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BRIEF REPORT

Depressive Symptoms Predict Decline in Perceptual Speed in Older Adulthood

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Depressive symptoms and cognitive decline are associated in older age, but research is inconsistent about whether one condition influences the development of the other. We examined the directionality of relations between depressive symptoms and perceptual speed using bivariate dual change score models. Assessments of depressive symptoms and perceptual speed were completed by 1,206 nondemented older adults at baseline, and after 2, 8, 11, and 15 years. After controlling for age, education, baseline general cognitive ability, and self-reported health, allowing depressive symptoms to predict subsequent change in perceptual speed provided the best fit. More depressive symptoms predicted subsequently stronger declines in perceptual speed over time lags of 1 year.

Keywords: depressive symptoms, cognitive decline, perceptual speed, coupling, temporal relations

The coexistence of depressive symptoms and cognitive decline in older adulthood is well established, whereby greater depressive symptoms are associated with poorer cognitive functioning and cognitive decline (Dotson, Resnick, & Zonderman, 2008). However, the causal and temporal relations between depressive symptoms and cognition remain ill-defined.

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There are suggestions that depressive symptoms may increase an individual's risk for subsequent cognitive decline. For example, there are reports that those with more baseline depressive symptoms had greater cognitive decline over time than those without depressive symptoms (Köhler et al., 2010; Yaffe et al., 1999). Another study found no increase in depressive symptoms during the prodromal phase of Alzheimer's disease (AD; Wilson, Arnold, Beck, Bienias, & Bennett, 2008), and concluded that depression acts to change cognition, and not the other way around. In contrast, lower cognitive functioning may increase the risk for depressive symptoms. Studies that have found depressive symptoms were unrelated to the rate of cognitive decline (Ganguli, Yangchun, Dodge, Ratcliff, & Chang, 2006; Vinkers, Gussekloo, Stek, Westendorp, & van der Mast, 2004) have consequently concluded that the alternative directional explanation (i.e., cognitive decline leads to depressive symptamotology) is more probable.

Thus, the connection between aspects of depression and cognition remains equivocal. Further, it may not be best expressed by a simple unidirectional temporal pathway. Han and colleagues (2006) attributed the link between depressive symptoms and general cognitive functioning to short-term situational factors, in that the relation is concurrent or temporary, rather than leading to increased risk. Alternatively, Jorm (2000) noted various hypotheses concerning the common coexistence of depressive symptoms and dementia including other conceptualizations such that depression and dementia share common risk factors (e.g., vascular disease).

Nonetheless, we restrict our focus to the time-ordered relation between depressive symptoms and cognitive decline. Insight into the directionality of this association will aid in understanding whether and how their interrelation develops over time, and pos-

sibly provide key information into the disease process of dementia and major depression.

Although longitudinal analysis has informed our understanding of covariation between depressive symptoms and cognitive decline (Chen, Ganguli, Mulsant, & DeKosky, 1999; Ganguli et al., 2006; Han et al., 2006; Vinkers et al., 2004; Yaffe et al., 1999), there has been no direct test of the directionality of the relation whereby a dynamic association is allowed to occur across time, or where one variable is modeled as being related to change in the other variable over time. With recent advances in statistical modeling, the bivariate dual change score model (BDCSM; McArdle & Hamagami, 2001) indeed allows for such temporal comparisons. This model is based in a structural equation modeling framework, and builds upon the latent growth curve model that estimates latent intercept and slope factors at the population level. The BDCSM allows for examining time-ordered associations between two constructs, thereby testing whether one variable at a given point in time predicts subsequent change in the other variable (see Figure 1). This model has been successfully applied to empirically test associations among other psychological domains, including competing hypotheses about the nature of lead-lag associations between cognition and leisure activities (Lövdén, Ghisletta, & Lindenberger, 2005), cognition and well-being (Gerstorf, Lövdén, Röcke, Smith, & Lindenberger, 2007), and the dynamic associations of cognitive functioning between spouses (Gerstorf, Hoppmann, Anstey, & Luszcz, 2009).

