Published in final edited form as:
J Psychiatr Res. 2012 June ; 46(6): 782–789. doi:10.1016/j.jpsychires.2012.03.010.

The Latent Structure of Attention Deficit/Hyperactivity Disorder in an Adult Sample

David K. Marcus[a], **Alyssa L. Norris**[a], and **Emil F. Coccaro**[b]

[a]Department of Psychology, Washington State University, Pullman, WA 99164

[b]Department of Psychiatry & Behavioral Neuroscience, University of Chicago, 5841 South Maryland Avenue, Chicago, IL 60637

Abstract

The vast majority of studies that have examined the latent structure of attention deficit/hyperactivity disorder (ADHD) in children and adolescents have concluded that ADHD has a dimensional latent structure. In other words, ADHD symptomatology exists along a continuum and there is no natural boundary or qualitative distinction (i.e., taxon) separating youth with ADHD from those with subclinical inattention or hyperactivity/impulsivity problems. Although adult ADHD appears to be less prevalent than ADHD in youth (which could suggest a more severe adult ADHD taxon), researchers have yet to examine the latent structure of ADHD in adults. The present study used a sample ($N = 600$) of adults who completed a self-report measure of ADHD symptoms. The taxometric analyses revealed a dimensional latent structure for inattention, hyperactivity/impulsivity, and ADHD. These findings are consistent with previous taxometric studies that examined ADHD in children and adolescents, and with contemporary polygenic and multifactorial models of ADHD.

Keywords

ADHD; taxometric analysis; latent structure

Attention-deficit/hyperactivity disorder (ADHD) is one of the most prevalent psychiatric conditions, affecting roughly 8% of children and adolescents (CDC, 2005) and 4.4% of adults (Kessler et al., 2006) in the United States. Despite considerable advances in the diagnosis, treatment, and etiology of ADHD, a number of unresolved issues remain, including questions about (a) whether ADHD is the consequence of a single core deficit (e.g., Barkley, 1997) or whether multiple pathways can lead to ADHD (e.g., Sonuga-Barke, 2002), (b) how to understand the high comorbidity between ADHD and other externalizing disorders (e.g., Waschbusch, 2002), and (c) when symptoms of inattention and hyperactivity/impulsivity are sufficiently severe to warrant treatment and medication.

A clear understanding of the latent structure of ADHD can help inform many of these unresolved issues. In other words, knowing whether ADHD is a categorically distinct

Correspondence should be addressed to: David K. Marcus, Department of Psychology, Washington State University, Pullman, WA 99164, david.marcus@wsu.edu.

condition or "taxon" that is either present or absent (like Type 1 diabetes) or whether it is dimensional and exists on a continuum (like Type 2 diabetes), can inform questions of assessment, etiology, diagnosis, and treatment. In recent years a growing research literature has examined the latent structure of ADHD using a variety of statistical methods, including latent class analysis and Meehl's taxometric procedures (Waller & Meehl, 1998). Most (but not all) of these studies have yielded dimensional results. The findings from both Hudziak et al.'s (1998) and Neuman et al.'s (1999) latent class analyses of parent reports of adolescent symptoms suggested that these ADHD symptoms were continuously distributed and not taxonic. Similarly, using factor mixture modeling (which combines latent class analysis with factor analysis) to analyze parent reports of child ADHD symptoms in a national registry sample of male twins, Lubke and colleagues (2009) concluded that ADHD symptoms are dimensional and that children with ADHD are at the far end of this continuum. In contrast, Todd et al.'s (2001) latent class analysis of data from adolescent female twins yielded findings that could be interpreted as supporting a taxonic latent structure.

Three studies have used Meehl's taxometric procedures to examine the latent structure of ADHD. Unlike latent class analysis, Meehl's taxometric procedures were specifically designed to test whether a construct has a taxonic or dimensional latent structure. Haslam and colleagues (2006) applied taxometric procedures to parent report data from two epidemiological samples. One sample consisted of children between the ages of 6 and 12, and the other was an adolescent sample (13–17). Their analyses yielded dimensional findings in both samples. Using parent and teacher reports as well as measures of cognitive ability, Frazier et al. (2007) examined the latent structure of each set of symptoms associated with ADHD (e.g., academic underachievement, sustained attention), as well as a combination of ADHD symptoms across these domains. Like Haslam et al., Frazier and colleagues found consistent evidence that both ADHD and its components had a dimensional latent structure. Most recently, Marcus and Barry (2011) used taxometric procedures to examine data from the NICHD Study of Early Child Care and Youth Development that were collected when the children were in third grade. This data set included parent reports, standardized tests of achievement, and laboratory measures. These taxometric analyses yielded dimensional results for inattention, hyperactivity/impulsivity, and ADHD as a whole.

