The Role of Chronic Illness in Theory of Mind Performance in Older Adults

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Laura Cecilia Walzak
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Approval

Name: Laura C. Walzak  
Degree: Master of Arts  
Title: The Role of Chronic Illness in Theory of Mind Performance in Older Adults  

Examining Committee:  
Chair: Dr. Rachel Fouladi  
Associate Professor  
Department of Psychology

Dr. Wendy J. L. Thornton  
Senior Supervisor  
Associate Professor

Dr. Allen Thornton  
Supervisor  
Professor

Dr. David Cox  
Supervisor  
Associate Professor

Dr. Colette Smart  
External Examiner  
Associate Professor  
Department of Psychology  
University of Victoria

Date Defended/Approved: December 13, 2016
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Abstract

Theory of Mind (ToM) reflects the ability to reason about mental states in order to understand and predict behavior. Research has identified links between increased pulse pressure, a measure of vascular health, and reduced ToM in older adults. Furthermore, previous findings suggest that cognitive ToM is particularly vulnerable to increased pulse pressure. However, to date, the relationships between other chronic vascular and nonvascular conditions and reduced ToM are unknown. We aimed to investigate the effects of vascular and nonvascular illness burden on cognitive and affective ToM in $N = 86$ older adults (59 females; 27 males, $M = 72$ years). While vascular illness burden emerged as a significant predictor of older adults’ ToM, nonvascular illness burden was not significantly associated with ToM. Further, executive functioning and semantic memory mediated the relationship between vascular illness burden and cognitive ToM. Our findings highlight the specific importance of considering vascular health as a risk factor for declines in ToM in later life, beyond pulse pressure. Further elucidation of the associations between health, neurocognition and ToM will be valuable in developing effective interventions for older adults given the high prevalence of vascular illness in later life.

Keywords: theory of mind; aging; cognition; neuropsychological ability; vascular health
Dedication

This thesis is dedicated to my parents; my accomplishments would not be possible without their sacrifices, hard work and endless belief in my dreams. Thank you for encouraging me to aim for nothing less than the moon, and for reminding me to never underestimate the power of a good laugh.
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INTRODUCTION

Theory of Mind (ToM) reflects the ability to understand another’s perspective and be able to reason about it – essentially, the ability to put oneself “in another’s shoes.” Also called mentalizing, mental state reasoning, or cognitive empathy, ToM is a valuable tool of social cognition that aids in understanding others (Premack & Woodruff, 1978). ToM is of particular interest in aging populations because older adults tend to demonstrate declines in these abilities relative to younger adults (Moran, 2013), and such declines may hold important consequences for everyday life. For example, reduced social cognition in later life has been linked to lack of social engagement, reduction of social activities and declines in independence (Bailey & Henry, 2008; Fett et al., 2011). Further, failure to understand the mental states of others may have implications for older adults in interpersonal relationships, everyday interactions and more serious transgressions such as increased susceptibility to fraud and financial scams.

ToM has been associated with a selection of cognitive abilities across the lifespan, but variability in other aspects of cognition does not fully account for reductions in ToM in later life (Moran, 2013). ToM skills emerge following the development of foundational neurocognitive skills (e.g., executive functioning) in childhood and neuroscience perspectives suggest that ToM is compiled from more basic neurocognitive processes (Saxe & Powell, 2006). Furthermore, mounting evidence supports the delineation of ToM into distinct affective and cognitive domains, with indications for this functional dissociation at both the neuropsychological and neuroanatomical levels (Wang & Su, 2013; Shamay-Tsoory & Aharon-Peretz, 2007). Affective or “hot” ToM (A-ToM) refers to the ability to understand and reason about emotional states without necessarily experiencing them (Duval et al., 2011), while cognitive or “cold” ToM (C-ToM) describes the ability to understand and reason about cognitive states including beliefs, motivations and meta-cognitive concepts (e.g., sarcasm, faux pas, white lies; Kalbe et al., 2010).
In general, age differences favouring younger adults on a variety of ToM tasks are evident around the age of 60 (Cavallini, Lecce, Bottirolì, Palladino, & Pagnin, 2013; Henry et al., 2013; Pardini & Nichelli, 2009; Sandoz et al., 2014) and persist across both ToM domains (e.g., Fischer et al., 2016). Further, A-ToM and C-ToM have dissociable components that are differentially associated with patterns in cognitive functioning; reduced C-ToM has been linked to age-related declines in executive skills (Wang & Su, 2013), abstract reasoning (Ahmed & Miller, 2013), attention and working memory (McKinnon & Moscovitch, 2007), and semantic and episodic memory (Fischer et al., 2016; Fischer et al., 2014). Poor A-ToM abilities are associated with reduced executive functioning (Fischer et al., 2016; Wang & Su, 2013) and semantic memory (Fischer et al., 2016; Peterson & Miller, 2012; see Appendix B for additional discussion on the differential associations in ToM research). However, associated reductions in more basic neurocognitive abilities account for less than 20% of the variance in ToM (Ahmed & Miller, 2011; Bernstein, Thornton, & Sommerville, 2011; Fischer et al., 2014). Therefore, recent research has addressed other non-cognitive predictors of ToM performance and everyday functioning to better understand risk factors for declines in later life.

Recent findings suggest that vascular health (a well established predictor of cognitive variability in aging; Spiro & Brady, 2011) may account in part for ToM declines in older adults (Fischer et al., 2014; Fischer, O’Rourke, & Thornton, 2016). However, it remains unclear as to how the effects of other health risks, including cumulative health risks interface with ToM in later life. In the current study we aimed to clarify the associations between multiple health predictors and the maintenance and decline of ToM in older adulthood, and to examine the mediating influence of neurocognitive performance on the association between health predictors with cognitive and affective ToM.

**Illness Burden as a Predictor of ToM**

Approximately three out of every four Canadian seniors report having at least one chronic health condition, and one quarter of older adults reported a diagnosis of three or more conditions (i.e., multi-morbidity; Public Health Agency of Canada, 2010). Mounting evidence suggests that health status, rather than increasing age, predicts older adults’ access to primary healthcare (Canadian Institute for Health Information, 2011) as well as
other aspects of daily functioning, such as driving (Tuokko et al., 2013). Such findings indicate that age may be a proxy variable for underlying health status, which may in turn affect brain integrity and neurocognition, impacting social cognition and everyday functioning. Vascular diseases are among the leading contributors to mortality (World Health Organization, 2009) and represent a major public health concern due to their high prevalence in later life. Further, vascular conditions are associated with the development of Alzheimer’s disease, vascular cognitive impairment, and concurrent global neurocognitive difficulties (Nagai, Hoshide, & Kario, 2010; Birns & Kalra, 2009).

Hypertension has been identified as a key predictor of neurocognitive performance in older adults (Morra, Zade, McGlinchey & Milberg, 2013, Fischer et al., 2014, Fischer et al., 2016), notably in domains that are also linked to reduced ToM (e.g., executive functioning, episodic memory and processing speed; Mahy et al., 2014; McKinnon & Moscovitch, 2007; Phillips et al., 2011; Singer, Trollor, Baune, Sachdev, & Smith, 2014; Yassar et al., 2011). An emerging area of neuropsychological research that has garnered recent interest is how health factors such as hypertension affect more distal aspects of cognition (i.e., ToM; Fischer et al., 2014, 2016). Historically, studies examining blood pressure as a risk factor for cognitive decline have operationalized high blood pressure by following clinical guidelines that require elevations of both systolic (SBP) and diastolic (DBP) blood pressure (i.e., ≥ 140/90mmHG; American Heart Association, 2016) or a dichotomous diagnosis of hypertension (i.e., hypertensive vs. non-hypertensive; Thornton et al., 2007). However, older adults are more likely to experience isolated systolic hypertension in which SBP is clinically elevated (>140mmHg), but DBP falls within normal range (<90mmHg). Isolated systolic hypertension is the most common form of high blood pressure in older adults and is associated with serious health problems, but is not recognized under typical high blood pressure guidelines (Ogihara et al., 2010). As a result, contemporary research has underscored the advantage of pulse pressure (SBP – DBP) as an indicator of cognitive change given its parsimony between high SBP and low DBP. Pulse pressure is a marker of arterial stiffening (Waldstein et al., 2008) and shows strong predictive value for cardiovascular events and morbidities (Chobanian et al., 2007; Mancia et al., 2007). High pulse pressure (>60mmHg; Singer et al., 2014) signifies loss of elasticity in arterial walls due to changes in large central arteries at the cellular and structural levels (Nichols et al., 2011) and predicts worse neurocognitive performance, including ToM, in healthy older adults (Fischer et al., 2014).
Expanding upon previous work linking higher pulse pressure to reduced ToM (Fischer et al., 2014), Fischer and colleagues (2016) developed a comprehensive model to assess the relative influence of neurocognitive performance, pulse pressure and biological sex on cognitive and affective ToM in younger and older adults. Their results implicated several neurocognitive predictors of C-ToM (namely, semantic memory and episodic memory) and A-ToM (namely, semantic memory and executive functioning) and suggested that increased pulse pressure directly predicts lower C-ToM among older adults, but not A-ToM (Fischer et al., 2016). These findings highlight the importance of considering health, in this case specifically pulse pressure, as a potential mechanism for age-related reductions in ToM.

Despite the utility of pulse pressure as a marker of systemic vascular risk, other potential chronic illnesses or combinations of illnesses have been unexplored to date. This gap in the literature is critically important because the biological mechanisms that likely underlie the association between pulse pressure and ToM (namely, the effects of arterial stiffening on integrity of subcortical circuits and resultant elevated risk of cognitive impairment) are shared by other vascular conditions as well (e.g., diabetes, high cholesterol, obesity, cardiovascular disease; Kirkman et al., 2012). Hypertension, high cholesterol, and diabetes have all been implicated as risk factors for cardiovascular disease (Public Health Agency of Canada, 2010) and are highly comorbid with each other (Perk et al., 2012; Walker, Zariwala, Holness, & Sugden, 2007), motivating their inclusion in the present study. Diabetes is also linked to worse cognition; older adults with diabetes are 50% more likely to develop dementia than those without (Mayeda, Whitmer, & Yaffe, 2015), and are also more likely to experience cognitive impairment across global neuropsychological domains (Geijseelaers, Sep, Stehouwer, & Biessels, 2015; Yaffe et al., 2012; Yeung, Fischer & Dixon, 2009). Importantly, research suggests an accelerated decline in global cognition in individuals presenting with multiple vascular conditions; for example, Hassig and colleagues (2004) examined non-demented older adults with a) hypertension, b) diabetes, c) both, or d) neither, and found steepest declines in global cognition over a two-year period in individuals with both comorbid conditions. The combination of major vascular risk factors also contributes to the development of chronic systemic inflammation, linked to adverse cognitive outcomes in older adults (Fabbri & Rabe, 2007). In sum, the strong associations identified between vascular health and
neurocognition highlight the value of further extending the role of vascular illness to another neurocognitive domain, ToM.

