state of the other variables. In other words, γ_{d2} determines whether cognition is a leading indicator of changes in depressive symptoms, and vice versa for γ_{c2} .

The BDCS model shares the benefits of GCM in that both models study the intraindividual change and interindividual differences in change. In addition, the BDCS answers research questions that traditional GCM cannot answer, since it can examine the separate time-dependent changes in depressive symptoms and cognition, where their later developments can be predicted by their own previous time scores in depression and cognition, respectively (i.e., the autoregressive parameters β_{d1} and β_{c1}). Furthermore, the BDCS allows the estimation of the directional relationship between two latent variables (cognition and depression), captured by the coupling parameters γ_{d2} and γ_{c2} . The coupling parameters γ_{d2} and γ_{c2} are usually central to research questions in the BDCS model. Significant coupling parameters are evidence of decisive relations of one variable affecting the other variable.

In the present study, measurements of cognition (memory, speed, reasoning, space, vocabulary) and depression (somatic symptoms, depressed affect, positive affect) from five time

points of the VCAP are used. For each pair of dimensions (e.g. memory and somatic symptoms), three BDSC models were fitted. In the bidirectional BDCS model, all coupling parameters (from cognition to depression: $C \rightarrow D$; and from depression to cognition: $D \rightarrow C$) were freely estimated. In the unidirectional BDCS models, only one of the coupling parameters was estimated and the other fixed to zero. Therefore, to test the directionality from cognition to depression, only the coupling parameter $C \rightarrow D$ was estimated; and from depression to cognition, only the coupling parameter $D \rightarrow C$ was estimated.

All analyses were implemented in Onyx (Titanium, 2003) and R (Team, 2017). Model fit was evaluated based on the guidelines proposed by Hu and Bentler (1999) for good fit. In order to determine which model presented the best fit to the data (i.e., the bidirectional model, the unidirectional $D \rightarrow C$ model or the unidirectional $C \rightarrow D$ model, for each dimension of cognition and depression), the Akaike Information Criterion (AIC) (Akaike, 1987) and Bayesian Information Criterion (BIC) (Raftery, 1995) were used.

(FIGURE 1)

Ethical Standard

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

Results

Descriptive Statistics

The average performance was calculated for all time points for each cognitive domain: a) Memory ($M = 73.77$; $SD = 159.25$); b) Speed: ($M = 80.17$; $SD = 172.57$); c) Reasoning: ($M = 23.39$; $SD = 51.74$); d) Space: ($M = 16.44$; $SD = 38.46$); and for each dimension of CES-D scale: a) Somatic: ($M = 3.50$; $SD = 9.01$); b) Depressed Affect: ($M = 1.62$; $SD = 5.44$); and c) Positive Affect: ($M = 1.82$; $SD = 5.23$).

Longitudinal Associations

In this section the causality effects and directionality associations between the three depression dimensions and the five cognitive domains will be reported. The relative fit indices AIC and BIC (sample-sized adjusted) were used to determine the model that showed the best fit to the data. The estimates for each parameter of the best-fitting models are shown in Table 2 to 6 and the fit indices for all models are presented in Supplementary Table 1. It is important to note that the positive affect dimension of the CES-D scale is inverted; therefore, a lower score in positive affect represents, actually, a high level of positive affect.

Memory and Depression.

For memory and depression, all BDCS models presented an adequate fit to the data ($CFI = 1$, $RMSEA = .00$; 90% CI $.04 - .005$). For the somatic symptoms and depressed affect dimensions of depression, the unidirectional model in which depression at time t predicts changes (constant slope) in memory scores presented the best fit (somatic symptoms: $BIC = 8211,765$; $AICS = 8100,372$; depressed affect: $BIC = 8280,828$; $AICS = 8169,434$), compared to the bidirectional models (somatic symptoms: $BIC = 8218,906$; $AICS = 8101,883$; depressed affect:

BIC = 8288,384; AICS = 8171,361) and the unidirectional models of memory predicting changes in depression (somatic symptoms: BIC = 8215,951; AICS = 8104,557; depressed affect: BIC = 8284,643; AICS = 8173,25). On the other hand, the unidirectional model in which memory predicts changes in positive affect (depression) presented a better fit to the data (BIC = 8326,301; AICS = 8214,907), compared to the bidirectional model (BIC = 8333,907; AICS = 8216,885) and the unidimensional model of positive affect-memory (BIC = 8326,402; AICS = 8215,008).

