

COMMENT

Dementia Has a Categorical, Not Dimensional, Latent Structure

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Recently, Walters (“Dementia: Continuum or distinct entity?”, *Psychology and Aging*, 2010, 25, 534–544) published a taxometric study suggesting a dimensional latent structure for the construct of dementia. However, because that study did not conceptualize dementia according to accepted conventions (i.e., there were no measures of cognitive change or independent functioning), its results may represent a false negative error caused by insufficient content coverage. We replicated Walters, and we used the same taxometric methods and the same data source—but with indicators of cognitive change and functional independence. Our results support a categorical interpretation of dementia; whereas Walters’ results suggest that cognitive ability, rather than dementia, is dimensional in nature.

Keywords: taxometrics, latent structure, dementia, neuropsychological

Dementia has been defined as the presence of acquired deficits in memory and at least one additional cognitive domain, which represent a decline from previous ability levels and interfere with social or occupational functioning (American Psychiatric Association, 2000). Recent efforts to apply modern scientific advances, such as biomarkers, toward redefining dementia have retained the core features of (a) cognitive impairment, (b) a decline from previous levels of functioning, and (c) impaired activities of daily living (ADLs; Jack et al., 2011; McKhann et al., 2011). Many clinical criteria for neurodegenerative diseases require the presence of dementia for diagnosis. In Alzheimer’s disease (AD), for instance, the effect of cognitive difficulties on independent functioning (i.e., ADLs) is an important diagnostic consideration (American Psychiatric Association, 2000; McKhann et al., 1984, 2011); individuals with cognitive impairment—but little to no change in ADLs—may not meet criteria for dementia but, instead, may be diagnosed with mild cognitive impairment (MCI; Albert et al., 2011; Petersen et al., 2001; Winblad et al., 2004). In fact, a recent consensus statement by the National Institute on Aging and

the Alzheimer’s Association workgroup stated that “the differentiation of dementia from MCI rests on the determination of whether or not there is significant interference in the ability to function at work or in usual daily activities” (McKhann et al., 2011, p. 265). In other words, dementia cannot be diagnosed solely on the basis of low cognitive functioning; rather, evidence of cognitive decline and functional impairment are also necessary.

Recently, a study by Walters (2010) investigated the latent structure of dementia to determine whether it is best conceptualized as categorical (taxonic) or dimensional (continuous) in nature. The findings of that study suggested that—in contrast to the widely assumed notion that dementia is a discrete clinical entity—dementia is better understood as one extreme on a continuum of normal cognitive aging. Although the implications of those findings are extremely important, the results may be misleading due to the author’s choice of variables used as indicators of dementia. In his taxometric analysis of dementia, Walters chose four indicators: Delayed Logical Memory, a measure of delayed verbal recall from the Wechsler Memory Scales-Revised (WMS-R; Wechsler, 1987); Backward Digit Span, a measure of verbal attention and working memory from the Wechsler Adult Intelligence Scales-Revised (WAIS-R; Wechsler, 1981); Trail Making Test Part B, a measure of cognitive flexibility, mental set-shifting, and visuomotor processing speed; and the 30-item (odd numbers) version of the Boston Naming Test, a measure of visual confrontation naming. In Walters’ study, these four indicators provided cross-sectional information about participants’ cognitive functioning; however, as mentioned previously, low cognitive functioning, in and of itself, is not a sufficient indicator of dementia (McKhann et al., 2011). Without taking into account change in cognitive functioning and ADLs, a taxometric investigation that only measures static cognitive functioning does not adequately measure the construct of dementia. Although methodologically sound, Walters’ study appears to merely illustrate that *cognitive ability* is continuous. We undertook the present study in order to determine whether or not

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there is evidence for a dimensional structure of dementia when considering cognitive decline and ADLs in conjunction with cognitive ability.

In addition to the study by Walters (2010), the latent structure of dementia was also studied by Golden (1982); Golden found evidence to support a categorical interpretation of dementia. As such, the only two known taxometric studies to investigate the latent structure of dementia have produced discordant results. The two studies differ in several important ways. Walters relied upon more modern taxometric methods; thus, his work may be considered a stronger test of the latent structure. On the other hand, Golden utilized indicators of independent functioning and, implicitly, change from a previous level of cognitive functioning (e.g., by including items such as “does not know own age”), which, as addressed above, is one of the major weaknesses in Walters’ study. We believe that by combining the strengths of both studies—namely, by using more modern taxometric methods and more appropriate indicators of dementia—we can help to resolve these conflicting results.

We sought to replicate Walters’ (2010) taxometric analyses—using the same database that was used in that study—but our approach differed in that we sought to include indicators of ADLs and change from previous ability levels.¹ Because the causes of dementia are believed to be distinct from normal aging, it follows that the resulting dementia syndrome is also categorically distinct from normal aging. We hypothesized that when the taxometric analysis of dementia includes measures of ADLs and cognitive change, in addition to cross-sectional cognitive data, a dimensional structure will not be supported. We predicted that the current study would provide converging evidence for a categorical latent structure of dementia.