All of these ADHD studies that have yielded dimensional findings have used child or adolescent samples and the primary indicators in most of these studies have been parent reports of child symptoms. Given that ADHD is classified as a disorder "usually first diagnosed in infancy, childhood, or adolescence" (DSM-IV-TR; American Psychological Association, 2000), the focus on child and adolescent samples is appropriate. However, considering that the prevalence rate for adult ADHD is roughly half that for children and adolescents, it is conceivable that child and adolescent samples include individuals who do not belong in a putative ADHD taxon, but who have transient symptoms of inattention or hyperactivity/impulsivity, which might create the illusion of continuity across the ADHD spectrum. Perhaps a taxometric study of ADHD using an adult sample might reveal an ADHD taxon that was obscured in these child and adolescent samples. On the other hand, a replication of these dimensional results in an adult sample would strengthen the conclusion that ADHD and its various components are dimensional across the lifespan. Furthermore, whereas these other studies relied primarily on parent reports and not self-reports, a taxometric analysis of self-reported adult ADHD symptoms would test whether these dimensional findings are robust across multiple assessment methods.

Materials and Methods

Participants

Subjects—Six-hundred physically healthy subjects participated in this study. All subjects were systematically evaluated in regard to psychopathology as part of a larger program designed to study correlates of impulsive aggressive, and other personality-related, behaviors in human subjects. Subjects were recruited from clinical settings and through newspaper advertisements seeking out individuals who: a) reported psychosocial difficulty related to one or more Axis I and Axis II conditions (Axis I / II subjects: $n = 432$) or, b) had little evidence of psychopathology (Healthy Control subjects: $n = 168$). All subjects gave informed consent and signed the informed consent document approved by our Committee for the Protection of Human Subjects (IRB).

Of the subject group, 322 (53.7%) were male (mean ± sd for age = 35.3 ± 9.6 years; range: 18–65 years) and 278 (46.3%) were female (mean ± sd for age = 34.2 ± 10.0 years; range: 18–58 years). Three-hundred-thirty-five (55.8%) were Caucasian, 202 (33.7%) were African-American; the remaining 63 (10.5%) were self-identified as neither Caucasian nor African-American. Most subjects (60.3%) came from the upper middle two Hollingshead SocioEconomic Classes [I = 67 (11.2%); II = 243 (40.5%); III = 119 (19.8%); IV = 88 (14.7%); V = 83 (13.8%)].

Diagnostic Assessment—Axis I and Axis II Personality Disorder diagnoses were made according to DSM-IV criteria (American Psychiatric Association, 1994). The diagnosis of Intermittent Explosive Disorder was made by Research Criteria as previously described (Coccaro, 2011). Diagnoses were made using information from: (a) the Structured Clinical Interview for DSM Diagnoses (SCID-I; First et al., 1996) for Axis I disorders and the Structured Interview for the Diagnosis of DSM Personality Disorder [SIDP-IV; Pfohl et al., 1997) for Axis II disorders; (b) clinical interview by a research psychiatrist; and, (c) review of all other available clinical data. Final diagnoses were assigned by team best-estimate consensus procedures (Leckman et al., 1982; Klein et al., 1994) involving research psychiatrists and clinical psychologists as previously described (Coccaro et al., 2010). This methodology has previously been shown to enhance the accuracy of diagnosis over direct interview alone (Kosten & Rounsaville, 1992).