Table 1 shows that no significant causal effects and directionality associations were found between the somatic symptoms of depression and memory. However, depressed affect (DA) significantly predicts the change in cognition with a negative association, that is, higher levels of DA at one time point leads to a worse memory performance in subsequent time points ($\gamma_{Dep} = -0.32881$; SE = 0.17; $p < 0.05$). For positive affect (PA), no significant causal effects were found, but the covariance between the baseline levels of PA and memory ($\psi = -0.10775$, SE = 0.04, $p < 0.05$) was significant. Since the PA scale in the CES-D is inverted (higher scores imply a lower positive affect), this result shows that a higher memory performance is associated with a lower (inversed) PA score (therefore, a higher *actual* positive affect).

(TABLE 1)

Speed and Depression

For speed and depression, all BDCS models presented an adequate fit to the data (CFI = 1, RMSEA = .00; 90% CI .04 – .005). For the somatic symptoms and depressed affect dimensions of depression, the unidirectional model in which depression predicts changes (constant slope) in speed scores presented the best relative fit (somatic symptoms: BIC = 7967,638; AICS = 7856,244; depressed affect: BIC = 8042,618; AICS = 7931,224), compared to the bidirectional

model (somatic symptoms: BIC = 7975,038; AICS = 7858,015; depressed affect: BIC = 8050,127; AICS = 7933,104) and the unidirectional model of speed predicting changes in depression (somatic symptoms: BIC = 7971,303; AICS = 7859,909; depressed affect: BIC = 8043,427; AICS = 7932,033). Alternatively, the bidirectional model of speed and positive affect (PA) presented a better fit to the data (BIC = 8050,127; AICS = 7933,104) than the unidirectional model from speed to PA (BIC = 8086,695; AICS = 7975,301) and from PA to speed (BIC = 8088,056; AICS = 7976,662).

Table 2 shows that no significant causal effects and directionality associations were found between somatic symptoms, DA and PA and speed. However, significant covariances were found between: speed and somatic symptoms at the baseline ($\psi = -0.13106$; SE = 0.05; $p < 0.05$), meaning that high speed scores are associated with few somatic symptoms at baseline (and vice-versa); the baseline speed and the slope of DA ($\psi = -0.07772$; SE = 0.03; $p < 0.05$), suggesting that speed at baseline is negatively affecting the change in depression.

(TABLE 2)

Reasoning and Depression

For reasoning and depression, all the BDCS models presented adequate fit to the data for somatic symptoms and DA, but for PA only the unidirectional models fit the data well (CFI = 1, RMSEA = .00; 90% CI .04 – .005). For the somatic symptoms and DA dimensions of depression, the unidirectional model in which depression predicts changes (constant slope) in reasoning scores showed the best relative fit (somatic symptoms: BIC = 8431,065; AICS = 8319,671; depressed affect: BIC = 8499,585; AICS = 8388,191), compared to the bidirectional model (somatic symptoms: BIC = 8437,663; AICS = 8320,64; depressed affect: BIC = 8506,182; AICS

= 8389,16) and the unidirectional model from reasoning to depression (somatic symptoms: BIC = 8437,157; AICS = 8325,763; depressed affect: BIC = 8501,397; AICS = 8390,004). On the other hand, the unidirectional model in which reasoning predicts changes in PA showed a better fit to the data (BIC = 8546,782; AICS = 8435,389), compared to the bidirectional model (BIC = 9305,523; AICS = 9188,5) and the unidimensional model from PA to reasoning (BIC = 8543,934; AICS = 8432,54).