Method

Participants

Participant data were obtained by a request to the National Alzheimer’s Coordinating Center (NACC). The NACC coordinates data collection across 29 NIA-funded Alzheimer’s disease Centers (ADCs) nationwide. Each ADC collects data for the Uniform Data Set (UDS; Beekly et al., 2007; Morris et al., 2006). The UDS is a standardized set of questions and test procedures given to participants and their study partners; it has been in place since 2005. Participants are studied longitudinally via annual evaluations that ask participants to provide a detailed medical history, family history, and social history; they are also asked to complete questionnaires and interviews pertaining to their current cognitive health and any changes in cognitive function. Participants are examined via neurologic examination and neuropsychological evaluation. Each year, based on the results of that year’s evaluation, a clinician or consensus team at each ADC decides on a diagnosis based on standard criteria (e.g., McKhann et al., 1984). We requested from NACC data provided by participants whose baseline visit was in 2005 or later (UDS era) and who were 65 years or older at baseline; who spoke English as their primary language; and with no history of stroke, seizures, Parkinson’s disease, central nervous system neoplasm, recent or active abuse of alcohol or other substances, or active psychiatric disorders. The

data obtained from NACC contained 27,892 person-visits between 2005 and 2010, and the data were from 12,069 unique participants.

Measures

The UDS contains the following test variables used to measure cognitive functioning.

The Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) is a 30-point instrument used to obtain a screening measurement of mental status, including orientation, attention, working memory, short-term recall, language, and visuospatial construction. The total score ranges from 0 to 30. Higher scores represent higher cognitive functioning.

Animal and Vegetable Fluency are two tests of rapid verbal generativity for lexical-semantic access to members of a specific category. Higher scores represent better performance.

Boston Naming Test (BNT; 30-item [odd] version; Goodglass, Kaplan, & Barresi, 2001; Kaplan, Goodglass, & Weintraub, 1983), a test of visual confrontation naming, is used to evaluate one’s ability to name common objects. Possible scores range from 0 to 30. Higher scores represent better performance.

Logical Memory, Immediate and *Logical Memory, Delayed* (LM-I and LM-D; Wechsler, 1987) are tests used to measure immediate and delayed free recall of a prose narrative. Participants are read Story A from the WMS-R Logical Memory subtest, they are asked to repeat it from memory immediately after hearing it, and they are asked to freely recall the story after a 20 to 30-min delay. Both immediate and delayed scores range from 0 to 25. Higher scores represent better performance.

Digit Span Forward and Digit Span Backward (DS-F and DS-B; Wechsler, 1981) measure verbal attention and working memory, respectively. In the DS-F condition, participants are asked to repeat a series of digits; whereas in the DS-B condition, participants are asked to repeat the digits in reverse. Scores range from 0 to 12 in both conditions. Higher scores reflect better performance.

Digit Symbol (Wechsler, 1981) is a measure of visuomotor processing speed and attention that requires participants to quickly match symbols to numbers by drawing symbols below the correct numbers. Possible scores range from 0 to 93. Higher scores reflect better performance.

Trail Making Test Parts A and B (TMT-A and TMT-B; Reitan & Wolfson, 1993) require participants to connect numbers (Part A) or numbers and letters (Part B) pseudorandomly scattered across a page. Part A measures simple visuomotor attention and processing speed; possible scores range from 0 to 150 s. Part B also requires visuomotor attention and processing speed; but, it also requires planning, mental flexibility, and response set maintenance. Scores on Part B range from 0 to 300 s. For both parts, a higher score reflects a worse performance.

The UDS also contains the Functional Activities Questionnaire (FAQ; Pfeffer, Kurosaki, Harrah, Chance, & Filos, 1982), completed by a study partner, which provides information about participants’ ability to perform 10 instrumental activities of daily living (IADLs). The FAQ is a reliable and valid measure of IADLs

¹ Please refer to Walters (2010) for a more complete description of the methods, replicated here.

that is sensitive and specific to the presence of dementia (Juva et al., 1997; Olazarán, Mourante, & Bermejo, 2005; Teng et al., 2010). The NACC version of the FAQ differs from the original version in that it allows respondents to select “8,” (N/A). When deriving a total FAQ score, we excluded any “8” response. We essentially treated it as a score of 0 for that item. FAQ total scores range from 0 to 30. Higher scores reflect greater dependence on others to complete IADLs.

