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## Social Cognition in Alzheimer's disease: A separate construct contributing to dependence

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### Abstract

The extent to which social cognitive changes reflect a discrete constellation of symptoms dissociable from general cognitive changes in Alzheimer's disease (AD) is unclear. Moreover, whether social cognitive symptoms contribute to disease severity and progression is unknown. The current multicenter study investigated cross-sectional and longitudinal associations between social cognition, general cognition, and dependence in 517 participants with Probable AD. Participants were followed every 6-months for 5.5 years. Results from multivariate latent growth curve models adjusted for sex, age, education, depression, and recruitment site revealed that social cognition and general cognition were unrelated cross-sectionally and over time. However, baseline levels of each were independently related to dependence, and change values of each were independently related to change in dependence. These findings highlight the separability of social and general cognition

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in AD. Results underscore the relevance of considering social cognition when modeling disease and estimating clinical outcomes related to patient disability.

## Keywords

Alzheimer's disease; social cognition; cognition; dependence

## 1. INTRODUCTION

Social cognition has been described as a set of converging implicit and explicit processes that are engaged to understand or interpret the self in relation to others (Fiske, 1993; Forbes & Grafman, 2010). This set of processes forms the basis of the complex set of behaviors and mutually shared expectations that enable individuals to successfully interact with one another across a range of situations (Sollberger, Rankin, & Miller, 2010). In contrast to certain presentations of frontotemporal dementia (FTD), early Alzheimer's disease (AD) is frequently characterized by preserved social cognition (Rankin, Kramer, Mychack, & Miller, 2003). In fact, social cognition often remains intact into the moderate stages of the disease (Sabat & Gladstone, 2010; Sabat & Lee, 2010). However, a small subset of individuals with AD evidence marked changes in social cognition early in the disease, sometimes severe enough to elicit misdiagnoses of FTD. Even in cases that fit the typical AD profile with amnesic deficits being prominent, individuals have been shown to demonstrate impairment on objective social cognitive tests including Theory of Mind (ToM) and emotion recognition (Freedman, Binns, Black, Murphy, & Stuss, 2012; Miller et al., 2012), and in some cases these impairments can be commensurate with those in FTD. See (Harciarek & Cosentino, in press) for a review.

The extent to which social cognitive deficits represent a component of the disease that is separable from general cognitive deficits in AD has not been formally examined. Existing work has demonstrated that functional deficits and psychiatric symptoms (i.e., depression, psychosis, agitation), while related to general cognitive impairment in AD, are separable elements of the disease (Tractenberg, Aisen, Weiner, Cummings, & Hancock, 2006; Tractenberg, Weiner, Cummings, Patterson, & Thal, 2005; J. Zahodne, Ornstein, Cosentino, Devanand, & Stern, in press). Social cognitive symptoms may represent yet another specific manifestation of disease pathology that is anatomically and/or behaviorally distinct from general cognitive impairment. Indeed, social behaviors and executive functioning, while both frontally mediated, have distinct neural circuitry originating in the orbitofrontal and dorsolateral regions of the PFC, respectively, that map onto dissociable regions (e.g., dorsal versus ventral) of subcortical structures including the striatum, globus pallidus, and thalamus before projecting back to the originating PFC regions (Lichter & Cummings, 2001; Mega & Cummings, 1994). Behavioral evidence for these parallel but distinct frontal-subcortical circuits in neurodegenerative disease can be seen in the frequent dissociation between social cognition and general cognition in behavioral variant FTD (Eslinger et al., 2007; Libon et al., 2007; Lough, Gregory, & Hodges, 2001; Lough & Hodges, 2002; Narvid et al., 2009).

Alternatively, it has been suggested that social cognitive deficits, revealed on objective tests of emotion recognition and ToM, may stem from general cognitive deficits in AD

related to visuoception or executive functioning, for example (Shany-Ur et al., 2012). The first aim of this study was to examine the cross-sectional and longitudinal associations between subjectively rated social cognitive symptoms and general cognition in a large cohort of individuals with AD for the first time. A second related aim was to clarify the extent to which subjectively rated social cognitive symptoms, while potentially unrelated to general cognitive symptoms, have relevance for disease severity and course assessed with the Dependence Scale (Stern et al., 1994), a measure that has been recommended for modeling AD progression (McLaughlin et al., 2010).