Table 3 shows that no significant causal effects and directionality associations were found between depression and reasoning. However, a number of significant covariances were found. Among these we highlight: a) cognition and somatic symptoms at baseline ($\psi = -0.10848$, SE = 0.04; $p < 0.05$); b) initial levels of reasoning positively affecting the change in somatic symptoms ($\psi = 0.4615$; SE = 0.08; $p < 0.05$) and initial levels of somatic symptoms positively associated with the slope of reasoning ($\psi = 0.10597$; SE = 0.04; $p < 0.05$); c) a negative covariance between DA and reasoning at baseline ($\psi = -0.11213$; SE = 0.05; $p < 0.05$), and both variables at baseline are inversely affecting the slope of the other; and d) PA at baseline and the slope of reasoning ($\psi = -0.07286$, SE = 0.03, $p < 0.05$).

(TABLE 3)

Spatial visualization and Depression

For space and depression, all BDCS models fitted the data adequately (CFI = 1, RMSEA = .00; 90% CI .04 – .005). For all dimensions of depression, the unidirectional model in which depression predicts changes in spatial visualization scores showed the best relative fit (somatic symptoms: BIC = 7924,348; AICS = 7817,397; depressed affect: BIC = 7987,879; AICS = 7876,485; positive affect: BIC = 8034,756; AICS = 7923,363), compared to the bidirectional

model (somatic symptoms: BIC = 7936,151; AICS = 7819,128; depressed affect: BIC = 7995,455; AICS = 7878,433; positive affect: BIC = 8042,244; AICS = 7925,221) and the unidirectional model from space to depression (somatic symptoms: BIC = 7928,992; AICS = 7817,598; depressed affect: BIC = 7988,404; AICS = 7877,01; positive affect: BIC = 8035,283; AICS = 7923,89). Table 4 shows that no significant causal effects and directionality associations were found between depression and spatial visualization, although a significant covariance for DA was found, with the baseline levels of DA inversely affecting the slope of spatial visualization ($\psi = -0.9422$; SE = 0.04; $p < 0.05$).

(TABLE 4)

Vocabulary and Depression

Finally, for vocabulary and depression all BDCS models for DA and PA fit the data well, and for the somatic symptoms dimension only the unidirectional models reached a good fit (CFI = 1, RMSEA = .00; 90% CI .04 – .005), in contrast with the bidirectional model (CFI = 0; RMSEA = 0,179). For DA and PA (depression), the unidirectional model in which depression predicts changes in vocabulary scores showed the best relative fit (depressed affect: BIC = 7890,458; AICS = 7779,064; positive affect: BIC = 7944,073; AICS = 7832,679), compared to the bidirectional model (depressed affect: BIC = 7896,965; AICS = 7779,943; positive affect: BIC = 7951,586; AICS = 7834,564) and the unidirectional model from vocabulary to depression (depressed affect: BIC = 7889,846; AICS = 7778,452; positive affect: BIC = 7944,261; AICS = 7832,867). For the somatic symptoms dimension, the unidirectional model in which vocabulary predicts changes in depression scores presented the best relative fit (BIC = 7828,023; AICS = 7716,629) compared to the unidirectional model from somatic to vocabulary (BIC = 7843,9; AICS = 7732,506).

Table 5 shows that no significant causal effects and directionality associations were found for vocabulary and depression. For DA and PA, significant covariances were found: a) vocabulary at baseline is negatively associated with the slope of DA ($\psi = -0.12598$; $SE = 0.04$); and b) vocabulary and PA are significantly related at the baseline ($\psi = -0.1441$; $SE = 0.05$; $p < 0.05$).

(TABLE 5)

Conclusions

Conclusions

The present study found a unidirectional effect in which depressive symptoms at time t predicted change in cognition ($D \rightarrow C$) for most of the associations. Somatic symptoms predicted change in four of the five cognitive domains investigated, including memory, speed, reasoning and space; DA predicted change in all the five cognitive domains; and PA predicted change in two of the five cognitive domains: space and vocabulary. These findings agree with the literature reports that the unidirectional association in which depression predicts cognition over time is the most common direction (Bielak et al., 2011; Gale et al., 2012; van Kommer et al., 2013; Köhler et al., 2010; Oi, 2017).