Although unaddressed in ToM literature to date, non-vascular illnesses have also been linked to neurocognitive decline in older adults. For example, osteoporosis is associated with worse cognitive function and increased risk of Alzheimer's disease among older adults (Bailon et al., 2012; Wilkins et al., 2006). Osteoporosis is also highly comorbid with chronic obstructive pulmonary disease (COPD) in up to 70% of patients and can result in chronic inflammation (Fabbri & Rabe, 2007), a key risk factor for poor cognition as discussed above. Thyroid imbalance also predicts poor cognitive performance in older adults (Samuels, 2014; Gussekloo et al., 2004; Wijsman et al., 2013;), particularly in the memory, attention and visuospatial organization domains (Begin, Langlois, Lorrain & Cunnane, 2008); however, vascular health may underlie this relationship in a secondary manner given that abnormal thyroid function affects serum cholesterol levels, heart rhythm and rate, ventricular function, and risk of coronary artery disease (Cappola et al., 2006; Joffe, Pearce, Hennessey, Ryan, & Stern, 2013). While it is difficult to parse out the impact that non-vascular illnesses may have on cognition due to the overlapping mechanisms of disease and frequency of comorbidities in later life, research has identified several nonspecific factors related to chronic illness which influence cognitive decline among older adults. For example, comorbid chronic illnesses are linked to frailty and a plethora of negative functional outcomes including disability, loss of independence, increased incidence of falls, hospitalization, and death (Clegg, Young, Iliffe, Rikkert, & Rockwood, 2013; Fried et al., 2001; Tinetti, Fried, & Boyd, 2012). Multiple chronic illnesses are also associated with sleep disorders such as insomnia, which negatively impact cognition as well as mood and everyday functioning (Pandharipande et al., 2013).

More broadly, health conditions that cause debilitating pain (e.g., rheumatoid arthritis and osteoarthritis) are associated with a number of negative outcomes in the biological, psychological and social context. Biomarkers of inflammation have been linked to increased pain and disease progression as well as poor cognition in older adults (Keefe et al., 2013). Persistent pain is also strongly associated with psychological distress (e.g., anxiety, depression, mood disorders), restricted mobility, and social isolation, factors that have far-reaching effects on cognition and can exacerbate existing comorbid health conditions in older adults (Abdulla et al., 2013; Moayedi et al., 2010). To summarize, the
demonstrated negative effect of chronic illnesses on cognitive functioning has implications for ToM in older populations. It is reasonable to predict that the relationship between chronic health conditions and associated declines in cognitive functioning may underlie age-related declines in ToM performance, as chronic illnesses are substantially more prevalent in older adults. Although the literature has identified a strong link between comorbid chronic conditions and poor cognition in older adults, it remains unclear as to whether certain types of illness burden have a differential impact on ToM.

Chronic diseases typically develop together (Boyd et al., 2005; Tinetti et al., 2012) and their overlapping effects have serious implications for overall cognitive health in later life. We chose to evaluate cumulative health risks as a summation of diagnoses in separate vascular and nonvascular subsets. Conceptualizing illness burden as a summation of diagnoses has been previously utilized in many studies examining the cumulative effects of comorbid diseases on various aspects of cognitive functioning (e.g., see Whitfield et al., 2004). More recently, an algorithm for developing severity indices has shown validity in a study of risk factors for neurocognitive functioning in HIV patients using beta weights (see Patel et al., 2013) and similar methodology was used in the current study to incorporate both vascular and nonvascular illness burden effects. While vascular risk is a well established contributor to cognitive decline and has been implicated as a predictor of C-ToM in older adults, nonvascular conditions have not been empirically examined in relation to ToM. Further, the current ToM literature lacks a comprehensive examination of the cumulative effects of both vascular and nonvascular illnesses on cognitive and affective ToM.

Objectives and Hypotheses

The present study is an extension of Fischer et al. (2016) re-analyzing the older adult sample to more closely evaluate the differential relationships between vascular and nonvascular illness and ToM. In this study, we examined the associations between chronic illness burden, neurocognitive functioning, and ToM ability among older adults through the following research questions: (1) Does vascular or nonvascular illness burden explain variance in C-ToM after the contributions of age, sex and neurocognitive functioning are considered? (2) Does vascular or nonvascular illness burden explain variance in A-ToM after age, sex and neurocognitive functioning are considered? (3) Are changes in
neurocognition crucial to any observed relationships between illness burden and ToM? That is, does neurocognition fully or partially mediate the relationships between illness burden and C-ToM or A-ToM?

Based on previous literature, we expected that vascular and nonvascular illness burden would account for significant variance in C-ToM among older adults beyond age, sex and neurocognitive functioning. If reduced C-ToM is linked to vascular health burden, individuals with a greater vascular illness burden were expected to show poorer C-ToM performance. However, if this relationship is influenced by more general health factors (e.g., nonspecific illness-related issues including pain, inflammation, lack of mobility, reduced social engagement), nonvascular illness burden was also expected to show significant contributions to C-ToM. Although less is known about predictors of A-ToM and their underlying mechanisms, we expected that greater vascular and nonvascular illness burden would also predict poorer A-ToM performance over and above the effects of age, sex and neurocognitive functioning. Concerning neurocognitive functioning as a mediator, we anticipated that executive functioning, semantic memory and episodic memory would partially account for the relationship between vascular/nonvascular illness burden and poorer ToM performance.
METHOD

Participants

The study utilized previously collected data as part of a larger project in the Cognitive Aging Laboratory at Simon Fraser University (SFU). Study participants (N = 86, M = 71.40, SD = 5.46, 59 females and 27 males) were recruited from the metro Vancouver area through advertisements posted widely throughout the community. The age range of 60 years or higher was selected to ensure adequate sampling of the older population and due to consistently higher rates of vascular illness among this cohort (Public Health Agency of Canada, 2010). We required that all participants have a minimum grade six education to ensure that reading level was adequate for the questionnaire and ToM protocols. Other eligibility criteria included capability of providing informed consent, English fluency and no impairments in vision, hearing, or other sensory/motor functions that could interfere with testing (Thornton et al., 2007).

The study employed a cross-sectional design in which participants attended the lab on one occasion and underwent a battery of physiological and neuropsychological tests. All participants were tested individually during a two and a half hour session with trained graduate students at the SFU Burnaby or SFU Surrey campus. Participants also completed questionnaires to gather information about their demographic details and medical history. All study protocols were approved by the SFU Research Ethics Board.

Health Measures

**Pulse Pressure.** Blood pressure readings were obtained prior to administration of the neurocognitive and ToM measures. Pulse pressure was measured using systolic (SBP) and diastolic (DBP) parameters (pulse pressure = SBP – DBP). We obtained four blood pressure readings for each participant on the nondominant arm using an automatic oscillometric upper arm monitor (Microlife BC 3AC1-1PC). An initial reading was taken to orient the participant to the procedure, followed by three additional consecutive readings after a five-minute delay. Each reading was separated by a one-minute rest interval. Average pulse pressure was calculated over the final three readings and re-coded
according to current guidelines indicating that high pulse pressure is > 60mmHg (Singer et al., 2014). Each participant was then assigned presence (1) or absence (0) of high pulse pressure to be used in the vascular illness burden variable.

**Illness Burden.** Chronic illness burden was assessed using a self-report health questionnaire developed by the Cognitive Aging Lab (Thornton et al., 2007; see Appendix D). Self-report information included demographic information (age, sex, education) and history of medical illnesses (vascular and nonvascular) and treatment. Self-report diagnosis by a physician was verified by prescription treatment and coded according to presence (1) or absence (0) of the condition under treatment. Participants were coded as having a condition if they self-reported a physician diagnoses that could be verified by current medication use (i.e., we requested that participants provide pill bottles or pharmacy receipts of any current medications for verification purposes). The vascular illnesses included in the analyses (hypertension, diabetes mellitus, high cholesterol, and pulse pressure) were chosen because their primary disease mechanisms directly involve the vascular system (Cohen et al., 2009, Triantafyllidi et al., 2009). Non-vascular chronic illnesses included in the analyses (thyroid dysfunction, rheumatoid arthritis, osteoarthritis, and osteoporosis) were chosen because they generally do not have primary disease mechanisms that directly involve the vascular system (Delles, 2007; Flynn, MacDonald, Morris, Jung & Leese, 2004), however they may be associated with pain, sleep disturbances, disability and reduced cognition in older adults (Fried et al., 2001; Miller et al., 2006; Begin et al., 2008; Wilkins et al., 2015).

As indicated in Table 1 (Appendix A), we entered these illnesses individually into a linear regression with global ToM (computed as a summation of C-ToM and A-ToM scores) as the outcome measure to determine the unique contribution of each illness through unstandardized beta weights. The beta weights were then used to weight the contribution of each variable according to its impact on the outcome variable with respect to the other variables in the model (as per methodology outlined in Patel et al., 2013). The resulting values were summed into composite variables for vascular and nonvascular illnesses respectively to create two indices of illness severity: vascular illness burden and nonvascular illness burden.
Neurocognitive Measures

Executive Functions. The Colour-Word Interference subtest (CW) from the Delis-Kaplan Executive Function System (D-KEFS) was used to assess response inhibition (Delis, Kaplan, & Kramer, 2001). In this test, participants were required to name the colours of printed ink squares (C1) and read printed colour words (C2) as fast as possible. In the inhibition condition (C3), participants were instructed to name the ink colour of words printed in incongruent-coloured ink, suppressing the automatic response to read the word itself. The outcome measure was latency (in seconds) of the baseline subtracted from the inhibition condition (i.e. C3 latency – C1 latency). This method was proposed by Delis et al. (2001) to generate a pure measure of response inhibition. The D-KEFS CW scores were reverse-coded so that higher scores reflect better performance. The CW subtest has demonstrated adequate internal consistency reliability (α = .75; Delis et al., 2001), but reliability among contrast scores may be reduced among older adults (Crawford, Sutherland, & Garthwaite, 2008).

The Letter-Number Sequencing subtest from the Wechsler Adult Intelligence Scale – 3rd Edition (WAIS-III) was used as a measure of working memory (Wechsler, 1997). Participants were required to recall a sequence of numbers and letters by stating the numbers in ascending order followed by the letters in alphabetical order, forming an overall outcome measure of correctly recalled sequences. Among older adults, this subtest demonstrates adequate test-retest reliability (intraclass correlation = .73; Lemay, Bédard, Rouleau, & Tremblay, 2004).

Finally, the Backward Digit Span subtest from the WAIS-III was used as a measure of auditory attention (Wechsler, 1997). Participants were required to recall sequences of numbers in the reverse order in which they heard them, again forming an overall outcome measure of correctly recalled sequences. The Digit Span subtest demonstrates high internal consistency reliability across clinical populations (α = .92; Zhu, Tulsky, Price, & Chen, 2001).

Semantic Memory. The Kaufman Brief Intelligence Test – 2nd Edition (KBIT-2) Verbal Knowledge subtest (Kaufman & Kaufman, 2004) was used to assess semantic memory. In this task, participants selected the correct picture out of six choices that
describe a target word. The outcome measure for the current study was the number of correct target words as an indicator of semantic memory, with theoretical scores ranging from 0 to 60. Mean reliability estimates are high (α = .91) for internal consistency, and test-retest reliability ranges from α = .88 to α = .93, with reliabilities increasing with age. The KBIT-2 has also been demonstrated to have adequate convergent and discriminant validity (Kaufman & Kaufman, 2004).