## 2. MATERIAL AND METHODS

### 2.1 Participants and Procedures

The present sample included 517 patients diagnosed with probable AD and enrolled in the Multicenter Study of Predictors of Disease Course in Alzheimer's disease. Local Institutional Review Boards at all participating sites approved the study. Full study procedures are described elsewhere (Richards et al., 1993; Stern et al., 1993). In brief, patients were recruited in two waves at outpatient clinics and clinical research centers at four sites in the United States and Europe: Columbia University Medical Center (N=208), Johns Hopkins School of Medicine (N=147), Massachusetts General Hospital (N=124), and the Hôpital de la Salpêtrière in Paris, France (N=38). Diagnoses of probable AD were made using NINCDS-ADRDA criteria [13] at consensus conferences attended by at least two physicians specializing in dementia and one neuropsychologist. Complete inclusion and exclusion criteria for the study have been described previously (Richards et al., 1993; Stern et al., 1993). All patients were required to have mild dementia defined by a Modified Mini-Mental State Exam (mMMSE) score  $\geq$  30 (see description of this measure below), which is approximately equivalent to a Folstein MMSE score  $\geq$  16. Exclusion criteria were evidence for a cause of dementia other than AD, parkinsonism, stroke, alcoholism, schizophrenia, schizoaffective disorder, and electroconvulsive therapy within 2 years preceding study enrollment or a history of 10 or more electroconvulsive treatments in a single course.

### 2.2. Measures

Social cognitive symptoms were assessed at each occasion on a scale from 0–6 based on a subset of informant-rated items from the Blessed Dementia Rating Scale (Blessed, Tomlinson, & Roth, 1968) that query whether the patient is: 1) more stubborn than before and less able to adapt to change; 2) more self-centered than before, 3) unconcerned about others' feelings; 4) unable to control emotions; 5) easily angered; and 6) likely to make strange jokes or laugh at things that aren't funny. These items were previously shown to load together on a single factor that was independent from three other factors assessing symptoms related to general cognitive symptoms (e.g., remembering short lists; finding way around the house), apathy (e.g., less interest in starting new things, less likely to participate in hobbies), and basic self-care (e.g., eating, dressing, and bladder control) (Stern, Hesdorffer, Sano, & Mayeux, 1990).

Global cognitive status was evaluated at each occasion with the mMMSE (Mayeux, Stern, Rosen, & Leventhal, 1981). In addition to items from the MMSE (Folstein, Folstein, &

McHugh, 1975), the mMMSE includes items allowing for more comprehensive assessment of working memory, calculation, recall of the current and four previous presidents of the United States, confrontation naming, repetition, and visuoconstruction. The scale was translated and modified for assessments at the Paris site. Scores range from 0 to 57, with higher scores indicating better cognitive functioning.

Depressive symptoms were assessed at baseline with the Columbia University Scale for Psychopathology in AD (CUSPAD) (Devanand et al., 1992). The CUSPAD is a semi-structured interview conducted with an informant assessing the presence and severity of psychiatric symptoms over the past month. Depressive symptom severity scores were used in the present study to rule out the possibility that elements of the social cognitive index reflected depression. Scores range from 0 to 4, with higher scores indicating greater psychopathology. Good inter-rater reliability for concurrent ratings of the depressive symptoms portion of a single interview has been reported ( $\kappa=0.80$ ). (Devanand et al., 1992).

The main outcome measure was the Dependence Scale (DS) a 13-item instrument administered to a caregiver (Stern et al., 1994). The DS comprises 11 dichotomous items (e.g., “Does the patient need to be watched or kept company when awake?”) and two items scored on a 3-point Likert-type scale indicating the frequency of need (e.g., “Does the patient need reminders or advice to manage chores, do shopping, cook, play games or handle money?”) Scores range from 0 to 15, with higher scores indicating greater dependence. Interrater reliability, internal consistency, convergent validity, criterion validity, and sensitivity to change range from acceptable to excellent (Brickman et al., 2002; Stern et al., 1994).