Diagnoses—Four-hundred-thirty-two subjects met DSM-IV criteria for an Axis I and/or II Disorder. Among this group, current Axis I disorders were as follows: Any Depressive Mood Disorder (n = 71); Any Anxiety Disorder (n = 100); Intermittent Explosive Disorder (n = 207); Other Impulse Control Disorder (n = 11); Substance Dependence Disorder (n = 13); Eating Disorder (n = 18); Somatoform Disorder (n = 7); Adjustment Disorder (n = 7); lifetime Axis I disorders were as follows: Any Depressive Mood Disorder (n = 222); Any Anxiety Disorder (n = 142); Intermittent Explosive Disorder (n = 273); Other Impulse Control Disorder (n = 30); Substance Dependence Disorder (n = 153); Eating Disorder (n = 36); Somatoform Disorder (n = 7); Adjustment Disorder (n = 23). Among the 363 subjects with an Axis II disorder 243 (67%) met DSM-IV criteria for a specific personality disorder as follows: a) Cluster A (n = 58): Paranoid (n = 53), Schizoid (n = 5), Schizotypal (n = 1); b) Cluster B (n = 170), Borderline (n = 105), Antisocial (n = 68); Narcissistic (n = 66); Histrionic (n = 14); c) Cluster C (n = 108): Obsessive-Compulsive (n = 65); Avoidant (n = 46); Dependent (n = 5). The remaining 120 (33%) of PD subjects were diagnosed as Personality Disorder-Not Otherwise Specified (PD-NOS). These subjects met DSM-IV General Diagnostic Criteria for Personality Disorder, had pathological personality traits from a variety of personality disorder categories and had clear evidence of impaired

psychosocial functioning [Mean (± SD) Global Assessment of Function score = 60.6 ± 7.4]. By definition, healthy controls had no current or life history of any Axis I or II disorder.

Measure

The Self-Report Wender-Reimherr Adult Attention Deficit Disorder Scale (SR-WRAADDS; Reimherr et al., 2007) includes 18 items that correspond to the nine DSM-IV-TR inattention symptoms and the nine DSM-IV-TR hyperactivity/impulsivity symptoms. Responses are provided using a 4-point Likert-type scale ranging from 0 (never or rarely) to 3 (very often). The inattention scale was internally consistent ($\alpha = .88$) in the current sample, as was the hyperactivity/impulsivity scale ($\alpha = .86$), and the total ADHD score ($\alpha = .92$).

Taxometric Data Analysis

Three nonredundant taxometric procedures were used to analyze the current data: mean above minus mean below a cut (MAMBAC; Meehl & Yonce, 1994), maximum eigenvalue (MAXEIG; Waller & Meehl, 1998), and latent mode (L-Mode; Waller & Meehl, 1998). The MAMBAC procedure requires two variables for analysis: an input and output indicator. The input indicator data are sorted along the *x*-axis and a series of cuts are made along this axis (50 in the current study). At each cut, the plotted *y*-value is the difference between the mean of the output values above the cut and the mean of those below the cut. If a taxon is present, the graph will have an inverse U-shape, with the highest point representing the taxon base rate. In contrast, when a dimensional construct is analyzed the graph is prototypically U-shaped. When there are more than two indicator variables, one variable may be used as the output indicator and the remaining variables summed to create the input variable. The procedure is then repeated with each variable serving as the output indicator. Because each indicator was a single item from the SR-WRAADDS, this summing method was used in the present study (Walters & Ruscio, 2009).

In MAXEIG (Waller & Meehl, 1998) the input indicator is sorted along the *x*-axis, but here the sample is divided into series of overlapping windows (25 windows with .90 overlap in the present study, Walters & Ruscio, 2010). The remaining variables are entered into a principle component analysis and the eigenvalue of the first principle component (i.e., the multivariate equivalent of the covariance) is graphed on the *y*-axis for each slice. A taxonic construct would be represented by an inverse U-shaped plot, with the peak eigenvalue corresponding to the window in which there are approximately equal numbers of members from the taxon and complement groups, which can then serve as an estimate of the taxon base rate. A dimensional construct will typically yield a flat, U-shaped, or irregular MAXEIG graph.

L-Mode factor analyzes all of the indicators and graphs the first principle factor. A taxonic construct produces a bimodal graph, whereas a unimodal graph indicates a dimensional construct.