The present study also found one significant causal effect to the unidirectional model $D \rightarrow C$. DA at time t significantly predicted the change in memory with a negative association, that is, higher levels of DA at time t predicted a worse performance of memory over time ($\gamma_{Dep} = -0.32881$; $SE = 0.17$; $p < 0.05$), which is in accordance with other longitudinal studies (e.g., Oi, 2017; Gale et al., 2012; Köhler et al., 2010). The mechanisms underlying this association are

uncertain and have been a topic of intense debate: depressive symptoms have been reported either as a risk factor for subsequent development of dementia (Chodosh et al., 2007; Ownby, Crocco, Acevedo, John, & Loewenstein, 2006; Richard et al., 2013; Saczynski et al., 2010) or as a prodromal stage (early symptom) of dementia (Panza et al., 2010; Vinkers et al., 2004). Butters et al. (2008) advocates that both hypotheses (depression as a risk factor or a prodromal stage of dementia) are supported by large and robust evidence and so, it can be assumed that they are not mutually exclusive. Barnes et al. (2012) reported that chronic depression during life is associated with an increased risk for developing dementia, particularly vascular dementia, but when the first depressive episode occurs in late life, it may reflect a prodromal stage of dementia.

Other studies, however, reported memory as a leading predictor in the depression-cognition association, supporting the idea of depressive symptoms as an emotional reaction to the individual's awareness of his/her own memory decline. This was the conclusion reached by Jajodia and Borders (2011) using a change score model to test unidirectional and bidirectional models, and by Brailean et al. (2017) who also reported that memory at baseline (delay recall test) predicted a steeper increase in DA. The inconsistencies between these reports and the present study might be explained by the use of single tests to assess memory, compromising the robustness of the cognitive assessment in both studies. Another important methodological difference was the use of a growth curve model by Brailean et al. (2017) to test the directionality of the association instead of the dual change score approach. As shown by Usami, Murayama, & Hamaker (2019), the use of different cross-lagged models to investigate the same developmental question increases the risk of different results being reported.

The opposite direction, in which cognition at time t predicts change in depressive symptoms ($C \rightarrow D$) was found in the present study only for memory and reasoning predicting

PA, and vocabulary predicting somatic symptoms. Only one bidirectional effect was found between speed and PA. One possible explanation for cognition as a predictor of depression, is that depressive symptoms are merely mood reactions caused when the individual became aware of his cognitive loss (Engmann, 2011; Vinkers et al., 2004). The cognitive impairment may also negatively impact the individual's capacity to regulate mood and engage in activities that could prevent depressive symptoms or promote coping (Perrino et al., 2008).

It is important to notice that, with the exception of memory, the present study suggests a lack of significant causal effects between most of the pairs of dimensions tested. Fewer studies have reported no longitudinal association between depression and cognition for healthy older adults (e.g., Ganguli et al., 2006) and no systematic change in depression before and after dementia's diagnosis (Wilson et al., 2008). These findings suggest that cognition and depression are independent phenomena and their co-existence might be explained by common causes underlying major depression and dementia, like vascular risk factors (Butters et al., 2008; Panza et al., 2010), cerebrovascular lesions and damage on the hypothalamic-pituitary-adrenal-stress axis (Butters et al., 2008; Lupien et al., 1998).

This study aims to contribute to the field with important advances, like the robustness of the cognitive assessment conducted in a large sample and the use of a latent change score model to test the directionality of the association. The findings, however, must be interpreted in light of a number of methodological limitations. First, the sample was not assessed through a formal diagnosis of depression and cognitive impairment and there is no clinical data of risk factors that have been linked to both depression and cognitive impairment (Anstey, von Sanden, SargentCox, & Luszcz, 2007). Additionally, the sample attrition across time resulted in a sample with a higher cognitive functioning.