### 2.3. Statistical Analysis

Data were analyzed in MPlus version 7 with a special case of structural equation modeling often referred to as latent growth curve (LGC) modeling using maximum likelihood estimation (Bollen & Curran, 2006; Muthen & Muthen, 1998). Missing data were managed with the full information maximum likelihood (FIML) method, which uses all available data for parameter estimation. This approach accumulates and maximizes casewise likelihood functions computed using all available data for each participant. Monte Carlo simulation has shown that FIML produces unbiased and more efficient estimates than alternative methods (e.g., listwise deletion, pairwise deletion, and similar response pattern imputation) (Enders & Bandalos, 2001). Models carry the assumptions of homogeneity of error variance and dependence of errors within each domain (i.e., function, cognition, depressive symptoms). A strength of LGC modeling is that it allows the study of multiple outcomes over time in a multivariate framework. The overall *level* (intercept) of and *amount of change* (slope) in each symptom type (i.e., dependence, cognition, social cognition) represented the key parameters. Additional information regarding parameter estimation in multivariate LGC and its application to the study of neurodegenerative disease are available elsewhere (Bollen & Curran, 2006; L. B. Zahodne et al., 2012). Model fit was assessed with the following, commonly-used statistics: chi square, root mean square error of approximation (RMSEA), comparative fit index (CFI), and Tucker-Lewis index (TLI). Smaller values of chi square

and RMSEA indicate better model fit. Values of CFI and TLI that are closer to 1 indicate better fit. Fit between nested models was compared statistically using the chi square test.

Model building proceeded in two broad stages. *First*, the trajectories of the three variables of interest were examined separately with *unconditional univariate growth curve models*. To characterize the functional forms of social cognitive, general cognitive and dependence trajectories, models that estimated only linear change were statistically compared to those that estimated both linear and quadratic change. *Second*, best-fitting univariate models were combined into a single, *conditional multivariate model*, in which obtained parameter estimates control for all included variables. In this model, correlations between initial levels and changes in the symptoms independent of the covariates (i.e., sex, age, education in years, recruitment site, and depression) can be estimated. In addition, covariate effects on both the overall levels of symptoms (intercepts) as well as on symptom changes (slopes) were examined. Certain covariates were centered to facilitate parameter interpretation. Specifically, values of 0 corresponded to the mean sample age of 74, and to 12 years of education, male sex, enrollment at the Columbia site, and absence of depressive symptoms at baseline.

### 3. RESULTS

#### 3.1. Descriptives

Participants had an average baseline age of 74.35 (SD = 8.66) and educational level of 13.61 (SD = 3.58) years. The ethnic distribution of the sample was 93% Caucasian, 6% African American, and 1% other ethnicities. 5% of the participants were Hispanic by self-report. 59.8% of the participants were female. Average baseline performance on the mMMSE was 37.63 (SD = 6.37). On average, caregivers reported that participants demonstrated 2.05 of 6 social cognitive symptoms at baseline, with a range of 0 to 6. Frequencies of individual symptoms at baseline ranged from 9% (making strange jokes) to 54% (more stubborn). Dependence scores were 5 out of 13 on average with a range of 0 to 12. See Table 1 for initial and change values of all variables. At least one follow-up assessment was available for 96.9% of the present sample. The average number of assessments was 7.7 (standard deviation = 3.3), indicating that the average participant was followed semiannually for over 4 years. Data from only the first 12 occasions (5.5 years) were included in the present study in order to maximize covariance coverage.

#### 3.2. Unconditional Univariate Models

Nested unconditional univariate models were built separately for the three outcome variables. In models allowing only linear change, social cognition (slope estimate=0.213;  $p = .001$ ), cognition (slope estimate=-1.384;  $p < .001$ ), and dependence (slope estimate= 1.819;  $p < .001$ ), worsened over the study period. Allowing for curvilinear change significantly improved model fit for social cognition ( $\chi^2(4) = -149.15$ ,  $p < .001$ ), cognition ( $\chi^2(4) = -503.18$ ,  $p < .001$ ), and dependence ( $\chi^2(4) = -201.80$ ,  $p < .001$ ). Thus, models including both linear and quadratic slopes were retained for subsequent model building.