**Episodic Memory.** The California Verbal Learning Test-II (CVLT-II) was used to assess verbal episodic memory (Delis, Kramer, Kaplan, & Ober, 2000). Participants were required to immediately recall 16 words over five learning trials, an interference trial, and a 20-minute delay trial. The outcome measure was composed of raw scores from the total number of items learned over the trials 1-5 (i.e. learning), short delay free recall (i.e. short-term verbal retention), and long delay free recall (i.e. long-term verbal retention). The CVLT-II has high split-half reliability among older adults over the age of 60 (α = .91 to .92; Delis et al., 2000) and adequate internal consistency reliability in healthy adults (α = .80 to .84; Woods, Delis, Scott, Kramer, & Holdnack, 2006).

**Theory of Mind Measures**

**Reading the Mind in the Eyes Test.** The Reading the Mind in the Eyes Test (RMET; Baron-Cohen, Wheelright, Hill, Raste, & Plumb, 2001) is widely used to examine ToM abilities in individuals across the lifespan (Henry et al., 2013). In this measure, participants were presented with 36 black-and-white photographs of human eyes and were asked to choose the best descriptor out of four choices for the emotional mental state represented in each face. Correct choices were summed to form the outcome measure, with theoretical scores ranging from 0-36 and higher scores reflecting higher aptitude in mentalizing, or putting themselves into the mind of another. The RMET has been used in over 250 studies to test emotion recognition across the lifespan (Kirkland et al., 2013), but reliability remains inadequate among studies that have reported internal consistency. Recent reliability estimates range from α = .61 (Vellante et al., 2013) to α = .64 (Söderstrand & Almkvist, 2012). However, it has been found to have acceptable test-retest reliability (r = .63, p < .01; Fernandez-Abascal, Cabello, Fernandez-Berrocal, & Baron-Cohen, 2013) and adequate validity (Vellante et al., 2013). As per guidelines by
Baron-Cohen and colleagues (2001) as well as previous reliability analyses in our lab (Fischer et al., 2016), we removed two items with poor response discrimination and eight items with low item-total agreement. The resulting internal consistency was poor (ICC = .48) but consistent with previous estimates. Please see Appendix C, Figure 1 for examples of the RMET stimuli used.

**Strange Stories Test.** The Strange Stories test (STORIES) was originally developed by Happé, Winner and Brownell (1998) for use with individuals with Autism Spectrum Disorder. In contemporary research, STORIES is one of the most widely used tests in ToM literature (Henry et al., 2013). In this test, participants were required to reason about the meta-cognitive mental states of different characters represented in stories accompanied by black-and-white drawings. After reading the presented story and related drawing, participants were asked “Why did [the character] say/do that?” Responses were given a score of 2 for a complete and accurate response, 1 for a partial or implied response, and 0 for incorrect or irrelevant response. The total summed score formed the outcome measure of cognitive ToM ability. Few studies to date have reported on the psychometric properties of the Strange Stories test, but preliminary evidence from Fischer et al. (2016) demonstrates excellent reliability (ICC = .95). We provide examples of the stories used and their respective response criteria in Appendix C, Table 1.

**Yoni Test.** The Yoni Test (Shamay-Tsoory & Aharon-Peretz, 2007) was originally developed for use in neuroimaging research and has been employed on a limited basis in aging populations (Narme et al., 2013; Fischer et al., 2016). This test is a measure of both cognitive and affective ToM, thus making it an attractive option for the assessment of mentalizing in clinical populations. Participants were required to use mental state information contained in available cues such as eye gaze and facial expression to indicate which picture the cartoon character Yoni is referring to. Each item was scored as either correct (1 point) or incorrect (0 points) and the responses were summed to form the outcome measures of C-ToM and A-ToM. Similar to other ToM measures, there is limited information available regarding the psychometric properties of the Yoni Test but current estimates (e.g. Fischer et al., 2016) suggest acceptable internal consistency reliability scores (ICC = .86 and .74 for YONI Cognitive and Affective trials, respectively). An example of the Yoni Test protocol is presented in Appendix C, Figure 2.
Strong correlations between the ToM measures have previously allowed for the use of composite A-ToM and C-ToM variables with good psychometric properties (e.g., Fischer et al., 2016). Guided by previous research, we used the RMET and YONI-A to assess A-ToM and used STORIES and YONI-C to assess C-ToM. Scores on each measure were converted into z-scores and summed to create the respective A-ToM and C-ToM composite variables. See Table 2 (Appendix A) for intercorrelations among the ToM measures and composite variables and Table 3 (Appendix A) for intercorrelations among the neurocognitive measures.
STATISTICAL ANALYSIS

Data Preparation

Before conducting analyses, predictor variables (i.e., illness burden, neurocognitive functioning, and demographics) and the outcome variables (i.e., C-ToM and A-ToM) were checked for missing values, accuracy of data entry, and distributions among the sample. Rates of missing data were negligible; in total we were missing data for three out of approximately 1800 cells (or ~ .1%). We accounted for missing values by using pairwise deletion to retain power by excluding cases that were missing data only for that specific analysis (Tabachnick & Fidell, 2013). Descriptive statistics for each continuous variable were examined to determine the central tendency of the data (mean, median, mode), variability (range, standard deviation), and distribution shape (kurtosis and skew). The Shapiro-Wilkes test determined that the standardized estimates of kurtosis and skew were within acceptable ranges. Frequencies and percentages were reported for the categorical variables. See Table 4 (Appendix A) for descriptive information of participant performance on the neurocognitive and ToM measures. We also examined histograms and Q-Q plots for the predictors to ensure that there were no violations of assumptions, including independence of observations and bivariate normality. We tested the parametric assumptions of multiple linear regression and no multivariate outliers (i.e., determined by extreme values of Mahalanobis distance) or influential points (i.e., using Cook’s distance < 1.00) were identified (see Tabachnik & Fidell, 2013). The regression models were not adversely affected by homoscedascity (i.e., using $F_{\text{max}}$ estimates) or multicollinearity between predictor variables (i.e., Low Condition Indices < 30). In sum, all parametric assumptions of multiple linear regression were met for all models. SPSS 24.0 was used for all analyses (IBM Corp, 2016).

Preliminary Analyses

As illustrated in Table 1, we examined descriptive statistics and corresponding correlation coefficients between demographic, health, and neurocognitive performance variables to identify variables important for inclusion as covariates in the regression models (i.e., if they were correlated with C-ToM or A-ToM at $r \geq .3$; Cohen, 1998).
Table 1  Correlation Matrices for Theory of Mind, Neurocognitive Variables, and Illness Burden

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Education</th>
<th>Sex&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Semantic Memory</th>
<th>Episodic Memory</th>
<th>Executive Functioning</th>
<th>Vascular Illness</th>
<th>Nonvascular Illness</th>
<th>Cognitive ToM</th>
<th>Affective ToM</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>-</td>
<td>- .15</td>
<td>.02</td>
<td>-.13</td>
<td>-.23</td>
<td>-.31**</td>
<td>-.11</td>
<td>-.01</td>
<td>-.33*</td>
<td>-.30**</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td>-</td>
<td>-</td>
<td>-.13</td>
<td>.25*</td>
<td>.41</td>
<td>.33</td>
<td>-.08</td>
<td>-.04</td>
<td>.24*</td>
<td>.15</td>
</tr>
<tr>
<td><strong>Sex (M/F)</strong></td>
<td>-</td>
<td>-</td>
<td>.12</td>
<td>.54***</td>
<td>.48***</td>
<td>-.23*</td>
<td>-.12</td>
<td>-.23*</td>
<td>-.23*</td>
<td>.12</td>
</tr>
<tr>
<td><strong>Semantic Memory</strong></td>
<td>-</td>
<td>-</td>
<td>.33**</td>
<td>.32**</td>
<td>-.25*</td>
<td>-.21*</td>
<td>.24**</td>
<td></td>
<td>.51***</td>
<td></td>
</tr>
<tr>
<td><strong>Episodic Memory</strong></td>
<td>-</td>
<td>-</td>
<td>.49***</td>
<td>.29**</td>
<td>.02</td>
<td>.32*</td>
<td>.23*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Executive Functioning</strong></td>
<td>-</td>
<td>-</td>
<td>-.31**</td>
<td>.04</td>
<td>.36**</td>
<td>.32**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Vascular Illness</strong></td>
<td>-</td>
<td>-</td>
<td>.10</td>
<td>-.46***</td>
<td>-.27**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Nonvascular Illness</strong></td>
<td>-</td>
<td>-</td>
<td>-.02</td>
<td>.53***</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

* N = 86
* correlation is significant at the 0.05 level (2-tailed)
** correlation is significant at the .01 level (2-tailed)
***correlation is significant at the .001 level (2-tailed)

Note: Age is presented in years and sex is 0 = male, 1 = female. Vascular Illness, Nonvascular Illness, Cognitive ToM and Affective ToM reflect composite z-score variables. Semantic Memory = KBIT II VK; Episodic Memory = CVLT-II memory z-score composite; Executive Functioning = Executive Functions z-score composite. For all ToM and neurocognitive variables, higher scores represent better performance. For the illness burden variables, higher scores represent greater severity of illness.

<sup>a</sup> All reported associations are presented as Pearson correlation coefficients with the exception of associations with sex (M/F), which reflect Spearman’s rank correlation coefficients.
As can be seen in Table 1, ToM was significantly associated with neuropsychological performance on all three derived neurocognitive composites. Specifically, less accurate cognitive inferences about mental state content (C-TOm) were associated with lower semantic memory (KBIT-II; \( r = .24, p < .01 \)), episodic memory (Episodic Memory composite; \( r = .32, p < .05 \)), and executive functioning (Executive Functioning composite; \( r = .36, p < .01 \)). Poorer A-ToM was also associated with lower semantic memory (KBIT-II; \( r = .51, p < .000 \)), episodic memory (Episodic Memory composite; \( r = .23, p < .01 \)), and executive functioning (Executive Functioning composite; \( r = .32, p < .01 \)).

As presented in Table 1, greater severity of vascular illness burden was associated with lower accuracy in both cognitive and affective mental state reasoning (C-TOm; \( r = -.46, p < .000 \); A-ToM; \( r = -.27, p < .01 \)). Nonvascular illness burden was not significantly correlated with ToM in either cognitive nor affective domains. In terms of demographic information, increasing age was associated with worse C-ToM and A-ToM performance (C-ToM; \( r = -.23, p < .05 \); A-ToM; \( r = -.30, p < .05 \)) as well as poor executive functioning ability (Executive Functioning composite; \( r = -.31, p < .01 \)). Education was positively correlated with both semantic memory (KBIT-II; \( r = .25, p < .05 \)) and C-ToM (C-ToM; \( r = .24, p < .05 \)) but we opted a priori to exclude education as a predictor in the main regression models because it tends to be significantly associated with general intellectual functioning (Kaufman, Kaufman, Liu, & Johnson, 2010) and traditionally accounts for a large proportion of variance in neuropsychological function compared to age, gender and other demographics (Heaton et al., 2009). Female sex was positively associated with better cognitive mental state reasoning (C-ToM; \( r = -.23, p < .05 \)) and male sex was correlated with greater vascular illness burden (\( r = -.23, p < .05 \)). Age was retained as a predictor in the subsequent regression models given its influence on illness burden and ToM performance. In sum, we included the following variables as covariates: age, semantic memory, episodic memory, and executive functioning. Notably, C-ToM and A-ToM were significantly correlated with each other (\( r = .53, p < .000 \)).