Procedure

Because severe cognitive impairment can prevent an individual from completing cognitive testing, we expected that our missing data were not random. However, listwise deletion of participants whose test scores were missing due to cognitive difficulties would have biased our sample; that is, by excluding the most severely demented individuals, we would lose important information about the underlying latent structure of dementia. Therefore, when data were coded as missing due to cognitive impairment (e.g., not due to physical difficulties, verbal refusal, or unknown reasons), we assigned the worst possible score for that test. We decided against using a modeling approach (e.g., multiple imputation, selection models) to handling missing data for two reasons. First, most modeling approaches for handling missing data assume that data are either missing at random or missing completely at random. When data are missing systematically along with other variables of interest, it is more difficult to make an unbiased estimate of missing data values. Second, UDS research protocol stipulates that when a person is unable to complete a test due to cognitive impairment, the data are coded as missing due to cognitive/behavioral problems. However, this is not consistent with the actual scoring procedures for the tests themselves. For example, if a person cannot complete Delayed Logical Memory due to cognitive problems, standard scoring procedures indicate that the person's score on the test should be 0 because he or she did not recall any details from the story. This approach to handling missing data is therefore consistent with the manner in which the cognitive tests are most commonly used, and it should make the data more generalizable to other settings. If data were missing for other reasons (e.g., physical difficulties, verbal refusal), those participants were excluded.

As the goal of the current study was to replicate Walters' (2010) analyses, but with the inclusion of additional information as described above, our selection procedures differed from the original study. In order to quantify participants' change in cognitive functioning from a previous level, we employed the following procedures. First, we identified all participants with data from at least three annual visits. A time period of two years (three visits) was chosen in order to provide a more robust measure of change compared with only one year (two visits), while retaining a larger sample than would be available with a requirement of at least three years of data (four or more visits). We designated the participants' most recent visit as Time 0 (T0), the second most recent visit as Time -1 (T-1), and their third most recent visit as Time -2 (T-2). Data from visits prior to T-2 were not utilized because these data would have been from visits outside of our desired time period. At T0 and T-2, we created a summary cognitive test score based on the UDS neuropsychological tests administered to the participants. This allowed us to compare each participant's test scores to scores

from a group of cognitively healthy older adults in order to scale all tests on the same metric, to generate an estimate of global cognitive functioning at each visit, and to estimate the magnitude of change across visits using the same tests. For each of the test variables listed above, we compared raw scores to the means and standard deviations reported in Weintraub et al. (2009) to derive standardized scores (z -scores; $M = 0$, $SD = 1$). We then calculated an average cognitive z -score for each visit by summing the 11 individual z -scores and dividing by 11. This provided a general estimate of the participants' global cognitive functioning at each visit. We then subtracted the T-2 summary z -scores from the T0 summary z -scores to derive an index of global cognitive change across three visits, or approximately two years. This variable, abbreviated Δz , indicated the change in z -scores between T-2 and T0; the lower the score, the more pronounced the cognitive decline. We chose not to examine decline on individual tests because of the improved reliability of the composite change score, and we wanted to adhere to a conceptualization of the diagnostic criteria for dementia, which do not require test-specific cognitive decline.

Following the implementation of these procedures, our sample included 4,189 individuals with no missing data on any variable. Participants ranged in age from 66 to 104 ($M = 79.9$, $SD = 6.9$), and participants ranged in years of education from 1 to 29 ($M = 15.3$, $SD = 3.0$). The sample contained 2,436 women (58.2%) and 1,753 men (41.8%). In terms of race, the sample included 3,559 (85%) White, 553 (13.2%) Black, 7 (0.2%) American Indian or Alaska Native, 2 (0.05%) Native Hawaiian or other Pacific Islander, 51 (1.2%) Asian, and 11 (0.3%) individuals of other races. Race was unknown for 6 (0.1%) participants. Hispanic ethnicity was endorsed by 78 (1.9%) participants. In terms of diagnosis, 1,298 (31.0%) individuals were diagnosed with dementia (AD, $n = 1,194$ [92.0%]; dementia with Lewy bodies, $n = 95$ [7.3%]; frontotemporal dementia, $n = 48$ [3.7%]; vascular dementia, $n = 44$ [3.4%]; other dementia etiologies, $n = 309$ [23.8%]; because of comorbidities, percentages total greater than 100%); 2,073 (49.5%) individuals were diagnosed as cognitively normal, and the remaining 818 (19.5%) individuals were diagnosed with MCI or an ambiguous diagnosis (e.g., “impaired, not MCI”). In 75.7% of the cases, the diagnosis was made by a multidisciplinary consensus team, and in the remaining 24.3% of the cases, the diagnosis was made by a single clinician.