In this study, we applied the BDCSM to 15-year longitudinal data of depressive symptoms and perceptual speed. Perceptual speed was selected as the marker of cognitive functioning, as it constitutes the limiting factor for other cognitive processes (Luszcz & Bryan, 1999) and is a highly reliable and sensitive marker of cognitive change throughout adulthood (Anstey, Hofer, & Luszcz, 2003; Salthouse, 2004). By simultaneously modeling longitudinal changes in depressive symptoms and cognition, we tested various hypotheses about their dynamic relation over time. First, we investigated both unidirectional hypotheses: whether the level of depressive symptoms at time (t) predicts subsequent change in perceptual speed between (t) and [$t + 1$], or whether the level of perceptual speed predicts changes in depressive symptomatology. Next, we analyzed bidirectional associations, including where both leading pathways exist and are of different magnitude, and where neither variable is associated with changes in the other. Finally, we covaried the effects of baseline age, education, general cognitive status, and number of medical conditions to ensure our findings do not simply reflect the influence of these variables on cognitive functioning and depressive symptoms (Bassuk, Berkman, & Wypij, 1998; Geerlings et al., 2000).

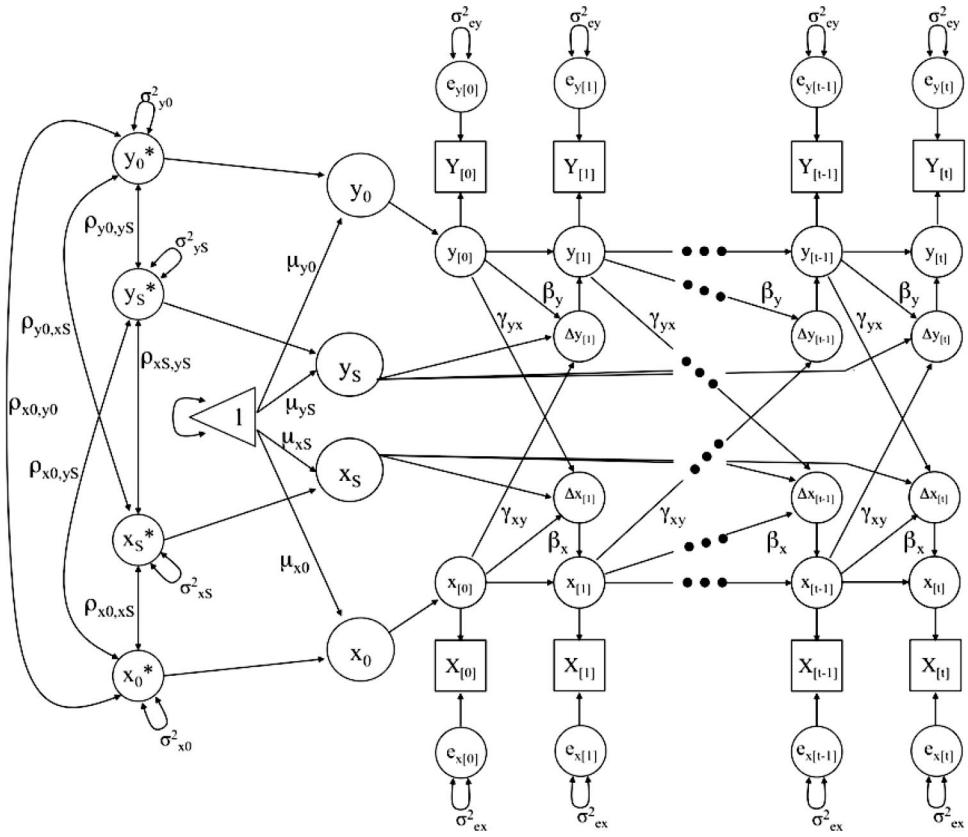

Figure 1. Graphical representation of a two-variable Dual Change Score Model (DCSM; McArdle & Hamagami, 2001) for a system of two variables *X* and *Y*. Observed (manifest) variables are represented by squares, unobserved (latent) variables by circles, regression weights by one-headed arrows, variances and covariances by two-headed arrows, and the triangle represents a constant indicating means and intercepts. All unlabeled paths are set to 1.