Given the importance of reliable ToM test scores to the interpretation of findings paired with the lack of published reliability data, we examined the item-level properties of
ToM measures. For each test, items with low response variability and negative or low Item-Total Correlations (i.e. \( r < .10 \); Meyers, Gamst, & Guarino, 2013) were deleted. Ten items were dropped from the RMET (26 items retained) and one item was dropped from Strange Stories (11 items retained). The revised versions of these tests, as devised by Fischer et al. (2016) were used for the remaining analyses.

Finally, z-score composites were derived to create two dependent variables: C-ToM (Strange Stories and YONI-C) and A-ToM (RMET and YONI-A). As discussed above, we summed the aggregate beta weights derived from linear regression as per Patel et al., 2013 to create the vascular and nonvascular illness burden variables. Three neuropsychological composite variables were created by summing the z-scores of the candidate neurocognitive measures that displayed significant correlations with ToM and whose constructs carry meaningful theoretical rationale in cognitive aging (e.g., Salthouse, 2009; Fischer et al., 2014). Principle Components Analysis (assessing the smallest number of components from the rotated matrix that best fit the data) revealed that one composite neurocognitive variable consisted of the executive functioning neurocognitive measures (namely, D-KEFS CW Interference, WAIS-III Letter Number Sequencing, WAIS-III Digit Span) and another neurocognitive variable consisted of the KBIT-2 to represent semantic memory. Episodic memory was composed of learning, short delay, and long delay scores from the CVLT-II. Tables 2 and 3 (Appendix A) present the intercorrelations among the individual ToM and neurocognitive variables as well as their correlations with the derived composites.

Regression Analyses

To determine the utility of illness burden (i.e., vascular and nonvascular subsets) in predicting ToM performance, a series of hierarchical analyses were conducted with composite C-ToM and A-ToM as the outcome variables while statistically controlling for demographic variables that were moderately correlated with ToM at an effect size of > .3 (Cohen, 1998). Block one of the regression model was entered as age and other demographic predictors, and neurocognitive composites were added in block two to determine the amount of variance associated with each predictor while controlling for one another. The candidate illness burden predictors (i.e., vascular and nonvascular subsets)
were entered individually into block three in separate models. All regression analyses were conducted using F-tests and their corresponding ΔR² values to determine whether each step added predictive utility to the model. Standardized regression coefficients were examined to assess the strength and direction of any significant predictors.

**Mediation Analyses**

To address the potential mediating effects of vascular and nonvascular illness burden, OLS regression-based path analysis was completed according to the guidelines developed by Preacher and Hayes (2015). We chose potential mediators based on theoretical and statistical rationale to determine how illness burden is associated with ToM. Unlike more traditional approaches that require criteria to establish mediation (e.g., an a priori association between X and Y is essential to conduct mediation), the mediation conditions established by Preacher and Hayes (2015) allow for an explicit estimate of the indirect effect without mandatory significance between variables. In the absence of rigorous assumptions in the Preacher and Hayes (2015) approach, bootstrapping was employed to conduct inferential tests of the direct and indirect effects. Bias-corrected bootstrap confidence intervals respect the irregularity of the sampling distribution of the indirect effect and are more powerful as statistical controls than previous methods in traditional mediation approaches (Preacher & Hayes, 2015). We began with a hypothesized multivariate model in which proposed mediators included executive functioning, semantic memory and episodic memory to predict both C-ToM and A-ToM. We examined whether changes in vascular or nonvascular illness burden accounted for changes in the proposed mediators and whether changes in the proposed mediators accounted for changes in the levels of the outcome variables (i.e., C-ToM and A-ToM in separate models). We also investigated whether the relationship between illness burden and ToM was maintained when the effects of the proposed mediators were controlled for.

**Error Control and Power**

In order to attain acceptable statistical power to test the research questions and control for type I and II error, all analyses were run at an alpha level of .05. Given a sample
size of N = 86 using multiple regression with one predictor variable in block one (demographic covariate: age), three variables in block two (neurocognitive functioning composites), and one predictor variable in block three (illness burden subset), a priori power calculations suggest that a medium effect size ($f^2 = 0.26$) can be detected using Cohen’s guidelines (Cohen, 1998). Similarly, the regression model for the mediation analyses is sufficient to detect a medium effect size. Power analyses were conducted using the G*Power calculator version 3.1.2 (Faul, Erdfelder, Buchner, & Lang, 2015).
RESULTS

Demographic Characteristics and Health Information

Table 2 presents means and standard deviations for participant characteristics including demographics (age, education, sex, ethnicity, birthplace, EAL status), global cognitive status (MMSE), and medication information. Overall level of education ($M = 14.28, SD = 2.18$) and global cognitive status (MMSE; $M = 29.16, SD = 1.13$) indicate that this sample was well-educated and demonstrated intact global cognitive performance. In addition, the sample was predominately female (68.6%).

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Demographic and Cognitive Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variable</td>
<td>Participants ($N = 86$)</td>
</tr>
<tr>
<td>Age</td>
<td>71.74 (5.42)</td>
</tr>
<tr>
<td>Range</td>
<td>64.60 – 87.86</td>
</tr>
<tr>
<td>Education</td>
<td>14.28 (2.18)</td>
</tr>
<tr>
<td>Range</td>
<td>9 – 20</td>
</tr>
<tr>
<td>Female (n; %)</td>
<td>59; 68.6</td>
</tr>
<tr>
<td>Ethnicity (n; %)</td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>82; 95.3</td>
</tr>
<tr>
<td>Asian</td>
<td>2; 2.3</td>
</tr>
<tr>
<td>Hispanic</td>
<td>1; 1.2</td>
</tr>
<tr>
<td>South Asian/Indian</td>
<td>1; 1.2</td>
</tr>
<tr>
<td>Birthplace$^a$ (n; % foreign born)</td>
<td>28; 32.6</td>
</tr>
<tr>
<td>EAL$^b$ (n; %)</td>
<td>10; 11.6</td>
</tr>
<tr>
<td>MMSE$^c$</td>
<td>29.16 (1.13)</td>
</tr>
</tbody>
</table>

Note: Means and standard deviations are presented as M (SD). Age and education are presented in years.

$^a$Birthplace (% foreign born) = reported birthplace outside of North America;

$^b$EAL = reported English as an additional language

$^c$MMSE = Mini-Mental Status Examination (range = 0 to 30)

Detailed health characteristics of participants are presented in Table 3 for vascular risks (hypertension, diabetes mellitus, high cholesterol, and high pulse pressure) and nonvascular risks (osteoporosis, osteoarthritis, rheumatoid arthritis, and thyroid dysfunction). Notably, the proportion of older adults in this sample with hypertension is somewhat consistent with prevalence in the general population (e.g., ~60%; Nguyen et
In addition, 55.6% of the sample reported a physician’s diagnosis of at least one vascular disease, and only 15% reported no physician’s diagnoses of any illness (vascular or nonvascular).

### Table 3  Health Characteristics

<table>
<thead>
<tr>
<th>Vascular Risks (n; % diagnosed)</th>
<th>N = 86</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>40; 46.5</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>14; 16.3</td>
</tr>
<tr>
<td>High cholesterol</td>
<td>30; 34.9</td>
</tr>
<tr>
<td>High pulse pressure</td>
<td>38; 44.2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Nonvascular Risks (n; % diagnosed)</th>
<th>N = 86</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteoporosis</td>
<td>20; 23.3</td>
</tr>
<tr>
<td>Osteoarthritis</td>
<td>29; 33.7</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>4; 4.7</td>
</tr>
<tr>
<td>Thyroid dysfunction</td>
<td>19; 22.1</td>
</tr>
</tbody>
</table>

Note: Includes all individuals who self-reported a physician’s diagnosis of condition at time of testing and were currently being treated for condition at time of testing

**Research Question 1: Vascular and Nonvascular Illness Burden as Independent Predictors of Cognitive ToM**

As expected, increasing age was associated with worse C-ToM ($\beta = -.232$) and was retained in all regression models. Biological sex did not predict C-ToM performance and was dropped from subsequent analyses. Better semantic memory ($\beta = .223$) and executive functioning ($\beta = .298$) abilities were associated with higher cognitive mental state reasoning ($R^2 = .266, F_{(3,78)} = 6.71, p < .001$) and the neurocognitive variables accounted for 26.6% of the total variance in C-ToM. As can be seen in Table 4, vascular illness burden was associated with poorer C-ToM performance and accounted for 43.1% unique variance in the outcome ($R^2 = .431, F_{(1,77)} = 11.10, p < .001$) beyond age and neurocognition. Contrary to our predictions, nonvascular illness burden severity was not significantly associated with poorer C-ToM performance ($R^2 = .266, F_{(1,77)} = 5.66, p = .95$).
### Table 4  Hierarchical Regressions of Vascular and Nonvascular Illness Burden as Predictors of Cognitive Theory of Mind

<table>
<thead>
<tr>
<th>Predictor</th>
<th>$B$</th>
<th>SE</th>
<th>$\beta$</th>
<th>$t$</th>
<th>$p$</th>
<th>$R^2$</th>
<th>$F$</th>
<th>$\Delta R^2$</th>
<th>$\Delta F$</th>
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<tbody>
<tr>
<td><strong>Block 1</strong></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.066</td>
<td>0.032</td>
<td>-0.232</td>
<td>-2.089</td>
<td>0.040</td>
<td>0.054</td>
<td>4.363*</td>
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</tr>
<tr>
<td><strong>Block 2</strong></td>
<td></td>
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<td></td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Semantic Memory</td>
<td>0.126</td>
<td>0.060</td>
<td>0.223</td>
<td>2.110</td>
<td>0.038</td>
<td>0.266</td>
<td>6.709***</td>
<td>0.213</td>
<td>7.144***</td>
</tr>
<tr>
<td>Episodic Memory</td>
<td>-0.047</td>
<td>0.065</td>
<td>-0.087</td>
<td>-0.735</td>
<td>0.465</td>
<td></td>
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</tr>
<tr>
<td>Exec. Func.</td>
<td>0.298</td>
<td>0.082</td>
<td>0.424</td>
<td>3.620</td>
<td>0.001</td>
<td></td>
<td></td>
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</tr>
<tr>
<td><strong>Block 3</strong></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Model 1: Vascular Burden</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.431</td>
<td>11.058***</td>
<td>0.131</td>
<td>21.144***</td>
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<tr>
<td>VIB</td>
<td>-0.552</td>
<td>0.120</td>
<td>-0.429</td>
<td>-4.598</td>
<td>0.000</td>
<td></td>
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<tr>
<td>Model 2: Nonvascular Burden</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.266</td>
<td>5.659</td>
<td>0.000</td>
<td>0.003</td>
</tr>
<tr>
<td>NVIB</td>
<td>-0.033</td>
<td>0.561</td>
<td>-0.006</td>
<td>-0.058</td>
<td>0.954</td>
<td></td>
<td></td>
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</tbody>
</table>

*N = 86*

*significant at the 0.05 level (2-tailed)*

**significant at the .01 level (2-tailed)**

***significant at the .001 level (2-tailed)**

Note: VIB = Vascular Illness Burden based on summed beta weights of illness severity; NVIB = Nonvascular Illness Burden based on summed beta weights of illness severity.
Research Question 2: Vascular and Nonvascular Illness Burden as Independent Predictors of Affective ToM