Taxometric analyses were initiated with 13 total indicators; 11 were measures of cognitive functioning at T0 (MMSE, animal fluency, vegetable fluency, BNT, LM-I, LM-D, DS-F, DS-B, Digit Symbol, TMT-A, TMT-B), one was a measure of IADLs at T0 (FAQ), and one was a measure of change in cognitive functioning from T-2 to T0 (Δz). Raw scores were used for all variables; in order to ensure unidirectional scaling, we transformed variables, when necessary, to ensure that high scores on all tests were associated with a greater degree of impairment. We also included a variable to indicate putative taxon membership (i.e., taxon vs. complement). Any participant whose ADC had assigned to them a consensus or clinician diagnosis of dementia at their T0 visit was coded as a putative taxon member ($n = 1,298$; 31%), and the remaining participants were coded as a putative member of the complement group ($n = 2,891$; 69%).

Data Analysis

Like Walters (2010), we implemented Ruscio's TaxProg taxometrics program (version 2010–07-26; Ruscio, 2010; Ruscio, Haslam, & Ruscio, 2006) in R (version 2.12.1; R Development Core Team, 2011). Using this software, we examined the data to determine whether it met several conditions necessary for taxometric analysis. First, we sought to identify and exclude any test variables with low test validity, as measured by the standardized difference between putative taxon members and complement group members, in terms of Cohen's (1988) d statistic. A d value greater than 1.25 is preferred for taxometric analyses (Meehl, 1995). This procedure allowed us to eliminate DS-F ($d = 0.96$) and DS-B ($d = 1.21$) due to low validity. We then examined the covariance matrices for all of the remaining indicators in three groups: the full sample, the putative taxon group, and the putative complement group. Indicator covariance of greater than .30 is needed in the full sample, but in each of the separate groups, this must be less than .30 in order to keep "nuisance covariance" to a minimum (Meehl, 1995). We examined these three covariance matrices, and we excluded test variables that did not allow us to achieve a full sample covariance of greater than .30 and within-group covariances of less than .30. This process led to the selection of four indicator variables: LM-D ($d = 2.39$), TMT-B ($d = 2.03$), FAQ ($d = 3.39$), and Δz ($d = 1.33$). The full sample indicator covariance among these variables was $r = .610$; within the putative taxon group, the indicator covariance was $r = .299$; and within the putative complement group, the covariance was $r = .272$. Thus, these four indicators were determined suitable for taxometric analysis. Like Walters, our indicators included LM-D and TMT-B, but we differed in our use of FAQ and Δz instead of DS-B and BNT. It is important to note that we met our goal of including indicators related to independent functioning (FAQ), change from a previous level of functioning (Δz), and current cognitive functioning (LM-D, TMT-B).

To replicate the analyses conducted by Walters (2010), our taxometric procedures included mean above minus below a cut (MAMBAC; Meehl & Yonce, 1994), maximum covariance (MAXCOV; Meehl & Yonce, 1996), and L-Mode (Waller & Meehl, 1998). For each procedure, we used the default settings in Ruscio's (2010) TaxProg software, which yields a comparison curve fit index (CCFI) to assist with interpretation of the results. CCFI values of less than .40 are associated with a greater fit to a simulated dimensional structure, whereas values of greater than .60 are associated with a greater fit to a simulated categorical structure; values between .40 and .60 are thought to be too ambiguous for interpretation.

Results

MAMBAC, MAXCOV, and L-Mode yielded CCFI values of .756, .739, and .757, respectively ($M = .751$, $SD = 0.01$); all of these support a categorical interpretation (Ruscio, Walters, Marcus, & Kaczetow, 2010). The curves produced by these methods are in Figure 1; in the left panels, the curves are compared with simulated categorical data, and in the right panels, the curves are compared with simulated dimensional data. Visually, the real data produced curves that better fit the simulated categorical data, consistent with the CCFI values. These findings are quite different

from those described by Walters (2010); Walters reported CCFI values of .332, .152, and .251 for MAMBAC, MAXCOV, and L-Mode, respectively. In the current study, MAMBAC and MAXCOV yielded base rate estimates of .237 ($SD = 0.17$) and .242 ($SD = 0.07$), respectively, which are lower than those reported by Walters.

Based on these taxometric analyses, we used Ruscio's (2010) P.Classify routine to assign participants to one of two predicted diagnostic groups—demented or not demented—on the basis of their total scores from all of the four indicators. Using an estimated base rate of .31 (as was observed in the current sample), we assigned the 1,298 participants with the highest total scores to the demented group and the remaining 2,891 participants to the not demented group. We then compared the concordance between these predicted groupings and the actual diagnostic status (demented or not demented) of the participants. The sensitivity and specificity of these predicted groupings to the UDS clinical consensus diagnosis were .77 and .90, respectively, which yield positive and negative predictive values of .77 and .90, respectively (the number of false positives and false negatives both equaled 303, which caused these values to be equal).