Method

Participants

The study sample was drawn from the Australian Longitudinal Study of Ageing (Luszcz et al., 2007). Potential participants resided in Adelaide, Australia, were 70 years or older, and lived in either community or residential care settings. Participants were recruited through the electoral roll, for which registration is compulsory for Australian citizens. Of the 2,703 residents eligible for study inclusion, 1,477 (55%) agreed to participate. Spouses of participants who were over 65 years of age and co-residents over 70 were also invited to participate because of potential interest in investigating the dynamics among couples, and to increase the number of participants, respectively. These invitations resulted in a further 610 participants.

At baseline, the 2,087 participants were aged between 65 and 103 ($M = 78.16$, $SD = 6.69$), and approximately half of the sample was female (49.4%). The outcome measures pertinent to this study were collected at Waves 1 (September, 1992–February, 1993), 3 (September, 1994–February, 1995), 6 (September, 2000–February, 2001), 7 (September, 2003-April, 2004), and 9 (November, 2007–June, 2008), or at baseline and after approximately 2, 8, 11, and 15 years. Data from these waves were collected in three formats: personal interview, self-completion questionnaire, and clinical assessment, all collected in the participant's residence. The personal interview and self-completion questionnaire included a range of self-reported demographic, psychosocial, and health measures. The clinical assessment included objective measures of psychological and physical functioning and was completed approximately 2 weeks later (Luszcz, Bryan, & Kent, 1997).

Although the structural equation modeling framework permitted the inclusion of all participants regardless of attrition, we chose to include the requirement that participants needed to have valid baseline data on all variables of interest. Consequently, the final sample was reduced to 1,206, with a mean length of follow-up of 5.95 years ($SD = 5.00$).

Participants who completed all relevant Wave 1 measures were significantly younger ($F(1, 2085) = 44.32$, $p < .001$, $\eta^2 = .02$; 77.88 years) and more likely to have stayed in school beyond age 14 ($\chi^2 = 26.80$, $df = 1$, $p < .001$, $\eta^2 = .12$; 48.3%) than those who did not have complete data for the first wave of measurement (79.84 years; 36.5%). They also had significantly fewer depressive symptoms ($M = 7.82$) than those who did not have complete data ($M = 8.85$; $F(1, 1991) = 9.33$, $p < .01$, $\eta^2 = .01$), but there was a wide range of depressive symptoms in the study sample (see Table 1). The vast majority of those who did not have complete Wave 1 data did not perform the perceptual speed task, thus preventing comparison on this variable, but those included in this report had higher Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) scores ($F(1, 2042) = 94.24$, $p < .001$, $\eta^2 = .04$; $M = 27.61$) than those who were not ($M = 25.82$). There were no significant differences in relation to sex composition or medical conditions. Table 1 describes the baseline characteristics of the sample.

Measures

Depressive symptoms were assessed using the 20-item Center for Epidemiological Studies Depression Scale (Radloff, 1977). *Perceptual speed* was measured by the Digit Symbol Substitution test (Wechsler, 1981). Participants were presented with a coding

Table 1
Descriptive Statistics for Measures Entered Into the Bivariate Dual Change Score Model

Measure	*n*	*M*	*SD*	Raw score range
Depressive symptoms				
Wave 1	1,206	50.10	9.93	0–48
Wave 3	955	49.31	9.34	0–42
Wave 6	447	51.20	8.98	0–39
Wave 7	301	50.21	9.96	0–40
Wave 9	135	51.61	9.53	0–35
Perceptual speed				
Wave 1	1,206	49.45	9.59	0–72
Wave 3	859	51.16	10.11	2–72
Wave 6	349	50.94	9.15	3–63
Wave 7	262	49.07	9.72	0–67
Wave 9	121	50.40	9.15	8–63
Covariates				
Age	1,206	77.88	6.30	65–98
Medical conditions	1,206	2.47	1.66	0–10
MMSE > 24	96	8.0%		
Education < 15 years of age	579	48.0%		

Note. Depressive symptoms and perceptual speed were assessed at baseline and after approximately 2, 8, 11, and 15 years. Depressive symptoms were assessed using the Center for Epidemiological Studies Depression Scale (Radloff, 1977). Depressive symptoms and perceptual speed scores were standardized to the T metric ($M = 50$, $SD = 10$) using the baseline sample. MMSE (Mini-mental Status Examination) was dichotomized as scoring less than 24, and 24 or more. Education information was limited to a dichotomized variable of leaving school before 15 years of age, or leaving at age 15 or later.

key pairing numbers 1–9 with nine symbols. The score was the number of items correctly coded in 90 sec.