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Conflicts of Interest

None

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Tables

Table 1. Parameter estimates obtained from the best fitting models for Memory and Depression

Direction: Somatic symptoms (Depression) → Memory		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level memory, constant slope memory)	-0.21703 (0.11)	-1.96*
ψ Cov(initial level somatic symptoms, constant slope somatic symptoms)	0.17251 (0.13)	1.34
γ_{Dep} (Somatic symptoms → memory coupling parameter)	-0.5322 (0.27)	-1.93
ψ Cov(initial level memory, initial level somatic symptoms)	-0.06904 (0.05)	-1.44
ψ Cov(initial level memory, constant slope somatic symptoms)	-0.01306 (0.025)	-0.52
ψ Cov(initial level somatic symptoms, constant slope memory)	0.28763 (0.15)	1.91
ψ Cov(constant slope memory, constant slope somatic symptoms)	0.09758 (0.08)	1.18
Direction: Depressed Affect (Depression) → Memory		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level memory, constant slope memory)	-0.20883 (0.11)	-1.98*
ψ Cov(initial level depressed affect, constant slope depressed affect)	0.24617 (0.07)	3.58*
γ_{Dep} (Depressed affect → memory coupling parameter)	-0.32881 (0.17)	-1.96*
ψ Cov(initial level memory, initial level depressed affect)	-0.06865 (0.05)	-1.39
ψ Cov(initial level memory, constant slope depressed affect)	-0.04996 (0.03)	-1.76
ψ Cov(initial level depressed affect, constant slope memory)	0.17722 (0.09)	2.04*
ψ Cov(constant slope memory, constant slope depressed affect)	0.09918 (0.05)	1.84
Direction: Memory → Positive Affect (Depression)		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level memory, constant slope memory)	-0.21911 (0.11)	-2.05*
ψ Cov(initial level positive affect, constant slope positive affect)	-0.17514 (0.20)	-0.88
γ_{Cog} (Memory → depression coupling parameter)	0.0701 (0.21)	0.33
ψ Cov(initial level memory, initial level positive affect)	-0.10775 (0.04)	-2.34*
ψ Cov(initial level memory, constant slope positive affect)	-0.02192 (0.15)	-0.15
ψ Cov(initial level positive affect, constant slope memory)	0.03507 (0.022)	1.53
ψ Cov(constant slope memory, constant slope positive affect)	0.00326 (0.04)	0.08

Table 2. Parameter estimates obtained from the best fit models for Speed and Depression

Direction: Somatic symptoms (Depression) → Speed		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level speed, constant slope speed)	-0.18983 (0.14)	-1.34
ψ Cov(initial level somatic symptoms, constant slope somatic symptoms)	0.20389 (0.12)	1.68
γ_{Dep} (Somatic symptoms → speed coupling parameter)	-0.47804 (0.26)	-1.86
ψ Cov(initial level speed, initial level somatic symptoms)	-0.13106 (0.05)	-2.79*
ψ Cov(initial level speed, constant slope somatic symptoms)	-0.05429 (0.04)	-1.43
ψ Cov(initial level somatic symptoms, constant slope speed)	0.26104 (0.13)	1.94
ψ Cov(constant slope speed, constant slope somatic symptoms)	0.11432 (0.09)	1.25
Direction: Depressed Affect (Depression) → Speed		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level speed, constant slope speed)	-0.17977 (0.14)	-1.25
ψ Cov(initial level depressed affect, constant slope depressed affect)	0.24551 (0.08)	3.16
γ_{Dep} (Depressed affect → speed coupling parameter)	-0.17217 (0.17)	-1.00
ψ Cov(initial level speed, initial level depressed affect)	-0.08201 (0.05)	-1.67
ψ Cov(initial level speed, constant slope depressed affect)	-0.07772 (0.03)	-2.57*
ψ Cov(initial level depressed affect, constant slope speed)	0.09581 (0.09)	1.06
ψ Cov(constant slope speed, constant slope depressed affect)	0.06629 (0.06)	1.17
Direction: Speed → Positive Affect (Depression)		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level speed, constant slope speed)	-0.17396 (0.14)	-1.21
ψ Cov(initial level positive affect, constant slope positive affect)	0.23567 (0.08)	2.85*
γ_{Cog} (Speed → positive affect coupling parameter)	-0.07664 (0.22)	-0.35
γ_{Dep} (Positive affect → speed coupling parameter)	-0.16229 (0.17)	-0.94
ψ Cov(initial level speed, initial level positive affect)	-0.08171 (0.05)	-1.66
ψ Cov(initial level speed, constant slope positive affect)	-0.02047 (0.17)	-0.12
ψ Cov(initial level positive affect, constant slope speed)	0.09006 (0.09)	0.99
ψ Cov(constant slope speed, constant slope positive affect)	0.05111 (0.07)	0.79