Discussion

In contrast to the findings reported by Walters (2010), who found evidence to suggest that the construct of dementia is best conceptualized as a continuous variable, our replication of these analyses supports the notion that dementia is a discrete categorical entity; the positive and negative predictive values reported here also lend support to the external validity of these findings in the diagnosis of dementia. Our impetus for replicating these analyses was due to the fact that two previous taxometric studies reported conflicting results, possibly due to differences in methodology and conceptualization. While the Walters study was methodologically sound, its four indicators of dementia were all measures of cognitive functioning at a single point in time. The major flaw in that approach is that it fails to take into account the fact that dementia cannot simply be defined as low cognitive functioning. The concept of dementia refers to the combination of cognitive impairment, change in cognitive functioning from previous levels, and impairment in one's ability to function independently (American Psychiatric Association, 2000; McKhann et al., 2011). We believe that our results conflict with those of Walters' because we included the FAQ, a measure of IADLs, and Δz , a measure of global cognitive change across three annual visits. Our indicators also measure episodic memory (LM-D) and multimodal cognitive functioning related to dementia (TMT-B; Bell-McGinty, Podell, Franzen, Baird, & Williams, 2002; Greenleaf, Margolis, & Erker, 1985; Spreen & Benton, 1965); together they provide excellent content coverage of the dementia construct. Although our results conflict with Walters', they are consistent with Golden (1982) and modern conceptualizations of dementia (McKhann et al., 2011) as a state that is only entered into as the result of neurological disruption—with this state being qualitatively distinct from "normal" (i.e., nondisordered) human cognitive aging.

Rather than a distinct disease entity, dementia represents a final common clinical pathway for a number of progressive (e.g., AD, frontotemporal lobar degeneration, chronic traumatic encephalopathy) and reversible (e.g., hypothyroidism, vitamin B12 defi-