As presented in Table 5, we also investigated the independent effects of the predictors on A-ToM in older adults. Age was significantly associated to A-ToM ($\beta = -0.300$) such that increasing age predicted poorer A-ToM ability. Similar to previous analyses, biological sex did not predict A-ToM and was dropped from subsequent analyses. As predicted, better semantic memory ($\beta = 0.421$) and executive functioning ($\beta = 0.231$) abilities were associated with higher affective mental state reasoning ($R^2 = 0.367$, $F_{(3,78)} = 10.71$, $p < 0.001$) and the neurocognitive variables accounted for 36.7% of the total variance in A-ToM. Contrary to our predictions, neither vascular illness burden nor nonvascular illness burden contributed significant unique variance in A-ToM beyond age and neurocognitive performance, as depicted in Table 5.
Table 5  
Hierarchical Regressions of Vascular and Nonvascular Illness Burden as Predictors of Affective Theory of Mind

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>β</th>
<th>t</th>
<th>p</th>
<th>R²</th>
<th>F</th>
<th>ΔR²</th>
<th>ΔF</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Block 1</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-.086</td>
<td>.031</td>
<td>-.300</td>
<td>-2.758</td>
<td>.007</td>
<td>.090</td>
<td>7.601**</td>
<td>--</td>
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</tr>
<tr>
<td><strong>Block 2</strong></td>
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<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Semantic Memory</td>
<td>.239</td>
<td>.056</td>
<td>.421</td>
<td>4.287</td>
<td>.000</td>
<td>.367</td>
<td>10.721***</td>
<td>.277</td>
<td>10.792***</td>
</tr>
<tr>
<td>Episodic Memory</td>
<td>.020</td>
<td>.060</td>
<td>.036</td>
<td>.325</td>
<td>.746</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Exec. Func.</td>
<td>.163</td>
<td>.077</td>
<td>.231</td>
<td>2.125</td>
<td>.037</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Block 3</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1: Vascular Burden</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.617</td>
<td>8.956</td>
<td>.013</td>
<td>1.569</td>
</tr>
<tr>
<td>VIB</td>
<td>-.158</td>
<td>.126</td>
<td>-.122</td>
<td>-1.253</td>
<td>.214</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 2: Nonvascular Burden</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.371</td>
<td>9.189</td>
<td>.004</td>
<td>.472</td>
</tr>
<tr>
<td>NVIB</td>
<td>-.358</td>
<td>.522</td>
<td>-.063</td>
<td>-.687</td>
<td>.494</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*N* = 86
*significant at the 0.05 level (2-tailed)*
**significant at the .01 level (2-tailed)*
***significant at the .001 level (2-tailed)*

Note: VIB = Vascular Illness Burden based on summed beta weights of illness severity; NVIB = Nonvascular Illness Burden based on summed beta weights of illness severity.
Research Question 3: Neurocognition as a Mediator of Illness Burden and ToM

In order to address if neurocognitive functioning mediated associations between vascular or nonvascular illness burden and ToM, we conducted a series of OLS-regression based path analyses with either C-ToM or A-ToM as the outcome variable depending on the model. We opted a priori to exclude episodic memory as a potential mediator given its lack of association with either ToM domain. Although nonvascular illness burden did not predict unique variance in either C-ToM or A-ToM in our initial regression models, we retained it as a predictor for mediation analyses as per Preacher and Hayes (2016) guidelines to examine specific information about the magnitude and direction of its effects. In the final models, we evaluated if semantic memory and executive functioning fully or partially accounted for relationships between C-ToM or A-ToM and illness burden in separate analyses. Age was entered as a covariate to statistically control for main effects in the model given that it demonstrates a strong negative correlation with both C-ToM and A-ToM.
Figure 1. Standardized regression coefficients for the relationship between vascular illness burden ($X_1$) and cognitive ToM as mediated by executive functioning and semantic memory. The standardized regression coefficient between the total effect of illness burden and cognitive ToM, controlling for executive functioning and semantic memory respectively, is in parenthesis.

Note: *$p < .05$, **$p < .01$, ***$p < .001$

In a mediation analysis using ordinary least squares path analysis, vascular illness burden ($X_1$) indirectly influenced C-ToM through its effect on neurocognition. As can be seen in Figure 1, participants who had a greater vascular illness burden performed worse on executive functioning tasks ($a_1 = -0.31$) and semantic memory tasks ($a_2 = -0.47$). Further, participants who performed well on executive functioning tasks tended to have stronger C-ToM abilities ($b_1 = .25$), but semantic memory did not demonstrate a significant effect. A bias-corrected bootstrap confidence interval for the indirect effect of executive functioning ($ab_1 = -0.10$) based on 10 000 bootstrap samples was entirely below zero [-.223, -.015]. Executive functioning as a mediator accounted for roughly 18% of the total effect, $P_M = .18$. There was also evidence that vascular illness burden influenced C-ToM independent of its effect on executive functioning ($c_1' = -0.56$, $p = .000$). The indirect effect of semantic memory ($ab_2 = -0.05$) was tested against bias-corrected bootstrap confidence intervals based on 10,000 bootstrap samples and contained zero [-.210, .014], and therefore was not a significant mediator. Age did not have a significant effect on C-ToM in the mediation model relative to other predictors ($cov = -.05$, 95% CI [-.187, .086]. Further, semantic memory accounted for roughly 3% of the total effect, $P_M = .03$.

The relationship between nonvascular illness burden and C-ToM was not mediated by executive functioning or semantic knowledge. We tested the significance of the indirect effects on executive functioning and semantic memory ($ab_1 = -.22$, $ab_2 = 0.029$) using bootstrapping procedures. Unstandardized indirect effects were computed for each of 10,000 bootstrapped samples, and 95% confidence intervals were computed through bias-correction. The 95% confidence intervals for both executive functioning [-0.188, 0.005] and semantic memory [-0.332, 0.047] contained zero. Thus, the indirect effects were not statistically significant and neurocognition does not appear to mediate the relationship between nonvascular illness burden and C-ToM. In addition, nonvascular illness burden did not appear to have an independent effect on C-ToM performance ($c_2' = -0.01$, $p = .988$).
Affective ToM as the outcome variable in the mediation models to determine its relationship to vascular and nonvascular illness burden with mediating neurocognitive variables. In model 2 with A-ToM as the outcome variable, vascular illness burden (X1) did not indirectly influence A-ToM through its effect on executive functioning and semantic memory. As can be seen in Figure 2, participants who had a greater vascular illness burden performed significantly worse on tasks of executive functioning (a1 = -0.31) and semantic memory (a2 = -0.47). Participants who performed well on these tasks tended to have stronger A-ToM abilities (executive functioning; b1 = .22, semantic memory; b2 = .28). The indirect effects were tested against bias-corrected bootstrap confidence intervals based on 10,000 bootstrap samples and were found to contain zero for both mediators (executive functioning; [-.189, .005], semantic memory; [-.332, .047]), indicating no mediating effect. Further, the mediators accounted for roughly 8% of the total effect, $P_M = .08$. Vascular illness burden did not appear to independently influence A-ToM independent of its effect on executive functioning ($c'_1 = -0.26, p = .06, 95\% CI [-.521, .010]$) or semantic memory ($c'_2 = -0.18, p = .14, 95\% CI [-.423, .060]$) either.
The association between a participant’s nonvascular illness burden ($X_2$) and their A-ToM ability was not mediated by neurocognitive performance. Using bootstrapping procedures with 10,000 samples, the unstandardized coefficient of the indirect effect ($ab_2 = 0.023$) was tested with a 95% confidence interval and found to be insignificant [-0.478, 0.530]. In addition, nonvascular illness burden did not appear to have an independent effect on A-ToM performance ($c'_2 = -0.82, p = .153$).
DISCUSSION

To our knowledge, this is the first study to examine the relative contributions of vascular and nonvascular chronic illnesses to ToM abilities in older adults. Pulse pressure has been previously identified as a significant health predictor of C-ToM (Fischer et al., 2016); however, it has been the only health condition investigated prior to the present study and it remained unclear if the effects were specific to pulse pressure or reflected other illness-related factors. Our results support and extend previous work in this area in several ways. We found that greater severity of vascular illness burden predicted lower C-ToM in older adults beyond the effects of age and neurocognition. Contrary to our predictions, neither vascular illness burden nor nonvascular illness burden contributed significant unique variance in A-ToM beyond age and neurocognitive performance. While nonvascular illness burden did not emerge as a significant risk factor for declines in ToM, this finding is valuable because it suggests that the underpinnings of ToM mechanisms may be uniquely related to vascular health rather than a nonspecific effect related to poor overall health (e.g., pain, inflammation, lack of mobility, reduced social engagement). Thus, disease processes of vascular pathology may have a unique impact upon mentalizing abilities and our findings suggest that older adults with poorer overall vascular health may be particularly vulnerable to reductions in C-ToM.

The composition of the vascular illness burden variable is critically relevant to the findings discussed above. As can be seen in Table 1 of Appendix A, diabetes emerged as the strongest predictor of global ToM performance in this sample and its associated beta weight heavily influenced the composition of the overall variable. Although diabetes appeared to be a substantial predictor of global ToM, the small cell size (n = 14) hindered our ability to detect any significant results in regression and mediation analyses. The addition of other vascular conditions (hypertension, high cholesterol and pulse pressure) increased the power and stabilized the vascular illness burden variable, allowing us to draw meaningful comparisons in the data. Nevertheless, it is important to recognize the potential contribution of diabetes on ToM in these analyses, especially in light of its robust and well-established associations to cognitive functioning in older adults (e.g., Geijselaers et al., 2015; Mayeda et al., 2015; Yeung et al., 2009). Assuming that ToM abilities are involved in a diverse cortical network, it is possible that the specific neuroanatomical
mechanisms of diabetes may uniquely affect ToM. For example, diabetes is thought to affect frontal structures responsible for verbal fluency and executive functioning (Christman, Vannorsdall, Pearson, Hill-Briggs, & Schretlen, 2010; Wahlin et al., 2002) and is related to reduced volumes of the hippocampus and amygdala (Stranahan et al., 2008; den Heijer et al., 2003) and reduced cerebral blood flow (Novak et al., 2006). Diabetes has also been linked to accelerated rates of age-related structural changes as evidenced by subcortical atrophies and white matter lesions (Verdelho et al., 2010; Manschot et al., 2006). The extent to which diabetes (and its unique underlying mechanisms) independently predicts ToM in later life remains unknown. Further, the effects of concurrent major illnesses that can confound or exacerbate the effects of diabetes have yet to be explored within the context of ToM abilities among older adults. Nonetheless, the current findings suggest that diabetes, as well as pulse pressure and other vascular illnesses may be important predictors of worse ToM performance in later life. Future studies including large samples with diverse illness profiles are needed to determine the distinct and synergistic effects of vascular health risks on ToM and other important cognitive functions in later life.