Factors in addition to the construct's latent structure, such as the skew of the data or the associations among the indicators, can also influence the shape of taxometric graphs. To address this issue, simulated data sets that reproduce many of the features of the actual data while varying its latent structure (taxonic or dimensional) can be generated from the research data and then analyzed with MAMBAC, MAXEIG, and L-Mode (Ruscio et al, 2007). For each analysis, we generated 100 samples each of simulated taxonic and dimensional comparison data and then used comparison curve fit indices (CCFI) to determine goodness-of-fit between the research output and the simulated taxonic and dimensional graphs. CCFI values less than .45 indicate that the actual data are more similar

to the simulated dimensional data, and those greater than .55 support a taxonic latent structure. There is solid empirical evidence that this simulation method and the CCFI values it yields are capable of identifying the latent structure of a construct accurately. For example, in a Monte Carlo study using 100,000 data sets, the latent structure was correctly classified 99.9% of the time when the three taxometric procedures all yielded CCFIs less than .45 or greater than .55 (Ruscio et al., 2010). The taxometric analyses were conducted using Ruscio's (2011) program for R.

Results

The present study included analyses examining the latent structure of: (a) inattention problems using the nine SR-WRAADDS inattention items as indicators, (b) hyperactivity/impulsivity using the corresponding nine items from the SR-WRAADDS as indicators, and (c) ADHD using the total scores from the SR-WRAADDS inattention scale and the hyperactivity/impulsivity scales as the two indicators. Taxometric analyses require valid indicators that demonstrate at least 1.25 standard deviation units (SDU) of separation between presumptive taxon and complement groups (Meehl, 1995). In the present sample, 9% of the participants met the criteria for having an attention/hyperactivity disorder during their lifetime, so this base rate was used to compute indicator validity values.[1] Using this base rate estimate, the SR-WRAADDS items had strong indicator validity with average d's of 2.22 SDU (range 1.81–2.72) and 2.04 (range 1.10–2.76) for the nine inattention items, and the nine hyperactivity/impulsivity items, respectively.[2] The average d for the two ADHD scales was 3.26. Although only 52 of the participants in the sample met the criteria for ADHD, previous Monte Carlo studies (Ruscio & Marcus, 2007) have found that the CCFI method is effective for identifying low base-rate taxa.

When the nine inattention items were used as indicators, all three taxometric procedures yielded clearly dimensional results. The nine MAMBAC graphs exhibited a rising cusp on the right side of the graph, which is indicative of either a low base-rate taxon or positively skewed indicators. This ambiguity was clarified by the simulation data. The average of these nine curves was much more similar to the simulated dimensional data than to the simulated taxonic data with a CCFI of .278 (Figure 1, top panel). All nine MAXEIG graphs appeared flat or irregular and none of them were suggestive of a taxon. Again the average curve corresponded more closely to the simulated dimensional data (CCFI = .338; Figure 1, middle panel). Finally, the L-Mode graph was unimodal and considerably more similar to the simulated dimensional data than to the simulated taxonic data (CCFI = .287; Figure 1, bottom panel).

The results of the taxometric analyses of the nine hyperactivity/impulsivity were also consistent with a dimensional latent structure. Again, all nine MAMBAC curves had rising cusps and the average curve was more similar to the simulated dimensional data than to the taxonic data, with a CCFI of .307 (Figure 2, top panel). The MAXEIG curves were once

[1]Although 9% was used to compute the indicator validity values, the base rate estimates used to generate all of the simulated taxonic data reported in the text were based on the graphs generated by the MAMBAC and MAXEIG procedures, which were averaged to yield a base rate of .20 for inattention and .22 for hyperactivity/impulsivity (the base rates yielded by the L-Mode curves were not used because when the data are dimensional they tend to be around 50%). The rationale for allowing the procedures to provide base rate estimates was that a presumptive taxon may not be isomorphic with the diagnostic criteria for these disorders. When the base rate for generating taxonic comparison data was set at 9%, the results remained consistently dimensional throughout the analyses, with CCFIs ranging between .293 and .334 for inattention, .334 and .428 for hyperactivity/impulsivity, and CCFIs of .357 (MAMBAC) and .439 (MAXSLOPE) for ADHD.