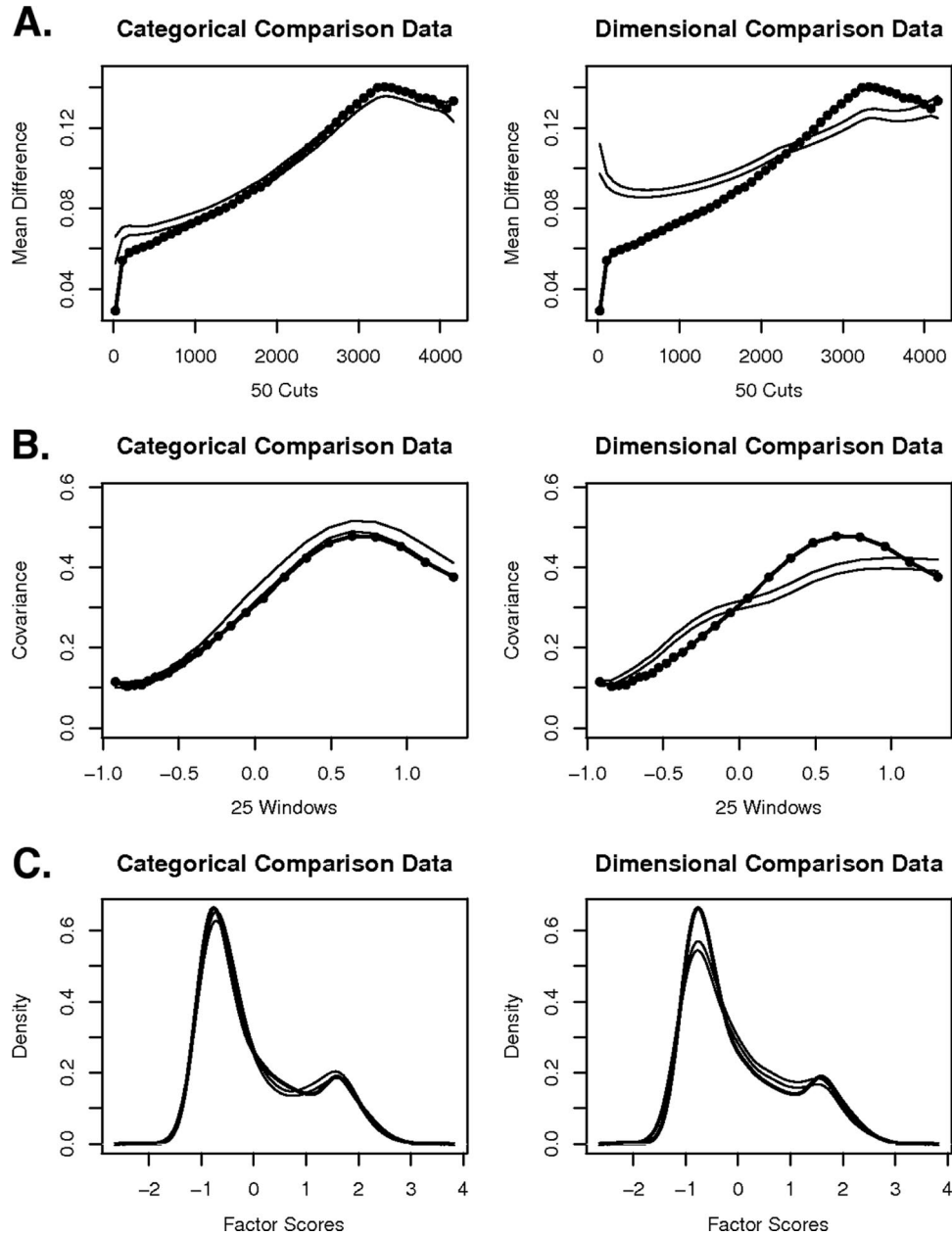


Figure 1. MAMBAC (A), MAXCOV (B), and L-Mode (C) curves. The left panels show the real data (heavy line) compared with data simulated with taxonicity assumed (double lines; ± 1 SD), whereas the right panels show the real data (heavy line) compared with data simulated with dimensionality assumed (double lines; ± 1 SD). In all cases, the real data more closely approximate a categorical latent structure.

ciency, major depression) medical conditions. The mere presence of dementia does not provide insight into its cause. From a developmental perspective of aging, knowing whether the latent structure of dementia is categorical or continuous does not appear to provide any unique insight into understanding normal versus abnormal aging processes in older adulthood. In contrast, investigating the latent structure of specific aging-related abnormalities such as AD may have the potential to inform prevention, detection, treatment, and caregiving efforts. For instance, Reser (2009) pos-

ited that AD may represent a specific evolutionarily mediated downregulation of metabolically expensive cerebral tissue that was advantageous in ancestral eras where aging was associated with a reduction in one's ability to obtain calories. If true, Reser's theory suggests that, in contrast to conventional wisdom, AD may actually be an extreme state on a continuum of normal aging. In contrast, a brain tumor may be another cause of dementia, but it is very implausible that this cause falls on a dimension of normal aging. Taxometrics can provide a method for testing the dimen-

sional theory of AD by examining the latent structure indicated by its specific clinical, biomarker, and neuropathological features. Support for a dimensional model of AD may have important implications for future efforts to detect and treat the disease as early as possible. Unfortunately, understanding the latent structure of dementia does not appear to offer the same possibilities for scientific advancement.

On the topic of AD, we must disagree with some of the conclusions drawn by Walters (2010). For example, previous research suggests that there may be important genetic and etiologic distinctions between early onset AD and sporadic AD (Rocchi, Pellegrini, Siciliano, & Murri, 2003). Walters conflates dementia (a clinical outcome) and AD (a pathological process) when attempting to address this issue. Early onset AD may differ from sporadic AD in many ways, but conclusions about whether or not these two disease processes are categorical or continuous cannot be made if the focus of the taxometric investigation is on a nonspecific phenotypic expression (dementia) that can be caused by conditions other than AD. Because approximately 20% of the cases of dementia studied by Walters were due to causes other than AD, it is impossible for these data to uncover meaningful information about the pathogenesis of AD. However, on the basis of these data, Walters concluded that the difference between early onset AD and sporadic AD is a matter of degree rather than kind. Meaningful advances in the understanding of normal and abnormal aging processes are more likely to emerge from taxometric investigations of specific disease processes (e.g., AD) as opposed to disease outcomes with a multitude of causes (e.g., dementia, aphasia).

The most recent guidelines for the diagnosis of dementia and Alzheimer's disease (Jack et al., 2011) indicate an important shift in the conceptualization of aging-related neurodegenerative conditions. Emphasis has been displaced from dementia to preclinical detection using a combination of cognitive measures and biomarkers (e.g., Albert et al., 2011; Dubois et al., 2007, 2010; Sperling et al., 2011). Based on taxometric research, it appears as though cognitive ability exists on a continuum (Walters, 2010), and when cognitive decline begins to exert a meaningful impact on one's functioning, a qualitatively distinct state of dementia emerges. These findings highlight the importance of neuropsychological assessment measures that are capable of reliably tracking longitudinal change (Mungas et al., 2010), and they highlight the importance of classification accuracy (O'Connell & Tuokko, 2010) when diagnosing dementia.