Regarding the covariates, we chose to control for the effects of age, education, general cognitive status, and number of medical conditions because of their well-established association with cognitive impairment and dementia (Williams, Plassman, Burke, Holsinger, & Benjamin, 2010). Baseline *age* was calculated to the nearest day and centered near sample mean at 78 years old. *Education* was assessed by a binary measure of the age participants left formal schooling: before age 15 ($n = 579$), or at age 15 or older ($n = 627$). *General cognitive status* was determined by Wave 1 MMSE scores and coded as a binary variable using the clinical dementia cutoff of less than 24 ($n = 96$), and 24 or more ($n = 1110$). *Medical conditions* were based on the number of self-reported current chronic conditions from a comprehensive list of 37 diseases (e.g., arthritis, heart disease, cancer).

Data Preparation and Statistical Analysis

To enable comparison across measures, depressive symptoms and perceptual speed were standardized to a T metric ($M = 50$, $SD = 10$), using the mean scores of the baseline sample as the reference. The intra-class correlations derived from the random-intercept only models revealed that there was substantial between-person and within-person variance in the data (depressive symptoms: 0.488; perceptual speed: 0.616), justifying the use of the BDCSM to describe these changes.

Figure 1 shows a graphical representation of the BDCSM, with depressive symptom scores defined by X, and perceptual speed scores defined by Y. The X[0] to X[t] and Y[0] to Y[t] represent the assessments of depressive symptoms and perceptual speed. By adding unmeasured 'node' variables for occasions on which a given variable was not assessed, we simplified the interpretation of model parameters and guaranteed an equal-interval, time-invariant scaling of approximately 1 year in-between occasion.

The latent scores x[t] (or y[t]) are defined as the unit-weighted sum of the latent score x[t-1] (or y[t-1]), or the latent score at the previous assessment, plus the latent difference score Δx[t] (or Δy[t]), resulting in the latent, reliable change between x[t-1] and x[t] (or y[t-1]) and y[t]) (McArdle & Nesselroade, 1994). The intercepts (x_0, y_0) and slope factors (x_s, y_s) represent scores at baseline and linear change score, respectively, and are estimated at the population level (μ_{x0}, μ_{xS}; μ_{y0}, μ_{yS}). They are allowed to vary (σ^2_{x0}, σ^2_{xS}; σ^2_{y0}, σ^2_{yS}), and to covary with one another (ρ_{x0xS}; ρ_{y0yS}; ρ_{x0y0}; ρ_{xSyS}; ρ_{x0yS}; ρ_{y0xS}). Error terms (e_x, e_y) are assumed to be normally distributed with a mean of zero, have a time-invariant variance, and be uncorrelated with other components.

As an extension of typical linear Latent Growth Curve models, the difference scores Δx[t] and Δy[t] are defined as the unit-weighted sum of the linear component of change within the target variable plus two additional influences. First, the auto-proportion parameter (βx, βy) indicates the effect of the target variable at time t-1 on change in the same variable between t-1 and t (e.g., the effect that level of depressive symptoms has on change in depressive symptoms). Second, an intervariable, cross-lagged dynamics parameter (γ_{xy}; γ_{yx}) estimates the effect of one variable (e.g., X) at time t-1 on subsequent change in the other variable (e.g., Y) between t-1 and t (e.g., the effect that level of depressive symptoms has on subsequent change in perceptual speed). Both β and γ parameters are assumed to be time-invariant. Further information on the assumptions of the BDCSM can be found elsewhere (Ferrer & McArdle, 2004; McArdle & Hamagami, 2001).