Parameter estimates in the best-fitting unconditional univariate LGC models are shown in the upper panel of Table 1. Intercepts (levels) refer to latent variables derived from all 12

occasions that reflect estimated initial levels of the outcomes independent of the growth process, not merely baseline scores. Linear slopes can be interpreted as the constant rates of change over time. Quadratic slopes can be interpreted as changes in the rates of change over time. As shown in Figure 1, social cognition, cognition, and dependence worsened over the study period, but leveled out over time. However, significant residual variances in both intercepts and slopes (all  $p$ 's<.001) in all three models indicate substantial individual differences both in initial levels and trajectories of social cognition, cognition, and dependence. Such residual variance is a precondition for further model building.

### 3.3. Conditional Multivariate Model

The three best-fitting univariate LGC models were combined into a single multivariate model, and five time-invariant predictors measured at baseline (i.e., age, sex, education, depression, and recruitment site) were added. The model provided the following fit statistics:  $\chi^2(747)=1253.652$  ( $p<.001$ ); CFI=0.96; TLI=0.95 and RMSEA=.037 (.033, .040). Intercepts shown in the lower panel of Table 1 can be interpreted as estimated initial values when all covariates are set to 0 (i.e., age=74; education=12 years; sex=male; site=Columbia; baseline depressive symptoms=0). As shown, worsening over time was evident for social cognition, cognition, and dependence after controlling for these variables.

Correlations between the factors in the conditional model are shown in Table 2. After controlling for the covariates, initial levels of each variable were related to subsequent change in these domains. Initial levels of both social cognition and cognition were related to initial dependence level, although they were not related to each other. Initial social cognitive status was not associated with subsequent changes in cognition or dependence. However, rates of change in social cognition and change in dependence over time were *correlated* (see Table 3). Rates of change in cognition were also related to rates of change in dependence.

Older age was independently associated with greater initial dependence (standardized parameter estimate=0.21; SE=0.05;  $p<.001$ ) but slower increase in social cognitive symptoms (standardized parameter estimate=1.51; SE=0.06;  $p=.017$ ) and slower general cognitive decline (standardized parameter estimate=0.24; SE=0.05;  $p<.001$ ). Female sex was associated with fewer initial social cognitive symptoms (standardized parameter estimate=-0.12; SE=0.05;  $p=.024$ ) but more rapid increase in social cognitive symptoms over time (standardized parameter estimate=0.16; SE=0.06;  $p=.010$ ), and lower initial cognition (standardized parameter estimate=-0.15; SE=0.05;  $p=.001$ ). Lower level of education was associated with lower general cognitive status at baseline (standardized parameter estimate=0.20; SE=0.05;  $p<.001$ ) but not with the rate of cognitive decline in any domain. Participants enrolled at different recruitment sites did not differ in initial levels or in rates of change in any of the three outcomes.

## 4. DISCUSSION

Social cognitive symptoms, while most commonly associated with FTD, also occur in individuals with AD. In the current study, individuals demonstrated an average of two out of six social cognitive symptoms at baseline (with a range of 0 to 6), as reported by a knowledgeable informant. Social cognitive symptoms increased an average of 0.25 points

per year. A primary question in this study was whether social cognitive symptoms map onto general cognition or whether they represent a separable component of the disease. Multivariate latent growth curve analysis demonstrated that social and general cognition were unrelated both at baseline and over time, indicating that social cognitive changes in AD are not a byproduct of changes in general cognition but rather reflect a distinct constellation of symptoms.

This dissociation is consistent with a longstanding literature detailing the divergent effects of damage to orbitomedial versus dorsolateral regions of the PFC, as well as the subcortical regions in their corresponding circuitry. Indeed, the separability of regions within the PFC is reinforced throughout their respective subcortical circuitry, such that individual loops originating in each area pass through discrete regions of the basal ganglia and thalamus corresponding to the origin of the fibers (Lichter & Cummings, 2001; Mega & Cummings, 1994). Damage to the DLPFC and its subcortical projections including the dorsal caudate and mediodorsal thalamus have primarily cognitive and specifically dysexecutive effects (Mendez, Adams, & Lewandowski, 1989; Sandson, Daffner, Carvalho, & Mesulam, 1991). In contrast, changes in personality, here conceived of as social cognitive symptoms, have long been reported in the context of orbitofrontal compromise as well as ventromedial caudate lesions (Blumer & Benson, 1975; Eslinger & Damasio, 1985; Reitman, 1946). More recent imaging work in both healthy adults and individuals with FTD has reinforced the role of orbitofrontal areas for social cognitive abilities including emotion recognition and ToM (Bertoux et al., 2012). It has also been suggested that the anterior cingulate contributes to social cognition through the identification and subjective experience of emotion (Hornak et al., 2003). To the extent that degenerative processes differentially affect these regions or their corresponding circuits, dissociations in general versus social cognition are likely to arise. This dissociation has been reported frequently in the behavioral variant of FTD, in which many aspects of general cognitive performance including executive functioning can remain intact despite marked changes in interpersonal behavior (Eslinger et al., 2007; Libon et al., 2007; Lough et al., 2001; Lough & Hodges, 2002; Narvid et al., 2009).