[2]When the taxometric analyses of the hyperactivity/impulsivity symptoms were run dropping the one indicator with a validity indicator of 1.06 SDU (which is less than 1.25 SDU recommendation), the results remained dimensional, with CCFIs of .342 (MAMBAC), .398 (MAXEIG), and .444 (L-Mode). The decision to include all of the indicators for the primary analysis is consistent with a recent Monte Carlo study (Ruscio & Carney, 2011), which found that maximizing the number of indicators was more likely to yield accurate results than removing indicators because of subthreshold validity coefficients.

again flat and none had a clear peak. The MAXEIG CCFI was .371, indicating that the average curve generated by the actual data was more similar to the curve generated by the simulated dimensional data (Figure 2, middle panel). The L-Mode graph was unimodal and was more similar to the simulated dimensional data (CCFI = .346; Figure 2, bottom panel).

Because the analyses examining whether inattention and hyperactivity/impulsivity problems combine to yield a qualitatively distinct syndrome involved just two indicators, neither MAXEIG nor L-Mode were applicable. Instead we supplemented MAMBAC with the MAXSLOPE procedure (Ruscio & Walters, 2011), using the .28 base rate yielded by MAMBAC to generate simulated taxonic data. Both MAMBAC curves had rising cusps, but lacked a clear peak. The average curve was more similar to the simulated dimensional data (CCFI = .324; Figure 3, top panel). The MAXSLOPE graph did not contain the clear peak that would be consistent with a taxonic structure and was more similar to the simulated dimensional data (CCFI = .312; Figure 3, bottom panel).

Finally, because the full sample included both a clinical sample and a healthy control group, we repeated all of the analyses using just the 432 clinical subjects. This group had a lifetime prevalence of ADHD of 12%, which was used as the base rate estimate for the subsequent analyses. These analyses also yielded consistently dimensional results, with the exception of one ambiguous CCFI. Specifically, the CCFIs for the inattention symptoms were .313 (MAMBAC), .311 (MAXEIG), and .327 (L-Mode). The CCFIs for the hyperactivity/impulsivity symptoms were .351 (MAMBAC), .397 (MAXEIG), and .466 (L-Mode). Finally, for ADHD the CCFIs were .365 (MAMBAC) and .385 (MAXSLOPE).

Discussion

All of the analyses consistently supported a dimensional latent structure for inattention, hyperactivity/impulsivity, and the ADHD syndrome when ADHD symptoms were assessed by self-report in a sample of adults. These findings were consistent with the results of studies that used taxometric procedures (Frazier et al., 2007; Haslam et al., 2006; Marcus & Barry, 2011) and other statistical methods (e.g., Lubke et al., 2009; Neuman et al., 1999) to examine the latent structure of ADHD in children and adolescents. This exercise in “critical multiplism” (Cook, 1985) in which studies that have varied by age group, assessment methods, and statistical methods all yield converging results, strengthens the confidence that can be placed in the conclusion that ADHD and each of its component deficits are best understood as existing on a continuum. Thus, there is no clear boundary between those who meet the diagnostic criteria for ADHD and those with subclinical symptoms of inattention, hyperactivity/impulsivity, or both.

One possible explanation for the lower rate of adult ADHD compared to child and adolescent ADHD is that youth diagnosed with ADHD include those with genuine ADHD (i.e., taxon members) and those who because of immaturity or situational factors only appear to have ADHD. Those in the latter group may then outgrow their apparent ADHD symptoms, leaving mainly taxon members in the adult group. Our current findings that adult ADHD, like youth ADHD, is dimensional do not support this possibility. Instead, this apparent decline in prevalence is more likely because the diagnostic criteria for ADHD are more developmentally appropriate for children than for adults and because child diagnoses are largely based on parent and teacher reports, whereas adult diagnoses are mainly based on self-report (e.g., Barkley, 2006).

Although the relation between latent structure and etiology is complex, these dimensional findings do have implications for etiological models of ADHD. Dimensional conditions cannot arise from a single dichotomous causal factor (e.g., a single gene, an environmental

toxin that is either present or absent), and are most typically the result of the interaction of multiple etiological factors. For example, both Nigg and colleagues (2004) and Sonuga-Barke (2005) have proposed multiple pathway models of ADHD, and such models would be consistent with a dimensional latent structure. Similarly, based on the results of their meta-analysis, Nickolas and Burt (2010) concluded that both inattention and hyperactivity had a multilocus genetic basis involving additive, dominant, and nonshared environmental influences. Consistent with a dimensional latent structure, each of these influences could contribute incrementally to the severity of ADHD symptoms.