Table 3. Parameter estimates obtained from the best fit models for Reasoning and Depression

Direction: Somatic symptoms (Depression) → Reasoning		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level reasoning, constant slope reasoning)	3.40126 (5.50)	0.62
ψ Cov(initial level somatic symptoms, constant slope somatic symptoms)	0.4615 (0.08)	6.12*
γ_{Dep} (Somatic symptoms → reasoning coupling parameter)	22.02799 (46.80)	0.47
ψ Cov(initial level reasoning, initial level somatic symptoms)	-0.10848 (0.04)	-2.49*
ψ Cov(initial level reasoning, constant slope somatic symptoms)	-0.10597 (0.04)	-2.70*
ψ Cov(initial level somatic symptoms, constant slope reasoning)	-11.6054 (24.22)	-0.48
ψ Cov(constant slope reasoning, constant slope somatic symptoms)	-10.3643 (21.73)	-0.48
Direction: Depressed Affect (Depression) → Reasoning		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level reasoning, constant slope reasoning)	0.37431(0.06)	6.06*
ψ Cov(initial level depressed affect, constant slope depressed affect)	0.28858 (0.08)	3.57*
γ_{Dep} (Depressed affect → reasoning coupling parameter)	0.26756 (0.18)	1.45
ψ Cov(initial level reasoning, initial level depressed affect)	-0.11213 (0.05)	-2.20*
ψ Cov(initial level reasoning, constant slope depressed affect)	-0.09661 (0.04)	-2.65*
ψ Cov(initial level depressed affect, constant slope reasoning)	-0.18939 (0.09)	-2.08*
ψ Cov(constant slope reasoning, constant slope depressed affect)	-0.16306 (0.08)	-2.04*
Direction: Reasoning → Positive Affect (Depression)		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level reasoning, constant slope reasoning)	0.34182 (0.06)	6.01*
ψ Cov(initial level positive affect, constant slope positive affect)	-0.2153 (0.23)	-0.92
γ_{Cog} (Reasoning → positive affect coupling parameter)	-0.10936 (0.17)	-0.66
ψ Cov(initial level reasoning, initial level positive affect)	-0.08763 (0.05)	-1.85
ψ Cov(initial level reasoning, constant slope positive affect)	0.07853 (0.12)	0.64
ψ Cov(initial level positive affect, constant slope reasoning)	-0.07286 (0.03)	-2.12*
ψ Cov(constant slope reasoning, constant slope depression)	0.07529 (0.10)	0.78

Table 4. Parameter estimates obtained from the best fit models for Space and Depression