The major point of interest in our study is the cross-lagged γ or coupling parameters, which model the predictive effects of depressive symptoms on subsequent change in perceptual speed ($\gamma_{Dep\rightarrow Speed}$), and the opposite predictive effect of perceptual speed on subsequent change in depressive symptoms ($\gamma_{Speed\rightarrow Dep}$). With slight variations to these γ parameters, we can directly compare the goodness-of-fit indices of four statistically nested models. First, a model that freely estimated both coupling parameters $\gamma_{Dep\rightarrow Speed}$ and $\gamma_{Speed\rightarrow Dep}$ was referred to as the full coupling model. This model was the most complex model tested and served as reference by which to judge all other models that estimated fewer parameters. Two of the models tested unidirectional hypotheses: in coupling $\gamma_{Dep\rightarrow Speed}$, only the predictive effects of depressive symptoms to change in perceptual speed were estimated; conversely, in coupling $\gamma_{Speed\rightarrow Dep}$, only the prediction of perceptual speed for depressive symptom change was estimated. The final model predicted that neither variable predicted change in the other by setting both parameters to 0 (no coupling). Models were fit using *Mplus* (Muthén & Muthén, 1998–2007).

Results

The results are presented in three main parts. First, we report a comparison of the four models used to evaluate the various accounts of the relation between depressive symptoms and perceptual speed. Next, we focus on the full coupling model to illustrate the effects of the differential magnitude of the coupling parameters over time. Finally, we investigate whether controlling for baseline age, MMSE score, education, and chronic health conditions had an effect on the dynamic relations between depressive symptoms and cognition.

Comparison of Various Coupling Models

The goodness-of-fit model statistics from the four different versions of the BDCSM are presented in Table 2. Compared to the full coupling model, the only model that did not result in a significant loss of model fit was the $\gamma_{Dep\rightarrow Speed}$ model, which permitted cross-lagged effects from depressive symptoms to perceptual speed, but did not allow predictive effects of perceptual speed for depressive symptoms. This means that we can reject the unidirectional hypothesis that perceptual speed predicts change in depressive symptoms. In contrast, fixing the predictive effect of depressive symptoms for change in perceptual speed to zero, and only estimating the opposite pathway (i.e., $\gamma_{Speed\rightarrow Dep}$; perceptual speed predicting change in depressive symptoms) resulted in a highly significant loss in model fit. In addition, the no coupling model described the data less precisely than the full coupling model suggesting that we also cannot accept this model. We obtained similar results with the other goodness-of-fit statistics. Both the root mean square error of approximation and the comparative fit index were comparable between the full coupling and the $\gamma_{Dep\rightarrow Speed}$ model, but provided noticeably poorer fit for the two other models. Overall, nested model comparisons suggest that depressive symptoms predict changes in perceptual speed, whereas we found no evidence for the hypotheses that perceptual speed

Table 2
Goodness-of-Fit Statistics for Alternative Bivariate Dual Change Score Models of Depressive Symptoms and Perceptual Speed

Model	Goodness-of-fit indices			
	χ^2 (*df*)	$\Delta\ \chi^2$(*df*)	RMSEA	CFI
Unidirectional				
$\gamma_{Dep \rightarrow Speed}$	82.87 (46)	1.98 (1)	.026	.981
$\gamma_{Speed \rightarrow Dep}$	130.27 (46)	49.38 (1)****	.039	.957
Bidirectional				
Full coupling	80.89 (45)	—	.026	.982
No coupling	130.70 (47)	49.81 (2)****	.038	.957
Covarying age, education, cognitive status, and medical conditions				
Unidirectional				
$\gamma_{Dep \rightarrow Speed}$	121.28 (70)	3.28 (1)	.025	.980
$\gamma_{Speed \rightarrow Dep}$	171.85 (70)	53.85 (1)****	.035	.961
Bidirectional				
Full coupling	118.00 (69)	—	.024	.981
No coupling	171.88 (71)	53.88 (2)****	.034	.961

Note. Significance refers to loss in χ^2 assuming the full coupling model to be correct. RMSEA = root mean square error of approximation; CFI = comparative fit index.
**** $p < .0001$.

predicts subsequent changes in depressive symptoms or that the associations do not exist.