The current study also examined the relevance of social cognitive changes for dependence, an index of disease severity. The dependence scale accounts for more variance in clinical outcomes than cognitive scores alone (McLaughlin et al., 2010). Indeed, when compared with other markers of disease including the MMSE, Disability Assessment in Dementia (DAD), and CDR, the DS accounted for the highest amount of variance in a variety of economic and quality of life outcomes for patients and caregivers. It has thus been recommended for use in models of long term disease progression in AD. In the current study, social cognition contributed to higher levels of dependence at baseline, independent of general cognition. Moreover, rate of change in social cognition correlated with rate of change in dependence across participants, independent of rate of general cognitive change. Importantly, it is not the case that dependence would necessarily be related to social cognition as the large majority of the dependence scale items measure non-social dependencies (i.e., needs help managing chores, remembering information, bathing, eating, toileting, etc.). As such, social cognitive symptoms appear to have direct implications for a primary disease outcome via a route that is separate from the effects of general cognitive deficits. This dissociation is consistent with the idea that multiple discrete factors constitute

disease severity and progression (Tractenberg et al., 2006; Tractenberg et al., 2005). Using a latent variable modeling approach in two separate samples of individuals with AD, Tractenberg and colleagues (2006) found that the model which best fit the data included a general neurologic factor as well as three symptom factors including cognition, function, and behavior. That is, while there was a seemingly common underlying disease process accounting for the shared variance across discrete symptom types, each set of symptoms also acted as separate constructs independently relevant for disease severity. Social cognitive impairment may represent another element of disease that should be considered when estimating clinical outcomes related to patient disability, and potentially other important outcomes such as family impact and healthcare utilization.

A final issue addressed in this study was whether or not social cognitive symptoms herald a more aggressive disease course. Existing work has demonstrated that atypical presentations of AD, including those that implicate a disproportionate involvement of frontal and/or subcortical regions, are often associated with more rapid general cognitive and functional decline (Mez et al., in press; Scarmeas et al., 2005). Moreover, individuals with behavioral variant FTD appear to decline more quickly than other subtypes of FTD (Chan et al., 2001; Mioshi, Hsieh, Savage, Hornberger, & Hodges, 2010; Roberson et al., 2005) suggesting that involvement of the brain regions and/or genetic factors that underlie social cognitive changes may reflect an aggressive variant of disease, and this could be true for AD as well. Alternatively, frontal lobe involvement could hasten disease course secondary to behavioral problems that could lead to overmedication or other differences in quality of medical care (Roberson et al., 2005). However, the severity of social cognitive symptoms at baseline did not predict future change in dependence in the current study. Therefore, although such symptoms contribute to and track disease severity over time, their cross-sectional presence in early AD appears to have little prognostic value with respect to future disease course, and this was in contrast to the predictive utility of general cognitive symptoms for change in dependence. The lack of a predictive relationship between social cognitive symptoms and change in dependence was somewhat unexpected given their association at baseline, and the previously reported prognostic value of other frontally mediated symptoms in both AD and FTD.