The mediational results suggest that the relationship between vascular illness burden and C-ToM is mediated by neurocognition (namely, semantic memory and executive functions). Executive functions in particular appeared to account for a large proportion of the mediating effect, consistent with literature implicating executive functioning as a predictor of C-ToM (Fischer et al., 2016). Vascular illness burden also appeared to have a direct effect on C-ToM independent of its mediated path. In terms of affective ToM, vascular illness burden did not indirectly influence A-ToM through its effect on semantic memory or executive functioning. Contrary to our hypotheses, vascular illness burden did not appear to independently influence A-ToM independent of its effect on neurocognition, nor did nonvascular illness burden. These findings highlight the important mediating role of neurocognitive abilities in ToM expression and suggest that the unique relationship between vascular health and C-ToM is influenced to some extent by the neurocognitive processes that underlie them.

Consistent with previous literature, the findings in the present study support the notion that ToM abilities are underpinned by neurocognitive processes in older adults.
Past research using this sample (i.e., Fischer et al., 2016) found that executive functions predicted C-ToM in younger adults, while semantic memory and episodic memory predicted C-ToM in older adults. In terms of A-ToM, semantic memory was a significant predictor for both younger and older adults and executive functioning was significant only in older adults. Our pattern of results is somewhat consistent with this research, suggesting that executive functions and semantic memory play a key role in mentalizing in later life; however, we did not identify episodic memory as a significant predictor of either C-ToM or A-ToM in this sample. It is likely that the inclusion of a thorough inventory of health conditions in our models differentially impacted the associations between neurocognition and ToM, and controlling for the influences of multiple health predictors may have attenuated variance in ToM previously attributed to neurocognition.

In addition, we found moderate associations between executive functions and C-ToM ($r = -.36; 95\% \text{ CI} [-.30$ to $-.41]$), and strong associations between semantic memory and A-ToM ($r = -.51; 95\% \text{ CI} [-.45$ to $-.57]$). These findings map onto previous work showing that crystallized knowledge (i.e., semantic memory) is a robust predictor of A-ToM (Peterson & Miller, 2012) and supports the proposed developmental trajectory of ToM abilities, with A-ToM skills (and crystallized knowledge) thought to develop later than C-ToM skills (Vetter et al., 2013). Further, A-ToM may be more strongly susceptible to development and refinement through social and cultural experiences that accumulate over time. Of note, our C-ToM tasks were verbally-based and thus individuals with better semantic memory may have had an advantage on these measures as well.

**Limitations**

These results should be considered within the context of several limitations. The subjective self-report nature of the health measure (with the exception of pulse pressure) may not have accurately captured all individuals with an objective physical illness. A longitudinal research study on community-dwelling older adults revealed that objective, quantitative measures of health were better predictors of mortality than clinical history (yes/no responses) of disease (Fried et al., 1998). Such objective, direct measures quantitatively assess the current presence and severity of subclinical disease rather than
past health events in clinical history, and are not subject to false negatives or false positives. Further, the self-report diagnosis utilized in this study represented individuals receiving treatment for their condition, but not severity and duration of symptoms associated with a particular condition; such complex facets of self-rated health may be a more accurate predictor of overall cognitive functioning (Jylhä, 2009) and thus ToM ability. The use of objective health measures in future studies would allow us to draw robust inferences regarding the relationship between health status and ToM abilities and could offer additional insight into the potential role of nonspecific health factors that were not associated with ToM in the present study.

The sample in this study was highly educated ($M_{YEARS} = 14.28$) but we opted a priori to exclude education as a predictor in the main regression models because it tends to be significantly associated with general intellectual functioning (Kaufman, Kaufman, Liu, & Johnson, 2010) and traditionally accounts for a large proportion of variance in neuropsychological function compared to age, gender and other demographics (Heaton et al., 2009). This decision may have obscured potential differential associations between education and ToM performance, and future studies may consider the effects of high vs. low education in the expression of C-ToM and A-ToM. Further, our sample demonstrated intact global cognitive performance on a screening measure (MMSE; $M = 29.16$), but research suggests that subjective cognitive decline may be a better predictor of non-normative cognitive decline in the absence of objective cognitive dysfunction (Rabin, Smart, Crane, et al., 2015). Subjective cognitive decline is also linked to common chronic illnesses in older adults (Caracciolo et al., 2013) as well as anxiety and depression. Inclusion of a subjective cognitive decline measure (e.g., see Bondi et al., 2014) in future studies may account for the differential associations identified between chronic illness, neurocognition and ToM in this sample. Although we were able to detect medium effects, the sample size for this study was relatively small for multivariate analyses, increasing the chances of making a type II error - thereby reducing our ability to detect significant effects and limiting interpretability of the results. Additionally, the cross-sectional nature of this study limited our ability to examine the long-term relationships between ToM, neurocognitive functioning, and illness burden over time and prevented us from drawing causal inferences between these variables among the aging population. However, a major strength of this study was using a novel measure of C-ToM and A-ToM (Yoni) that we
combined with conventional measures (Strange Stories, RMET) into composite variables. The Yoni test has demonstrated good psychometric properties (Fischer et al., 2016) and stabilized the reliability of the existing cognitive and affective measures, allowing us to measure ToM more broadly and robustly across its differential domains.

**Conclusion & Future Directions**

This study underscores the utility of analyzing chronic illness burden in future research on age-related changes in mental state reasoning. Neurocognitive functioning examined in isolation may be insufficient to account for the nature of changes in ToM ability as we age. Findings addressing the influence of illness burden have key implications for understanding ToM as a multifaceted construct that remains conceptually stable across the adult lifespan due to shared underlying neurocognitive and health mechanisms. Parsing out the unique roles of diabetes and pulse pressure relative to other vascular comorbidities is a top priority in understanding the maintenance and decline of ToM in later life. Future studies may also consider the investigation of other possible health modifiers including self-rated health and wellbeing, medical records, or other indicators of vascular risk such as exercise level. Additionally, longitudinal studies may inform about long-term implications of the role of health in ToM and may determine if cognitive and health factors that influence ToM have cumulative effects over time.

Recent developments in adulthood and aging research suggest that a deeper examination of how and why ToM is impacted in aging is of critical importance. The findings presented in this study have important implications for public policy and the healthcare system, as the aging baby boomer generation is anticipated to account for the greatest utilization of health services in the coming years (Canadian Institute for Health Information, 2011). ToM difficulties also have important consequences for social functioning in clinical groups such as those with autism and schizophrenia (Brown, Tas, Can, Esen-Danaci, & Brüne, 2014), as well as age-related neurodegenerative pathologies such as Alzheimer’s disease and Parkinson’s disease (Sandoz et al., 2014). Among frail older adults, reduced social cognition has been linked to linked to lack of social engagement and declines in independence (Fett et al., 2011). Preliminary evidence indicates that even cognitively healthy older adults experience ToM-mediated reductions
in social activity (Bailey et al., 2008), supporting the notion that ToM acts as an influential predictor of behavior in everyday functioning.

The phenomenon of successful aging, defined as the maintenance of physical and cognitive health, has become increasingly popular as older adults enjoy better access to resources that maintain social engagement and improve overall quality of life. This study provides an important foundation for future investigations clarifying the role of chronic illness among the well-established relationship between neurocognitive functioning and ToM, and may provide insight into key changes in older adulthood that have real-world implications at both the personal and policy levels.
REFERENCES


Appendix A.
Additional Tables

Table A1  Linear Regressions of Vascular and Nonvascular Risk Factors as Predictors of Global Theory of Mind Performance

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>β</th>
<th>t</th>
<th>p</th>
<th>R²</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1: Vascular</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dx of hypertension</td>
<td>-.531</td>
<td>.694</td>
<td>-.098</td>
<td>-.766</td>
<td>.446</td>
<td></td>
<td></td>
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<td>Dx of diabetes mellitus</td>
<td>-2.411</td>
<td>.835</td>
<td>-.330</td>
<td>-2.888***</td>
<td>.005</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dx of high cholesterol</td>
<td>-.448</td>
<td>.643</td>
<td>-.079</td>
<td>-.697</td>
<td>.488</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High pulse pressure</td>
<td>-.444</td>
<td>.601</td>
<td>-.082</td>
<td>-.739</td>
<td>.462</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 2: Nonvascular</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dx of osteoporosis</td>
<td>-.458</td>
<td>.718</td>
<td>-.072</td>
<td>-.638</td>
<td>.525</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dx of osteoarthritis</td>
<td>.263</td>
<td>.649</td>
<td>.046</td>
<td>.405</td>
<td>.687</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dx of rheumatoid arth.</td>
<td>.645</td>
<td>1.450</td>
<td>.050</td>
<td>.445</td>
<td>.658</td>
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<td></td>
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<tr>
<td>Hx of thyroid dysfunc.</td>
<td>.240</td>
<td>.748</td>
<td>.037</td>
<td>.321</td>
<td>.749</td>
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</table>

N = 86
*significant at the 0.05 level (2-tailed)
**significant at the .01 level (2-tailed)
***significant at the .001 level (2-tailed)

Note: Vascular and Nonvascular Risk factors based on individuals who self-reported a physician's diagnosis of condition at time of testing (0 = no illness, 1 = presence of illness). All predictors were entered individually against Global Theory of Mind Performance (constructed from the sum of Cognitive ToM and Affective ToM composite z-score variables).
Table A2  Correlation Matrix for Theory of Mind Variables

<table>
<thead>
<tr>
<th></th>
<th>Strange Stories</th>
<th>RMET</th>
<th>YONI C-TOM</th>
<th>YONI A-TOM</th>
<th>Cognitive TOM</th>
<th>Affective TOM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strange Stories</td>
<td>-</td>
<td></td>
<td>.20*</td>
<td>.24*</td>
<td>.77***</td>
<td>.24*</td>
</tr>
<tr>
<td>RMET</td>
<td>-</td>
<td>.10</td>
<td></td>
<td>.22*</td>
<td>.15</td>
<td>.78***</td>
</tr>
<tr>
<td>YONI C-TOM</td>
<td>-</td>
<td></td>
<td>.81***</td>
<td></td>
<td>.77***</td>
<td>.58***</td>
</tr>
<tr>
<td>YONI A-TOM</td>
<td>-</td>
<td></td>
<td></td>
<td>.68***</td>
<td></td>
<td>.78***</td>
</tr>
<tr>
<td>Cognitive ToM</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.53***</td>
</tr>
<tr>
<td>Affective ToM</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*N* = 86  
* correlation is significant at the 0.05 level (2-tailed)  
** correlation is significant at the .01 level (2-tailed)  
***correlation is significant at the .001 level (2-tailed)  
Note: Higher scores indicate better performance for all variables. Cognitive ToM and Affective ToM represent the composite z-score variables.
Table A3  Correlation Matrix for Neurocognitive Variables