Differential Magnitude of Depressive Symptoms-Perceptual Speed Couplings

To appreciate the size of predictive effects of depressive symptoms for subsequent changes in perceptual speed, we present a series of graphical examples. To illustrate the differential magnitude of the coupling parameters and their effects over time, we varied the baseline sample means for one target variable by half a standard deviation (i.e., 5 T-score units) while keeping the baseline sample means for the other variable constant (Lövdén et al., 2005). This hypothetical scenario demonstrates how the coupling parameter of one variable predicts the estimated mean longitudinal trajectories of the other variable. The figures were produced using estimates from the full coupling model and the formulas $x[t] = 1 \times X_s + (1 + \beta x) \times x[t\text{-}1] + \gamma_{yx} \times y[t\text{-}1]$ and $y[t] = 1 \times Y_s + (1 + \beta y) \times y[t\text{-}1] + \gamma_{xy} \times x[t\text{-}1]$. Panel A of Figure 2 shows the model-implied change in depressive symptoms over 15 years, in the hypothetical case that all participants showed the same amount of depressive symptoms at baseline, but differed in their initial perceptual speed performance. As can be seen, varying initial levels of perceptual speed performance does little to alter the subsequent changes in depressive symptoms across time. In contrast, panel B shows the model-implied change of perceptual speed when the initial sample mean of depressive symptoms is varied by 0.5 *SD*, but the initial performance in perceptual speed is kept constant. There are clear differences as a result of varying the amount of baseline depressive symptoms. Individuals with more depressive symptoms at Wave 1 (+0.5 *SD*) showed steeper decline in perceptual speed, whereas individuals with fewer initial depressive symptoms (−0.5 *SD*) experienced shallower declines in perceptual speed.

The Role of Age, Education, Cognitive Status, and Medical Conditions

In a final step, we investigated whether individual differences in baseline age, education, cognitive status, and chronic health conditions would have an effect on dynamic associations between depressive symptoms and perceptual speed. The additional variables were included in the model as time-invariant covariates of the latent intercept and slope factors, thereby residualizing all model parameters for the covariates.

The pattern of results remained the same (see lower half of Table 2), with the coupling $\gamma_{Dep \rightarrow Speed}$ model not significantly differing from the full coupling model. Conversely, the two other models resulted in a significantly poorer fit to the data compared to the full coupling model. The directionality of the effects was also reflected by the coupling parameters in the full coupling model: The dynamic effect of depressive symptoms for perceptual speed change was −0.98 ($SE = .25$, $p < .01$), whereas the opposite coupling parameter $\gamma_{Speed \rightarrow Dep}$ was modest and not significant (0.09, $SE = .06$, $p > .10$). Further parameter estimates are shown in Table 3. Although inclusion of the covariates did not substantively alter the dynamics reported, they contributed significant proportions of overall explained variance to the model parameters (e.g., the level and change of perceptual speed, and level and change of depressive symptoms were: $R^2 = .22, .06, .03, .12$ with age only covaried vs. $R^2 = .34, .12, .15, .20$ with all four covariates).

Discussion

We examined time-ordered relations between depressive symptoms and perceptual speed across 15 years using models that allowed for the comparison of competing hypotheses of directionality. Our data best fit the hypothesis that depressive symptoms