References

- Albert, M. S., DeKosky, S. T., Dickson, D., Dubois, B., Feldman, H., Fox, N. C., . . . Phelps, C. H. (2011). The diagnosis of mild cognitive impairment due to Alzheimer's disease: Recommendations from the National Institute on Aging and Alzheimer's Association workgroup. *Alzheimer's & Dementia*, 7, 270–279. doi:10.1016/j.jalz.2011.03.008
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (Revised 4th ed.). Washington, DC: Author.
- Beekly, D. L., Ramos, E. M., Lee, W. W., Deitrich, W. D., Jacka, M. E., Wu, J., . . . the NIA ADCs. (2007). The National Alzheimer's Coordinating Center (NACC) Database: The Uniform Data Set. *Alzheimer Disease and Associated Disorders*, 21, 249–258. doi:10.1097/WAD.0b013e318142774e
- Bell-McGinty, S., Podell, K., Franzen, M., Baird, A. D., & Williams, M. J. (2002). Standard measures of executive function in predicting instrumental activities of daily living in older adults. *International Journal of Geriatric Psychiatry*, 17, 828–834. doi:10.1002/gps.646
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*. Hillsdale, NJ: Lawrence-Erlbaum.
- Dubois, B., Feldman, H. H., Jacova, C., Cummings, J., Dekosky, S. T., Barberger-Gateau, P., . . . Scheltens, P. (2010). Revising the definition of Alzheimer's disease: A new lexicon. *The Lancet Neurology*, 9, 1118–1127. doi:10.1016/S1474-4422(10)70223-4
- Dubois, B., Feldman, H. H., Jacova, C., Dekosky, S. T., Barberger-Gateau, P., Cummings, J., . . . Scheltens, P. (2007). Research criteria for the diagnosis of Alzheimer's disease: Revising the NINCDS-ADRDA criteria. *The Lancet Neurology*, 6, 734–746. doi:10.1016/S1474-4422(07)70178-3
- Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "Mini-mental state." A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, 12, 189–198. doi:10.1016/0022-3956(75)90026-6
- Golden, R. R. (1982). A taxometric model for the detection of a conjectured latent taxon. *Multivariate Behavioral Research*, 17, 389–416. doi:10.1207/s15327906mbr1703_6
- Goodglass, H., Kaplan, E., & Barresi, B. (2001). *Boston Diagnostic Aphasia Examination Third Edition* (BDAE-3). Austin, TX: Pro-Ed.
- Greenleaf, C. L., Margolis, R. B., & Erker, G. J. (1985). Application of the Trail Making Test in differentiating neuropsychological impairment of elderly persons. *Perceptual and Motor Skills*, 61, 1283–1289. doi:10.2466/pms.1985.61.3f.1283
- Jack, C. R., Albert, M. S., Knopman, D. S., McKhann, G. M., Sperling, R. A., Carrillo, M. C., . . . Phelps, C. H. (2011). Introduction to the recommendations from the National Institute on Aging and the Alzheimer's Association workgroup on diagnostic guidelines for Alzheimer's disease. *Alzheimer's & Dementia*, 7, 257–262. doi:10.1016/j.jalz.2011.03.004
- Juva, K., Mäkelä, M., Erkinjuntti, T., Sulkava, R., Ylikoski, R., Valvanne, J., & Tilvis, R. (1997). Functional assessment scales in detecting dementia. *Age and Ageing*, 26, 393–400. doi:10.1093/ageing/26.5.393
- Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *The Boston Naming Test*. Philadelphia: Lea and Febiger.
- McKhann, G., Drachman, D., Folstein, M., Katzman, R., Price, D., & Stadlan, E. M. (1984). Clinical diagnosis of Alzheimer's disease: Report of the NINCDS-ADRDS Work Group under the auspices of Department of Health and Human Services Task Force on Alzheimer's disease. *Neurology*, 34, 939–944.
- McKhann, G. M., Knopman, D. S., Chertkow, H., Hyman, B. T., Jack, C. R. Jr., Kawas, C. H., . . . Phelps, C. H. (2011). The diagnosis of dementia due to Alzheimer's disease: Recommendations from the National Institute on Aging and the Alzheimer's Association workgroup. *Alzheimer's & Dementia*, 7, 263–269. doi:10.1016/j.jalz.2011.03.005
- Meehl, P. E. (1995). Bootstraps taxometrics: Solving the classification problem in psychopathology. *American Psychologist*, 50, 266–275. doi:10.1037/0003-066X.50.4.266
- Meehl, P. E., & Yonce, L. J. (1994). Taxometric analysis: I. Detecting taxonicity with two quantitative indicators using means above and below a sliding cut (MAMBAC procedure). *Psychological Reports*, 74, 1059–1274.
- Meehl, P. E., & Yonce, L. J. (1996). Taxometric analysis: II. Detecting taxonicity using covariance of two quantitative indicators in successive intervals of a third indicator (MAXCOV procedure). *Psychological Reports*, 78, 1091–1227. doi:10.2466/pr0.1996.78.3c.1091
- Morris, J. C., Weintraub, S., Chui, H. C., Cummings, J., Decarli, C., Ferris, S., . . . Kukull, W. A. (2006). The Uniform Data Set (UDS): Clinical and cognitive variables and descriptive data from Alzheimer disease centers. *Alzheimer Disease and Associated Disorders*, 20, 210–216. doi:10.1097/01.wad.0000213865.09806.92