Given the controversies surrounding the diagnosis and treatment of ADHD, in many respects these dimensional findings are unfortunate. If ADHD had proven to be taxonic, then the question of whether ADHD is over-diagnosed could be reduced to a psychometric issue, with research devoted to developing the most valid measures and the most accurate cut-scores. However, if, as appears to be the case, ADHD exists on a continuum, the question of how much inattention or hyperactivity is pathological and merits diagnosis remains a pragmatic and possibly even a political issue. Moreover, the answer to this question will likely vary depending on the social context including local norms and values (e.g., Hinshaw et al., 2011). Similarly, if ADHD were taxonic, an argument could be made to provide stimulant medication to those in the taxon group and to deny such treatment to others (just as the standard of care is to provide antibiotics to those who test positive for a bacterial infection, but not those with viral infections). However, because the difference between ADHD and suboptimal inattention and hyperactivity is one of degree and not kind, there may not be an easy answer to the question of who should have access to these medications.

Acknowledgments

This work was supported, in part, by grants from the National Institute of Mental Health to Dr. Coccaro (RO1 MH46848, RO1 MH47495, RO1 MH60836, and RO1 MH66984).

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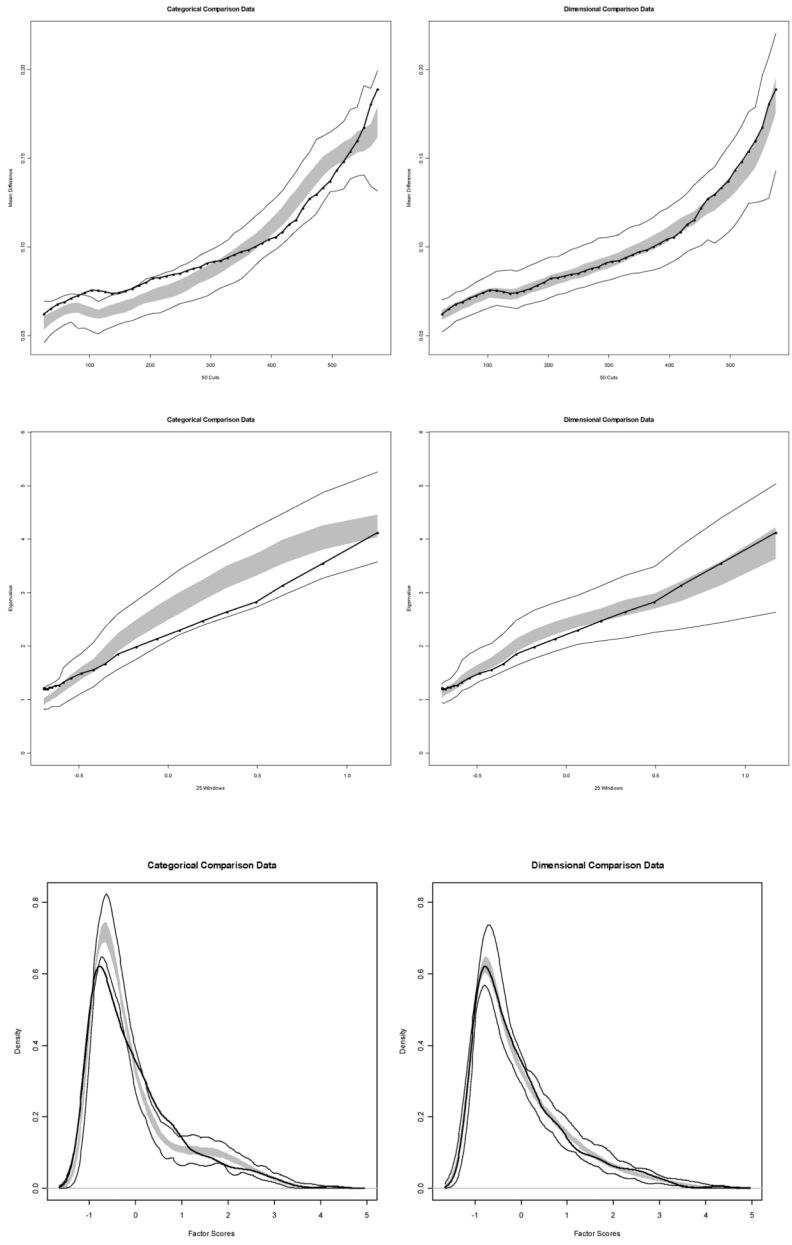

Figure 1.