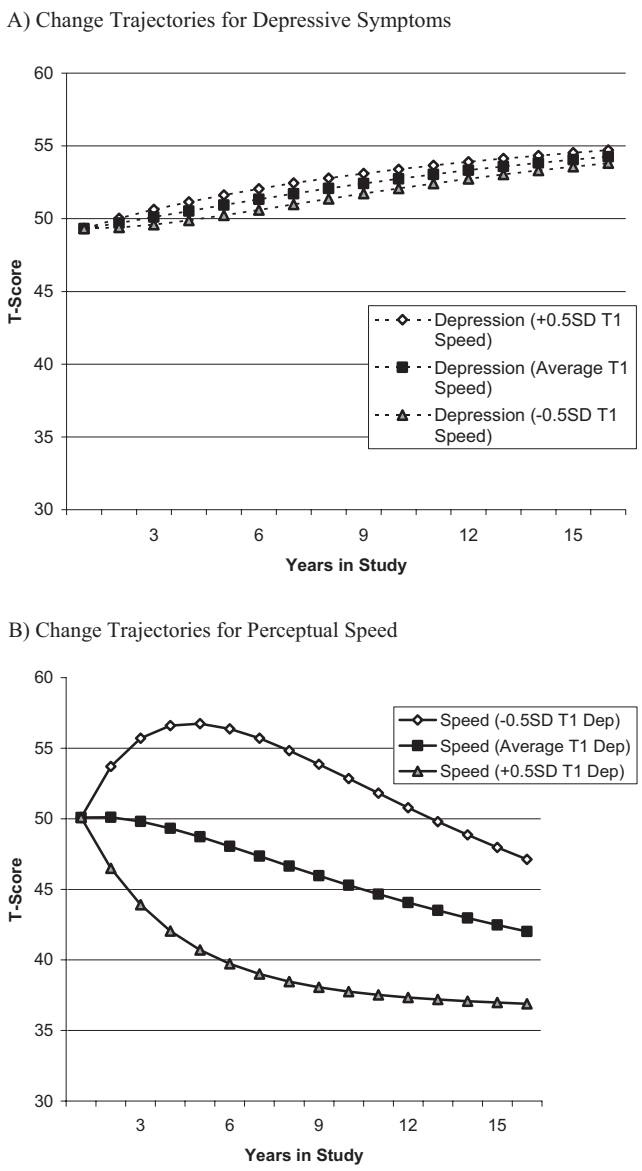

Figure 2. Graphical illustration of the differential magnitude of the coupling parameters and their effects over time. Model-implied means from the full coupling model were varied by half a standard deviation for one variable while the other was kept constant. Panel A shows change in depressive symptoms based on different initial perceptual speed scores. Panel B shows change in perceptual speed based on different initial depressive symptoms. Under the assumption of comparable perceptual speed performance at T1, Panel B shows that participants with few initial depressive symptoms (−0.5 *SD* T1 Dep) showed relatively shallow perceptual speed decline, whereas those with more depressive symptoms initially (+0.5 *SD* T1 Dep) showed relatively steep perceptual speed decline subsequently. In contrast, Panel A shows that depressive symptoms trajectories of change over time were minimally changed as a function of different initial levels of perceptual speed performance at T1.

predict subsequent changes in perceptual speed. This finding is consistent with studies suggesting that depression leads cognitive decline in older age (Jorm, 2001; Wilson et al., 2008; Yaffe et al., 1999), and demonstrates that a temporal relation exists even amongst relatively healthy older adults. It is worthwhile to note that the same pattern of results was found when participants with possible dementia at any wave across the 15 years were removed from the analyses (n = 218). Therefore, the importance of affect on normal cognitive aging should not be underestimated.

However, the mechanisms through which depressive symptoms relate to increased decline in perceptual speed are uncertain, and either end of the functioning spectrum may be driving the effect. Rather than more depressive symptoms acting as a risk for cognitive decline, it may be that having few depressive symptoms serves as a protective factor against decline. Regarding the highly prevalent comorbidity of depression and dementia (Forsell & Windblad, 1998), Jorm (2001) stated that the most likely explanation for why depression increases dementia risk involves all or one of three hypotheses: depression can be an early prodrome of dementia; depression brings forth the clinical manifestation of dementing diseases; or depression leads to hippocampal damage through a glucocorticoid cascade (Sapolsky, Krey, & McEwen, 1986). Further, Butters and colleagues (2008) proposed a reserve threshold theory whereby both depression and cognitive impairment can act to decrease cognitive reserve, thus increasing an individual's risk of developing other disorders. The coexistence of late-life depression and dementia may be the result of a number of possible pathways, beginning with either AD neuropathology, depression, or cerebrovascular disease, all of which can reduce reserve, and consequently result in the earlier expression of another disease. Therefore, although we found a unidirectional relation whereby higher depressive scores predicted lower processing speed, the exact cause of this leading relation is unknown. It is possible that similar investigations of entire samples with clinical depression or diagnosed dementia might elucidate the temporal association further. Finally, there are clearly other factors that influence the presence of both depression and dementia which further complicate any causal inferences that can be drawn.

Compared to other cognitive domains, processing speed undergoes the longest period of decline before dementia diagnosis (Thorvaldsson et al., 2011). Consequently, the temporal pathways associated with depressive symptoms may vary depending on the cognitive domain, and more importantly, the degree of cognitive impairment of the sample.

Similar to the research on depressive symptoms and cognitive decline, there have been inconsistent findings in the literature examining the directionality of the relation between depression and dementia (Brommelhoff et al., 2009; Chen et al., 1999; but see Jorm, 2001; Ownby, Crocco, Acevedo, John, & Loewenstein, 2006), and no study has directly compared competing temporal hypotheses linking depression and dementia. The present findings that depressive symptoms appear to lead changes in perceptual speed might hence be valuable to understanding the development of the link between these clinical conditions.