- Mungas, D., Beckett, L., Harvey, D., Farias, S., Reed, B., Carmichael, O., . . . DeCarli, C. (2010). Heterogeneity of cognitive trajectories in diverse older persons. *Psychology and Aging, 25*, 606–619. doi:10.1037/a0019502
- O'Connell, M. E., & Tuokko, H. (2010). Age corrections and dementia classification accuracy. *Archives of Clinical Neuropsychology, 25*, 126–138. doi:10.1093/arclin/acp111
- Olazarán, J., Mourante, P., & Bermejo, F. (2005). Clinical validity of two scales of instrumental activities in Alzheimer's disease. *Neurologia, 20*, 395–401.
- Petersen, R. C., Doody, R., Kurtz, A., Mohs, R. C., Morris, J. C., Rabins, P. V., . . . Winblad, B. (2001). Current concepts in mild cognitive impairment. *Archives of Neurology, 58*, 1985–1992. doi:10.1001/archneur.58.12.1985
- Pfeffer, R. I., Kurosaki, T. T., Harrah, C. H. Jr., Chance, J. M., & Filos, S. (1982). Measurement of functional activities in older adults in the community. *Journal of Gerontology, 37*, 323–329.
- R Development Core Team. (2011). R: A language and environment for statistical computing [Computer software]. Vienna, Austria: R Foundation for Statistical Computing. Retrieved from <http://www.R-project.org>
- Reitan, R. M., & Wolfson, D. (1993). *The Halstead-Reitan neuropsychological test battery: Theory and clinical interpretation* (2nd ed.). Tuscon, AZ: Neuropsychology Press.
- Reser, J. E. (2009). Alzheimer's disease and natural cognitive aging may represent adaptive metabolism reduction programs. *Behavioral and Brain Functions, 5*, 13–26. doi:10.1186/1744-9081-5-13
- Rocchi, A., Pellegrini, S., Siciliano, G., & Murri, L. (2003). Causative and susceptibility genes for Alzheimer's disease: A review. *Brain Research Bulletin, 61*, 1–24. doi:10.1016/S0361-9230(03)00067-4
- Ruscio, J. (2010). Taxometric program code [Computer software]. Retrieved from <http://www.tcnj.edu/~ruscio/taxometrics.html>
- Ruscio, J., Haslam, N., & Ruscio, A. M. (2006). *Introduction to the taxometric method: A practical guide*. Mahwah, NJ: Erlbaum.
- Ruscio, J., Walters, G. D., Marcus, D. K., & Kacetow, W. (2010). Comparing the relative fit of categorical and dimensional latent variable models using consistency tests. *Psychological Assessment, 22*, 5–21. doi:10.1037/a0018259
- Sperling, R. A., Aisen, P. S., Beckett, L. A., Bennett, D. A., Craft, S., Fagan, A. M., . . . Phelps, C. H. (2011). Toward defining the preclinical stages of Alzheimer's disease: Recommendations from the National Institute on Aging and the Alzheimer's Association workgroup. *Alzheimer's & Dementia, 7*, 280–292. doi:10.1016/j.jalz.2011.03.003
- Spren, O., & Benton, A. L. (1965). Comparative studies of some psychological tests for cerebral damage. *Journal of Nervous and Mental Disease, 140*, 323–333. doi:10.1097/00005053-196505000-00002
- Teng, E., Becker, B. W., Woo, E., Knopman, D. S., Cummings, J. L., & Lu, P. H. (2010). Utility of the functional activities questionnaire for distinguishing mild cognitive impairment from very mild Alzheimer disease. *Alzheimer Disease and Associated Disorders, 24*, 348–353. doi:10.1097/WAD.0b013e3181e2fc84
- Waller, N. G., & Meehl, P. E. (1998). *Multivariate taxometric procedures: Distinguishing types from continua*. Thousand Oaks, CA: Sage.
- Walters, G. D. (2010). Dementia: Continuum or distinct entity? *Psychology and Aging, 25*, 534–544. doi:10.1037/a0018167
- Wechsler, D. (1981). *Wechsler Adult Intelligence Scale-Revised*. San Antonio, TX: Psychological Corporation.
- Wechsler, D. (1987). *Wechsler Memory Scale-Revised*. San Antonio, TX: Psychological Corporation.
- Weintraub, S., Salmon, D., Mercaldo, N., Ferris, S., Graff-Radford, N. R., Chui, H., . . . Morris, J. C. (2009). The Alzheimer's Disease Centers' Uniform Data Set (UDS): The neuropsychological test battery. *Alzheimer Disease and Associated Disorders, 23*, 91–101. doi:10.1097/WAD.0b013e318191c7dd
- Winblad, B., Palmer, K., Kivipelto, M., Jelic, V., Fratiglioni, L., Wahlund, L. O., . . . Petersen, R. C. (2004). Mild cognitive impairment – beyond controversies, towards a consensus: Report of the International Working Group on mild cognitive impairment. *Journal of Internal Medicine, 256*, 240–246. doi:10.1111/j.1365-2796.2004.01380.x

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