<table>
<thead>
<tr>
<th></th>
<th>KBIT VK</th>
<th>CVLT Imm.</th>
<th>CVLT SD</th>
<th>CVLT LD</th>
<th>Episodic Mem.</th>
<th>WAIS LN</th>
<th>WAIS DS</th>
<th>DKEFS CW</th>
<th>Exec. Functioning</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>KBIT VK</strong></td>
<td>-</td>
<td>.331**</td>
<td>.305**</td>
<td>.288**</td>
<td>.330**</td>
<td>.170</td>
<td>.191</td>
<td>.129</td>
<td>.220*</td>
</tr>
<tr>
<td><strong>CVLT Imm.</strong></td>
<td>-</td>
<td>-</td>
<td>.864**</td>
<td>.850**</td>
<td>.948**</td>
<td>.521**</td>
<td>.242*</td>
<td>.275*</td>
<td>.471**</td>
</tr>
<tr>
<td><strong>CVLT SD</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.878**</td>
<td>.958**</td>
<td>.470**</td>
<td>.290**</td>
<td>.313**</td>
<td>.487**</td>
</tr>
<tr>
<td><strong>CVLT LD</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.954**</td>
<td>.396**</td>
<td>.333**</td>
<td>.288**</td>
<td>.462**</td>
</tr>
<tr>
<td><strong>Episodic Mem.</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.478**</td>
<td>.306**</td>
<td>.299**</td>
<td>.491**</td>
<td></td>
</tr>
<tr>
<td><strong>WAIS LN</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.293***</td>
<td>.367***</td>
<td>.753***</td>
<td></td>
</tr>
<tr>
<td><strong>WAIS DS</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.269**</td>
<td>.708***</td>
<td></td>
</tr>
<tr>
<td><strong>DKEFS CW</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.742***</td>
<td></td>
</tr>
</tbody>
</table>

*Note: Higher scores indicate better performance for all variables. Episodic Mem. represents the composite z-score variable for the episodic memory measures and Executive Functioning represents the composite z-score variable for the executive functioning measures excluding KBIT VK.**
## Table A4  Performance on Neurocognitive and Theory of Mind Measures

<table>
<thead>
<tr>
<th>Theory of Mind Variables</th>
<th>Theoretical Range</th>
<th>M (SD)</th>
<th>Correlation with age (r)</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Strange Stories</em></td>
<td>0 - 14</td>
<td>10.29 (1.77)</td>
<td>-.165</td>
</tr>
<tr>
<td><em>RMET</em></td>
<td>0 - 26</td>
<td>18.35 (2.93)</td>
<td>-.137</td>
</tr>
<tr>
<td><em>YONI C-TOM</em></td>
<td>0 - 28</td>
<td>23.91 (3.30)</td>
<td>-.193*</td>
</tr>
<tr>
<td><em>YONI A-TOM</em></td>
<td>0 - 28</td>
<td>22.91 (4.95)</td>
<td>-.329**</td>
</tr>
</tbody>
</table>

### Neurocognitive Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Theoretical Range</th>
<th>M (SD)</th>
<th>Correlation with age (r)</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>KBIT VK</em></td>
<td>0 - 60</td>
<td>54.96 (2.75)</td>
<td>-.126</td>
</tr>
<tr>
<td><em>CVLT Imm.</em></td>
<td>N/A</td>
<td>45.12 (11.78)</td>
<td>-.261*</td>
</tr>
<tr>
<td><em>CVLT SD</em></td>
<td>N/A</td>
<td>9.06 (3.40)</td>
<td>-.182</td>
</tr>
<tr>
<td><em>CVLT LD</em></td>
<td>N/A</td>
<td>9.52 (3.65)</td>
<td>-.191</td>
</tr>
<tr>
<td><em>WAIS LN</em></td>
<td>0 - 21</td>
<td>9.66 (2.31)</td>
<td>-.239*</td>
</tr>
<tr>
<td><em>WAIS DS</em></td>
<td>0 - 15</td>
<td>6.78 (2.04)</td>
<td>-.248*</td>
</tr>
<tr>
<td><em>DKEFS CW</em>²</td>
<td>0 - 120</td>
<td>32.67 (12.31)</td>
<td>-.271**</td>
</tr>
</tbody>
</table>

*N = 86*

* correlation is significant at the 0.05 level (2-tailed)
** correlation is significant at the .01 level (2-tailed)

Note: KBIT VK = KBIT-2 Verbal Knowledge; CVLT Imm. = CVLT-II Immediate Recall; CVLT SD = CVLT-II Short Delay Free Recall; CVLT LD = CVLT-II Long Delay Recall; WAIS LN = WAIS-III Letter Number Sequencing; WAIS DS = WAIS-III Backwards Digit Span; DKEFS CW = DKEFS Color-Word Interference Contrast Score. All measures coded with higher scores indicating better performance with the exception of (a) noted below. Correlations with age are represented as Pearson’s *r* correlation coefficients for the association between each variable and age in years (from 64-88 years).

²On the DKEFS CW measure, higher scores in the Mean (SD) columns indicate slower performance. These scores were reverse coded to calculate the correlation with age and for all primary analyses to maintain a consistent metric with the other neurocognitive scores (i.e., where higher scores represent better performance).
Appendix B.
Differential Associations of ToM to Age, Biological Sex and Cognition

Few studies to date have attempted to parse out differential age effects on the separate cognitive and affective domains of ToM. There are strong indications of greater cognitive ToM difficulties among older adults compared to younger counterparts, and this finding is robustly supported (e.g., Fischer et al., 2016). In contrast, research examining affective ToM in older adults has produced inconsistent findings. Some studies propose that affective ToM is preserved in older adults (e.g., Castelli et al., 2010) while others report declines in this ability in later life (e.g., Mahy et al., 2014). Even in studies that analyzed age effects in both cognitive and affective ToM, it remains unclear as to whether age effects are limited to cognitive ToM (e.g., Bottiroli et al., 2016) or extend to affective ToM as well (e.g., Duval et al., 2011, Rakoczy et al., 2012).

In terms of biological sex effects, females typically acquire and develop ToM concepts earlier than males in early life (Kirkland, Peterson, Baker, Miller, & Pulos, 2013), but the effect of biological sex on ToM in later life is less clear. While neuroimaging studies have suggested that females employ additional brain regions that underlie emotion during ToM activities compared to same-age males (Christov-Moore et al., 2014), aging studies have reported mixed effects between sex and cognitive ToM (Franco & Smith, 2014). For example, Fischer et al. (2016) reported that male sex predicted better cognitive ToM in older adults, but there was no association between sex and affective ToM. Nevertheless, biological sex has a definitive influence on other neurocognitive skills (e.g., language) that are thought to underlie ToM mechanisms, so the potential role of this variable cannot be overlooked.

It is well established that fluid neurocognitive abilities including executive function, processing speed, and episodic memory tend to weaken with age, while crystallized verbal skills remain intact (Nisbett et al., 2012). ToM relies on neurocognitive resources throughout the life span, and neuropsychological perspectives suggest that A-ToM and C-ToM have dissociable components that are differentially associated with patterns in cognitive functioning. While reduced C-ToM has been linked to age-related declines in
executive skills (Wang & Su, 2013), abstract reasoning (Ahmed & Miller, 2013), attention and working memory (McKinnon & Moscovitch, 2007), and semantic and episodic memory (Fischer et al., 2016; Fischer et al., 2014), the association between A-ToM and neuropsychological performance is less clear (Duval et al., 2011; Wang & Su, 2013). Some evidence suggests that A-ToM is associated with aspects of executive functioning (i.e., inhibition; Wange & Su, 2013) and semantic memory (i.e., crystallized intelligence; Peterson & Miller, 2012) that persists even when language demands of the laboratory tasks are minimal. More recently our lab has found a significant association between A-ToM and executive functioning in older adults using a comprehensive composite variable comprised of inhibition, attention and working memory measures (Fischer et al., 2016). Semantic memory also emerged as a significant predictor of affective ToM in this study. Nevertheless, our understanding of concurrent contributions of neurocognitive factors on A-ToM and C-ToM is limited; the majority of current literature lacks an incorporation of multiple neurocognitive predictors of ToM or the inclusion of affective ToM measures. Further, it remains ambiguous as to whether ToM is indirectly influenced by neurocognition as a mediating variable.
Appendix C.
Sample Items from ToM Tests

Table C1  
Sample Items: Strange Stories Test (Cognitive ToM)

<table>
<thead>
<tr>
<th>Example 1</th>
<th>Sample Responses</th>
</tr>
</thead>
</table>
| Brian is always hungry. Today at school it is his favorite meal—sausages and beans. He is a very greedy boy, and he would like to have more sausages than anybody else, even though his mother will have made him a lovely meal when he gets home! But everyone is allowed two sausages and no more. When it is Brian's turn to be served, he says, "Oh, please can I have four sausages, because I won't be having any dinner when I get home!" | 2 points:  
He's lying to make them feel sorry for him  
He's greedy and wants to persuade them to give him more  

1 point:  
He likes sausages and wants more than anyone else  
He's a very greedy boy who loves sausages  

<table>
<thead>
<tr>
<th>Q: Why does Brian say this?</th>
<th>Sample Responses</th>
</tr>
</thead>
</table>
|                                                                                             | 2 points:  
Because his mom won’t be cooking for him tonight  
He wants to take the sausages home with him |

<table>
<thead>
<tr>
<th>Example 1</th>
<th>Sample Responses</th>
</tr>
</thead>
</table>
| Jill wanted to buy a kitten, so she went to see Mrs. Smith, who had lots of kittens that she didn't want. Now Mrs. Smith loved the kittens, and she wouldn't do anything to harm them, though she couldn't keep them all herself. When Jill visited she wasn't sure she wanted one of Mrs. Smith's kittens, since they were all males and she had wanted a female. But Mrs. Smith said, "If no one buys the kittens I'll just have to drown them!" | 2 points:  
So Jill will feel sorry for the kittens and want to buy one of them  
To manipulate or threaten her to take one  

1 point:  
Because she wanted Jill to buy one  
The old lady doesn't want to keep them all  

<table>
<thead>
<tr>
<th>Q: Why did Mrs. Smith say that?</th>
<th>Sample Responses</th>
</tr>
</thead>
</table>
|                                                                                             | 2 points:  
Because otherwise she would have to drown them  
She's a terrible woman and would kill them otherwise |
Example 1

*playful*  *comforting*

Example 2

*irritated*  *bored*

*terrified*  *upset*

*arrogant*  *annoyed*

**Figure C1**  Sample Items: Reading the Mind in the Eyes Test (Affective ToM)

Note: The correct answers are (1) playful and (2) upset.
Figure C2  Sample Items: Yoni Test (Cognitive & Affective ToM).

Note: The correct answers are (a) top right, (b) top left, (c) top right, and (d) top left.
Appendix D
Demographic and Health Questionnaire

*INDICATE ONLY CONDITIONS DIAGNOSED BY A HEALTH PROFESSIONAL.*

DEMOGRAPHIC AND HEALTH QUESTIONNAIRE

1. What is today’s date?

2. What is your Date of Birth?

3. What is your Sex? Male Female

4. In what City/Country were you born?
   (ex. Toronto, Canada): ____________________________

   4a. If you were born outside of North America, how many years ago did you move here? ____________________________

5. What is your height and weight?
   Height _________  Weight _________

6. What is your first language?
   (ex. English): ____________________________

7. What hand do you use to write?
   Right  Left

8. How many years in total have you completed in school? (please circle)
   1 2 3 4 5 6 7 8  9 10 11 12  1 2 3 4  5+
   Elementary  High School  College  Graduate  Medical  Law

9. What is the highest degree you earned in school?
   (ex. High School Diploma, or B.A.): ____________________________

10. What is your mother’s total years of education?

11. What is your father’s total years of education?

12. Do you have trouble with your vision that prevents you from reading ordinary print even when you have glasses on?
    No  Yes
13. Do you have trouble with your hearing that prevents you from hearing ordinary conversation?
   No  Yes

14. Do you wear a hearing aid?
   No  Yes

15. Do you drink alcoholic beverages?
   No  Yes

   **IF YES:**
   15a. On average, how many drinks do you consume per week? __________
   *(one drink = one ounce of hard liquor OR one beer OR one glass of wine)*

16. Have you ever been treated for alcohol or other drug abuse?
   No  Yes

   **IF YES:**
   16a. Do you continue to use alcohol or other substances?
   No  Yes

17. Do you smoke any tobacco products? (ex. cigarettes, pipe, cigars)
   No
   Yes, regularly
   Yes, every now and then
   No, I have previously smoked tobacco but have quit completely
   No, I have never smoked tobacco

   **IF YOU CURRENTLY/PREVIOUSLY HAVE SMOKED TOBACCO PRODUCTS:**
   17a. How long have you used/did you use tobacco products? __________

   17b. On average, how many packs do you smoke per week? __________
"INDICATE ONLY CONDITIONS DIAGNOSED BY A HEALTH PROFESSIONAL."