Average MAMBAC, MAXEIG, and L-Mode curves for the research data along with taxonic and dimensional comparison data for the nine Self-Report Wender-Reimherr Adult Attention Deficit Disorder Scale inattention indicators. Dark lines on the curves represent the actual data, the outer lines show the minimum and maximum values generated by the 100 simulated data sets, with the grey band containing the middle 50% of data points from these simulated data.

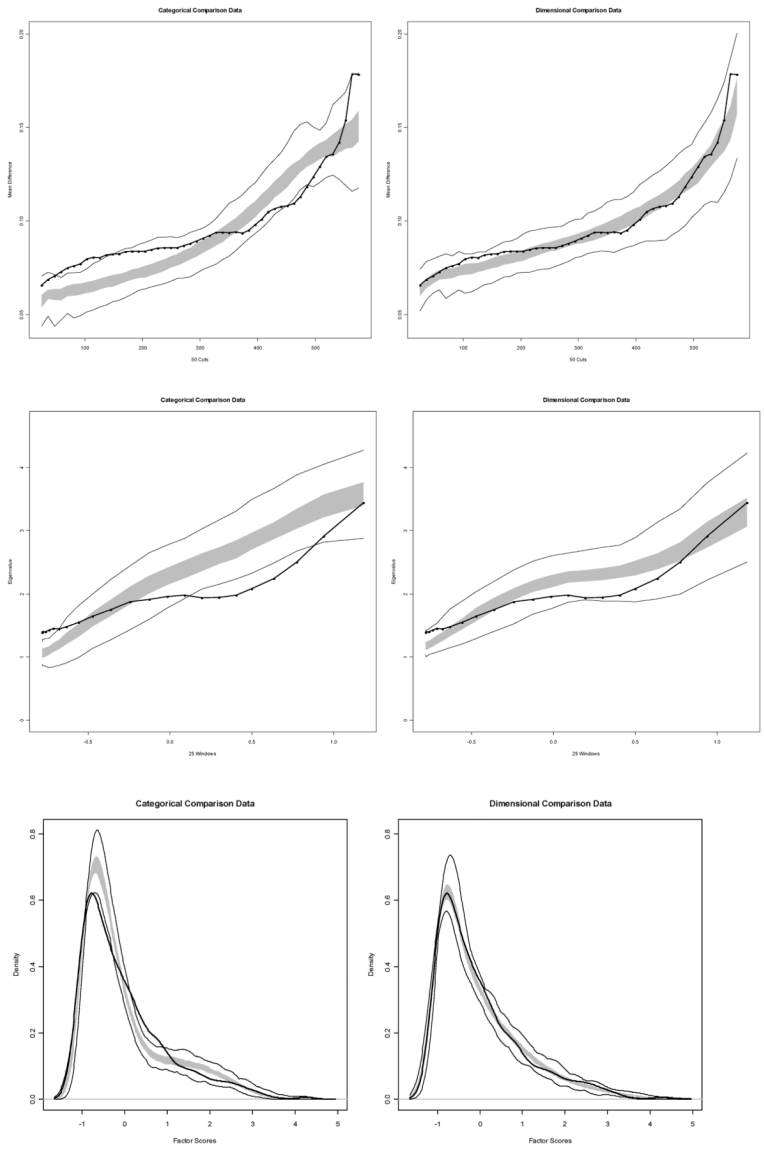

Figure 2.

Average MAMBAC, MAXEIG, and L-Mode curves for the research data along with taxonic and dimensional comparison data for the nine Self-Report Wender-Reimherr Adult Attention Deficit Disorder Scale hyperactivity/impulsivity indicators. Dark lines on the curves represent the actual data, the outer lines show the minimum and maximum values generated by the 100 simulated data sets, with the grey band containing the middle 50% of data points from these simulated data.