Direction: Somatic symptoms (Depression) → Space		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level spatial, constant slope space)	0.41991 (0.13)	3.24*
ψ Cov(initial level somatic symptoms, constant slope somatic symptoms)	0.16267 (0.15)	1.10
γ_{Dep} (Depression → spatial coupling parameter)	0.16497 (0.25)	0.65
ψ Cov(initial level spatial, initial level somatic symptoms)	-0.05951 (0.05)	-1.19
ψ Cov(initial level spatial, constant slope somatic symptoms)	-0.02355 (0.03)	-0.91
ψ Cov(initial level somatic symptoms, constant slope spatial)	-0.13811 (0.13)	-1.03
ψ Cov(constant slope spatial, constant slope somatic symptoms)	-0.05038 (0.06)	-0.89
Direction: Depressed Affect (Depression) → Space		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level spatial, constant slope space)	0.431 (0.13)	3.39
ψ Cov(initial level depressed affect, constant slope depressed affect)	0.26964 (0.08)	3.50
γ_{Dep} (Depression → spatial coupling parameter)	0.09228 (0.12)	0.74
ψ Cov(initial level spatial, initial level depressed affect)	-0.0965 (0.05)	-1.88
ψ Cov(initial level spatial, constant slope depressed affect)	-0.09422 (0.04)	-2.63*
ψ Cov(initial level depressed affect, constant slope space)	-0.08793 (0.07)	-1.34
ψ Cov(constant slope spatial, constant slope depressed affect)	-0.08135 (0.05)	-1.64
Direction: Positive Affect (Depression) → Space		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level spatial, constant slope spatial)	0.45828 (0.14)	3.23*
ψ Cov(initial level positive affect, constant slope positive affect)	-0.16491 (0.20)	-0.83
γ_{Cog} (Spatial → positive affect coupling parameter)	0.23677 (0.32)	0.75
ψ Cov(initial level spatial, initial level positive affect)	-0.08344 (0.05)	-1.79
ψ Cov(initial level spatial, constant slope positive affect)	0.01469 (0.04)	0.36
ψ Cov(initial level positive affect, constant slope spatial)	-0.16884 (0.16)	-1.03
ψ Cov(constant slope spatial, constant slope positive affect)	0.04639 (0.08)	0.55

Table 5. Parameter estimates obtained from the best fit models for Vocabulary and Depression

Direction: Somatic symptoms (Depression) → Vocabulary		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level vocabulary, constant slope vocabulary)	-0.11004 (0.18)	-0.61
ψ Cov(initial level somatic symptoms, constant slope somatic symptoms)	0.22399 (0.20)	1.11
γ_{Dep} (Somatic symptoms → vocabulary coupling parameter)	-0.25073 (0.28)	-0.88
ψ Cov(initial level vocabulary, initial level somatic symptoms)	-0.09376 (0.05)	-1.88
ψ Cov(initial level vocabulary, constant slope somatic symptoms)	0.10111 (0.25)	0.40
ψ Cov(initial level somatic symptoms, constant slope vocabulary)	-0.0097 (0.03)	-0.30
ψ Cov(constant slope vocabulary, constant slope somatic symptoms)	-0.00975 (0.04)	-0.27
Direction: Depressed Affect (Depression) → Vocabulary		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level vocabulary, constant slope vocabulary)	-0.20396 (0.21)	0.97
ψ Cov(initial level depressed affect, constant slope depressed affect)	0.27012 (0.08)	3.33*
γ_{Dep} (Depressed affect → vocabulary coupling parameter)	-0.1273 (0.18)	-0.71
ψ Cov(initial level vocabulary, initial level depressed affect)	-0.07077 (0.05)	-1.43
ψ Cov(initial level vocabulary, constant slope depressed affect)	-0.12598 (0.04)	-3.37*
ψ Cov(initial level depressed affect, constant slope vocabulary)	0.06155 (0.10)	0.62
ψ Cov(constant slope vocabulary, constant slope depressed affect)	0.06473 (0.08)	0.81
Direction: Positive Affect (Depression) → Vocabulary		
<i>Parameter's meaning</i>	<i>Estimate (SE)</i>	<i>Z</i>
ψ Cov(initial level vocabulary, constant slope vocabulary)	-0.15838 (0.19)	-0.82
ψ Cov(initial level positive affect, constant slope positive affect)	-0.19502 (0.21)	-0.91
γ_{Dep} (Positive affect → vocabulary coupling parameter)	0.14268 (0.31)	0.46
ψ Cov(initial level vocabulary, initial level positive affect)	-0.1441 (0.05)	-3.16*
ψ Cov(initial level vocabulary, constant slope positive affect)	0.04972 (0.06)	0.82
ψ Cov(initial level positive affect, constant slope vocabulary)	-0.05056 (0.16)	-0.31
ψ Cov(constant slope vocabulary, constant slope positive affect)	0.01675 (0.07)	0.25