Instructions for the following questions:
The following questions refer to physical and mental health conditions that you may be experiencing. Please indicate that you have a condition only if you have been diagnosed by a physician or a mental health practitioner.

18. Have you been diagnosed with high blood pressure?
   No
   Yes, not serious
   Yes, moderately serious
   Yes, very serious

IF YES:
18a. How do you control it? (Please select all that apply)
   Diet/Weight control   Prescription medication
   Exercise             Supplements

18b. How long has it been since you were diagnosed?
   (ex. 2 years and 3 months) : _______________________

18c. If you take prescription medication to control it,
   What is the name and dosage of this medication? : _______________________
   Number of years you have been taking this medication : _______________________

19. Has a health professional recommended that your monitor your blood pressure regularly?
   No    Yes

20. Do you monitor your blood pressure?
   No    Yes

IF YES:
20a. How do you monitor your blood pressure? (please circle all that apply)
   Home blood pressure machine
   Pharmacy/drug store
   Doctor's office
   Other (please explain) ____________________________________________
20b. How often do you check your blood pressure? (circle that which applies)
Daily Weekly Monthly Yearly
Other (please explain) ________________________________

21. Have you been diagnosed with diabetes?
No
Yes, not serious
Yes, moderately serious
Yes, very serious

**IF YES:**
21a. How do you control it? (Please select all that apply)
Diet/Weight control Prescription medication
Exercise Supplements

21b. If you take prescription medication to control it,
What is the name and dosage of this medication: __________________
Number of years you have been taking this medication: _____________

21c. Please circle the type of diabetes: Type 1 Type 2

21d. How long has it been since you were diagnosed?
(ex. 2 years, 3 months): ______________

22. Have you been diagnosed with high cholesterol?
No
Yes, not serious
Yes, moderately serious
Yes, very serious

**IF YES:**
22a. How do you control it? (Please select all that apply)
Diet/Weight control Prescription medication
Exercise Supplements

22b. How long has it been since you were diagnosed?
(ex. 2 years, 3 months): ______________
"INDICATE ONLY CONDITIONS DIAGNOSED BY A HEALTH PROFESSIONAL"

22c. If you take prescription medication to control it,
What is the name and dosage of this medication?: ______________________
Number of years you have been taking this medication: ______________________

23. If you know your HDL and LDL levels from your most recent blood work
please indicate them below:

HDL: ______________________
LDL: ______________________

24. Have you ever had a heart attack?
No
Yes, not serious
Yes, moderately serious
Yes, very serious

IF YES:
24a. How many? ______________________

24b. How long has it been since your most recent heart attack?
(ex. 2 years and 3 months): ______________________

25. Have you ever suffered a stroke?
No
Yes, not serious
Yes, moderately serious
Yes, very serious

IF YES:
25a. How many? ______________________

25b. How long has it been since your most recent stroke?
(ex. 2 years and 3 months): ______________________

26. Have you been diagnosed with cardiovascular/heart disease?
No
Yes, not serious
Yes, moderately serious
Yes, very serious
"INDICATE ONLY CONDITIONS DIAGNOSED BY A HEALTH PROFESSIONAL."

IF YES:
26a. How long has it been since you were diagnosed with this?
(ex. 2 years and 3 months):

26b. How do you control it? (Please select all that apply)
Diet/Weight control
Exercise
Supplements
Prescription medication

26c. If you take prescription medication to control it,
What is the name and dosage of this medication?:
Number of years you have been taking this medication:

27. Have you been diagnosed with osteoporosis?
No
Yes, not serious
Yes, moderately serious
Yes, very serious

IF YES:
27a. How long has it been since you were diagnosed?
(ex. 2 years, 3 months):

27b. How do you control it? (Please select all that apply)
Diet/Weight control
Exercise
Supplements
Prescription medication
Over-the-counter pain medication (as needed)
Over-the-counter pain medication (regularly)

27c. If you take prescription medication to control it,
What is the name and dosage of this medication:
Number of years you have been taking this medication:

28. Have you been diagnosed with osteo-arthritis?
No
Yes, not serious
Yes, moderately serious
Yes, very serious

Page 6
"INDICATE ONLY CONDITIONS DIAGNOSED BY A HEALTH PROFESSIONAL"

IF YES:
28a. How do you control it? (Please select all that apply)
    Diet/Weight control  Over-the-counter pain medication (as needed)
    Exercise  Over-the-counter pain medication (regularly)
    Supplements  Prescription medication

28b. How long has it been since you were diagnosed with this?
   (ex. 2 years and 3 months):

28c. If you take prescription medication to control it.
   What is the name and dosage of this medication?:
   Number of years you have been taking this medication:

29. Have you been diagnosed with rheumatoid arthritis?
   No
   Yes, not serious
   Yes, moderately serious
   Yes, very serious

IF YES:
29a. How do you control it? (Please select all that apply)
    Diet/Weight control  Over-the-counter pain medication (as needed)
    Exercise  Over-the-counter pain medication (regularly)
    Supplements  Prescription medication

29b. How long has it been since you were diagnosed with this?
   (ex. 2 years and 3 months):

29c. If you take prescription medication to control it.
   What is the name and dosage of this medication?:
   Number of years you have been taking this medication:

30. Have you ever had problems with your thyroid?
   No
   Yes, not serious
   Yes, moderately serious
   Yes, very serious
"INDICATE ONLY CONDITIONS DIAGNOSED BY A HEALTH PROFESSIONAL"

IF YES:
30a. Please indicate what type of problems below (circle)
   Low thyroid                      High thyroid
   (hypothyroidism)                 (hyperthyroidism/Grave's disease)

30b. How do you control it? (Please select all that apply)
   Diet/Weight control          Prescription medication
   Exercise                      Supplements

30c. How long has it been since you were diagnosed with this?
   (ex. 2 years and 3 months):

30d. If you take prescription medication to control it,
   What is the name and dosage of this medication?: ______________________
   Number of years you have been taking this medication: _______________

31. Have you ever had cancer?
   No                                   Yes

IF YES:
31a. What type of cancer?: _______________
31b. Is it in remission?: _______________

32. Have you ever had brain surgery?
   No                                   Yes

33. Have you ever hit your head so hard that you had to stop what you were doing because of dizziness, disorientation, or unconsciousness?
   No                                   Yes

IF YES, please answer the following:
33a. How many of these events have you experienced in your life? (please circle)
   1        4
   2        5 or more
   3

Please answer the following questions based on your most severe event:
33b. How old were you when this event occurred? _______________
33c. Were you unconscious?
No                     Yes

33d. If you were unconscious, for how long?
Under 1 minute        11 to 15 minutes
2 to 5 minutes         More than 15 minutes
6 to 10 minutes

33e. For approximately how long after the event did you feel dazed, disoriented, or confused?
Less than 1 hour
1 to 10 hours
11 to 24 hours
More than 24 hours

33f. Did you go to the hospital?
No
Yes - not overnight
Yes - overnight

33g. What was the cause of the event?
Bicycle accident       Motor vehicle accident
Sport-related accident  Fight
Fall                    Other

34. Do you suffer from a sleep disorder?
No
Yes, not serious
Yes, moderately serious
Yes, very serious

IF YES:
34a. What sleep disorder(s) are you presently experiencing?
(ex. insomnia, narcolepsy, sleep apnea) : ____________________________

34b. How do you control it? (Please select all that apply)
Diet/Weight control    Over-the-counter medication (as needed)
Exercise               Over-the-counter medication (regularly)
Supplements            Prescription medication
34c. If you take prescription medication to control it,
What is the name and dosage of this medication?
Number of years you have been taking this medication:

35. Have you ever been diagnosed with a mental or emotional problem
(ex. depression, anxiety)?
No
Yes, not serious
Yes, moderately serious
Yes, very serious

IF YES:
35a. What kind of problems?: ________________________________

35b. Do you CURRENTLY have a diagnosis of any mental or emotional
problems?
No Yes

35c. IF CURRENT, what kind of problems?: ________________________________

35d. If you CURRENTLY take prescription medication for a mental or
emotional problem.
What is the name and dosage of this medication?: ________________________________
Number of years you have been taking this medication: ________________________________

36. Have you ever been hospitalized with mental or emotional problems?
No Yes

37. Does anybody in your family suffer from Huntington’s disease?
No Yes
38. Please check if you have been diagnosed with any of the following:

<table>
<thead>
<tr>
<th>Condition</th>
<th>Check if YES</th>
<th>Severity (circle)</th>
<th>Length of diagnosis</th>
<th>Medication (name/dosage)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parkinson's disease</td>
<td></td>
<td>Mild</td>
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<td></td>
<td></td>
<td>Moderate</td>
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<tr>
<td></td>
<td></td>
<td>Severe</td>
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<tr>
<td>Huntington's disease</td>
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<td></td>
<td></td>
<td>Moderate</td>
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<tr>
<td></td>
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<td>Severe</td>
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<tr>
<td>Multiple sclerosis</td>
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<td></td>
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<td></td>
<td>Severe</td>
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<tr>
<td>Encephalitis/ Meningitis</td>
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<td>Mild</td>
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<td></td>
<td></td>
<td>Moderate</td>
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<tr>
<td></td>
<td></td>
<td>Severe</td>
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<td></td>
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<tr>
<td>Brain tumour</td>
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<td></td>
<td></td>
<td>Moderate</td>
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<td>Severe</td>
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<tr>
<td>Epilepsy</td>
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<td>Mild</td>
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<td>Moderate</td>
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<td></td>
<td>Severe</td>
<td></td>
<td></td>
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<tr>
<td>HIV/AIDS</td>
<td></td>
<td>Mild</td>
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<td></td>
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<td>Moderate</td>
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<td>Severe</td>
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<tr>
<td>Hepatitis C</td>
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<tr>
<td>Learning disability</td>
<td></td>
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<td>Severe</td>
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<tr>
<td>Attention disability</td>
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<td>Moderate</td>
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<td></td>
<td></td>
<td>Severe</td>
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</tbody>
</table>
39. Do you have any other health problems that were not asked about on this questionnaire?
   No      Yes

39a. If YES, please elaborate:

40. Please list all medications (including supplements) you are currently taking including their dosage:

<table>
<thead>
<tr>
<th>Name</th>
<th>Dosage</th>
<th>Reason</th>
</tr>
</thead>
</table>

"END OF QUESTIONS"