Our findings must be interpreted in light of a number of methodological limitations. First, sample attrition across time resulted in a relatively small sample at later waves, and both greater depressive symptoms and cognitive decline have been linked with study dropout and incomplete data in this sample (Anstey & Luszcz, 2002a, 2002b). Although the covariates included are typically informative of attrition and thus should have accounted for a good portion of this concern, the amount of cognitive decline and depressive symptoms are likely underestimated. Given this possi-

Table 3
Parameter Estimates From a Bivariate Dual Change Score Model (Full Dynamics) Between Depressive Symptoms and Perceptual Speed With Covariates

	Depressive symptoms		Perceptual speed			
Parameter	Estimate	*SE*	Estimate	*SE*	Estimate	*SE*
Fixed effects						
Intercept mean (μ_0)	46.24***	0.50	49.77***	0.51		
Slope mean (μ_S)	−9.73	8.30	67.72***	18.04		
Proportion (β)	0.13	0.11	−0.87***	0.14		
Random effects						
Intercept variance (σ^2_0)	42.06***	2.82	51.54***	3.13		
Slope variance (σ^2_S)	0.76	1.32	40.35	20.88		
Error variance (σ^2_e)	42.90***	1.50	20.96***	1.14		
Covariance/correlation						
Intercept ↔ slope	−4.32/−.63	4.30	9.61*/.16*	4.18		
Dynamics						
$\gamma_{Dep \rightarrow Speed}$					−0.98***	0.25
$\gamma_{Speed \rightarrow Dep}$					0.09	0.06
Covariance/correlation						
Dep intercept—Speed slope					34.38***/.72***	9.77
Speed intercept—Dep slope					−1.86/−.22	1.40
Dep intercept—Speed intercept					−13.03***/−.21***	2.04
Dep slope—Speed slope					−5.45/−.82	6.11

Note. Depressive symptoms and perceptual speed were standardized to the T metric using the T1 ALSA sample as the reference. Estimates are residualized for age, education, cognitive status, and medical conditions. Mean = 50, *SD* = 10. All estimates are unstandardized. Model fit statistics: χ^2 (*69*) = 118.00, CFI = .981; RMSEA = .024.
* $p < .05$. ** $p < .01$. *** $p < .001$.

ble attenuation of the range of scores, it is all the more compelling that the observed relationship is genuine. However, whether the nonrandom missingness led to an over- or under-estimation of the effect sizes of the across-domain couplings is unclear as this is dependent on which end of the functioning spectrum is driving the relation (i.e., risk versus protective factor). Next, we did not obtain a clinical diagnosis of depression, nor do we have clinical data on cardiovascular disease and other risk factors that have been linked to both depression and dementia (Anstey, von Sanden, Sargent-Cox, & Luszcz, 2007). However, the unidirectional pathway of depressive symptoms predicting declines in perceptual speed remained even when the number of medical conditions, including a range of cardiovascular health problems, was taken into account. Finally, there are a number of different factors that may have contributed to inconsistent findings between past studies, including variations in time frame, measurement lag, sample, measurement of depressive symptoms and cognition, and statistical analysis. Consequently, as all studies are, the present results are limited to the variables and factors presented.

From a methodological perspective, further caveats should be noted. Despite the ability of the bivariate dual change score model to permit a dynamic association to occur across time, very strict statistical assumptions were required. The relation between each variable at time (t) and change in the other variable between (t) and [$t + 1$] was assumed to be linear and identical across all participants and through time, and also limited to time-lags across one measurement occasion (i.e., it did not include lags over longer time intervals). Further, the best-fitting model was a function of the sample and the specific times of measurements, indicating that a different model might have been a better fit if the occasions were closer together. Most importantly, the demonstration of time-ordered association between the two variables does not equal causation.

The present study is the first to apply models which directly test the lead-lag relations between depressive symptoms and cognitive ability across time. We found higher levels of depressive symptoms predicted more rapid decline in perceptual speed, whereas there was no reliable predictive effect of perceptual speed on change in depressive symptoms. This pattern remained after controlling for age, education, baseline general cognitive status, and health. Therefore, depressive symptoms may be a risk factor for cognitive decline, rather than a prodrome of it.

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