Nevertheless, the current findings highlight the distinct nature of social cognitive and general cognitive changes in AD, and underscore the relevance of each aspect of the disease process for level of dependence. Models of disease should consider social cognitive symptoms and change in these symptoms as a separate process contributing to overall markers of severity such as dependence. Limitations of this study include the use of a retrospective measure of social cognition that was: 1) limited in scope, lacking information about a greater range of socially inappropriate behaviors; 2) subjective and therefore potentially influenced by caregiver bias; and 3) inclusive of several common behaviors in AD that may have limited the specificity of the measure. However, the social cognitive index compiled for the current study was based on a factor analysis in which these symptoms clustered together and were separable from caregiver reported symptoms related to general cognition, apathy, and basic self-care. Moreover, while objective measures of social cognition provide non-biased information, family report is a primary means by which clinicians determine the presence of social cognitive changes in a patient. Furthermore,



activation in brain regions underlying social cognition including the orbitofrontal and medial frontal cortex has been shown to vary parametrically with objective indices of social cognition including ToM as well as subjective indices such as an individual's social network size, suggesting that these different aspects of sociality may share a similar neural basis (Lewis, Rezaie, Brown, Roberts, & Dunbar, 2011). The investigation of social cognitive symptoms in this large longitudinal cohort of individuals with AD complements the primarily small experimental and case studies that have examined this topic thus far. It will be important for future work to include social cognitive symptoms in models of disease along with markers of cognition, function, and psychiatric symptoms. Moving forward, it will also be important to determine the extent to which social cognitive symptoms are separable from behavioral (e.g., agitation) and psychiatric symptoms.

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## Research in Context

### Systematic Review

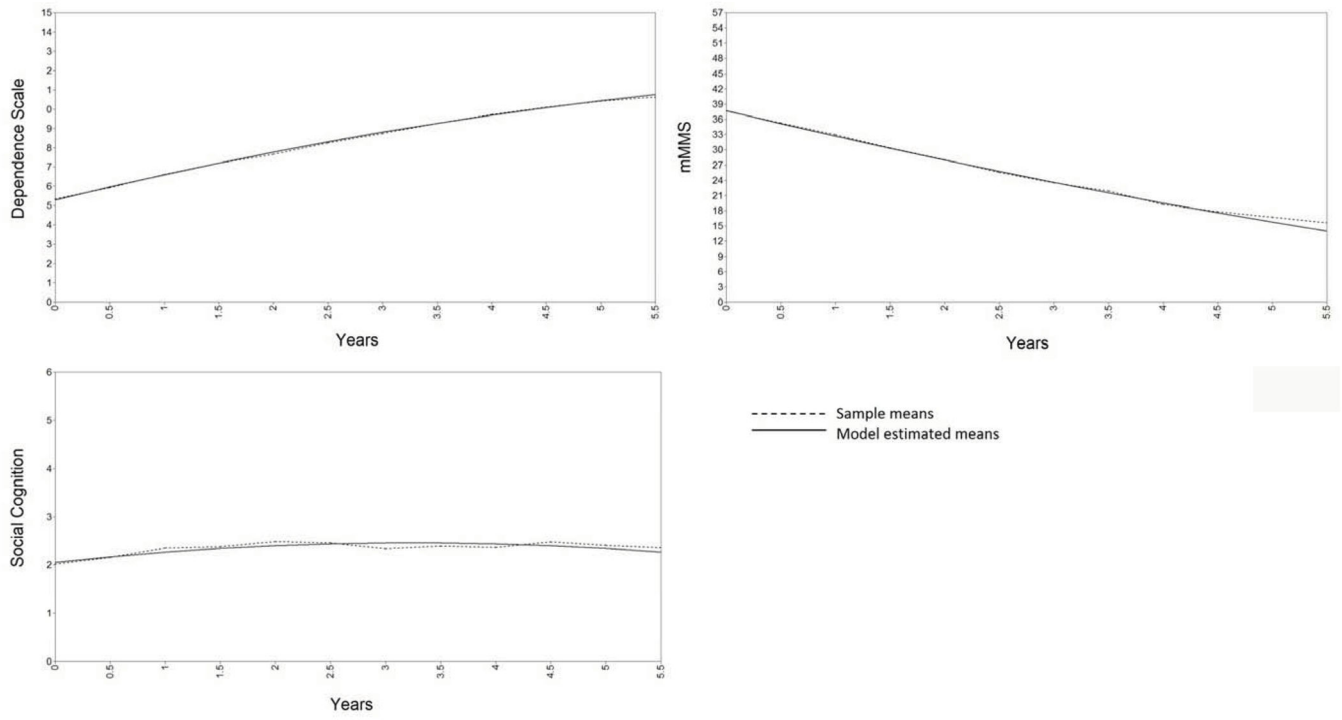
The accumulated knowledge related to the focus of the current manuscript was established by a comprehensive PubMed literature search related to social cognition in Alzheimer's disease and frontotemporal dementia.