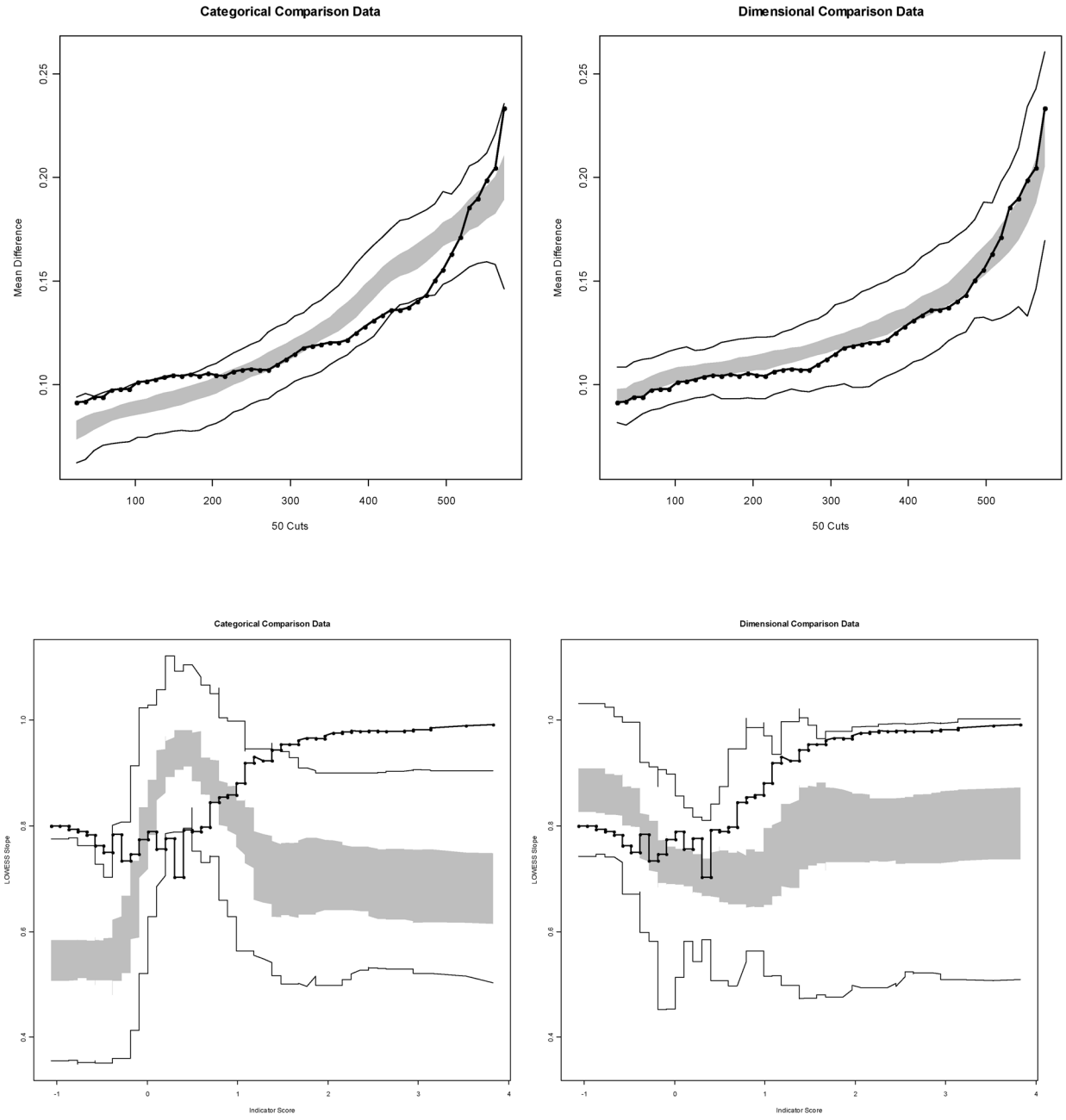

Figure 3.

Average MAMBAC and MAXSLOPE curves for the research data along with taxonic and dimensional comparison data for the inattention and hyperactivity/impulsivity subscales of the Self-Report Wender-Reimherr Adult Attention Deficit Disorder Scale. Dark lines on the curves represent the actual data, the outer lines show the minimum and maximum values generated by the 100 simulated data sets, with the grey band containing the middle 50% of data points from these simulated data.