### Interpretation

Current findings suggest that social cognitive changes in AD are not byproducts of cognitive change, but represent a distinct manifestation of disease. Second, the presence of social cognitive changes relates to higher levels of dependence at individual points in time, as well as over time. These findings implicate a role for social cognition in models of disease.

### Future Directions

To expand these findings, future studies should evaluate the interplay between social cognitive deficits and a range of psychiatric symptoms to determine the extent to which these manifestations of disease are independent. Additionally, work evaluating the relationship between subjective and objective social cognitive measures is needed to fully understand whether these different assessments tap into a similar construct or whether they evaluate dissociable abilities.



**Figure 1.** Unconditional univariate growth curves for the three dependent variables of interest. *Note.* mMMS = modified Mini Mental State Exam.

**Table 1**

Unstandardized Parameter Estimates

TnQ Table1	Intercept (SE)	Variance (SE)	Intercept (SE)	Variance (SE)	Intercept (SE)	Variance (SE)
<i>Unconditional Univariate<sup>a</sup></i>						
SC	2.050(0.067)**	1.929(0.168)**	0.253(0.056)**	0.784(0.099)**	-0.039(0.011)**	0.026(0.004)**
mMMS	37.699(0.284)**	37.874(2.617)**	-5.181(0.296)	34.209(2.808)**	0.159(0.057)*	0.947(0.105)**
Dependence	5.289(0.114)**	4.465(0.421)**	1.386(0.085)**	1.896(0.222)**	-0.071(0.014)**	0.044(0.006)**
<i>Conditional Multivariate<sup>b</sup></i>						
SC	2.047 (0.157)**	1.813(0.160)**	0.313(0.129)*	0.788(0.100)**	-0.048(0.026)	0.027(0.004)**
mMMS	38.515(0.614)**	34.230(2.442)**	-4.554(0.665)**	33.931(2.805)**	-0.053(0.131)	0.998(0.109)**
Dependence	4.972(0.246)**	3.859(0.377)**	1.133(0.197)**	2.004(0.230)**	-0.003(0.034)	0.045(0.006)**

Note. SE=Standard Error; SC= Social Cognition (range: 0 to 6); mMMS=modified Mini Mental State Exam (range: 0 to 57); Intercepts (levels) refer to latent variables derived from all 12 occasions that reflect estimated initial levels of the outcomes independent of the growth process, not merely baseline scores. The variance in the intercepts and slopes refers to the degree of individual differences in estimated initial levels and trajectories, respectively.

<sup>a</sup> Parameters estimated in three separate univariate models;

<sup>b</sup> Parameters estimated in a single conditional multivariate model;

\*  $p < .05$ ;

\*\*  $p < .001$

**Table 2**  
Standardized Between-Persons Associations Between the Latent Factors in the Conditional Multivariate Model

	SC			mMMS			Dependence		
	Intercept	Linear	Quadratic	Intercept	Linear	Quadratic	Intercept	Linear	Quadratic
<i>SC</i>									
Intercept	-								
Linear	-0.407**	-							
Quadratic	0.207*	-0.920**	-						
<i>mMMS</i>									
Intercept	-0.082	-0.072	0.132	-					
Linear	0.000	-0.137	0.235*	.393**	-				
Quadratic	0.005	-0.013	-0.088	-0.395**	-0.856**	-			
<i>Dependence</i>									
Intercept	0.318**	-0.049	-0.106	-0.378**	-0.210**	0.204*	-		
Linear	-0.046	0.197*	-0.259*	-0.264**	-0.695**	0.558**	-0.144*	-	
Quadratic	-0.029	-0.132	0.225*	0.213*	0.639**	-0.648**	0.030	-0.904**	--

Note. SC= Social Cognition; mMMS=modified Mini Mental State Exam;

\*  $p < .05$ ;

\*\*  $p < .001$