Factorial Invariance in Hierarchical Factor Models of Mental Disorders in African American and
European American Youths

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Abstract

Background: There is converging and compelling evidence that mental disorders are more optimally conceptualized in a hierarchical framework that transcends traditional categorical boundaries. However, the majority of this evidence comes from studies that draw upon predominantly Caucasian populations. Whether the hierarchical conceptualization of mental disorders generalizes across racial-ethnic groups, including for African American (AA) youths, is unclear. This research is especially crucial in light of the observed racial-ethnic differences in the prevalence rates of several mental disorders. **Methods:** We tested multidimensional and bifactor models of 15 *DSM-5* diagnoses and psychiatric traits in two groups, including AA ($n=3,088$) and European American (EA) ($n=5,147$) youths aged 8-21 from the Philadelphia Neurodevelopmental Cohort (PNC). We also conducted multigroup confirmatory factor analyses to test for factorial invariance between the best fitting AA and EA multidimensional and bifactor models. **Results:** In the multidimensional model tests, a three-factor model, specifying internalizing, externalizing, and thought dimensions, emerged as the best fitting model for AAs and EAs. In the bifactor model tests, a three-factor model (i.e., internalizing, externalizing, and thought dimensions) that also specified a general factor emerged as the optimal for both AAs and EAs. The general factor accounted for a significant proportion of the covariation between the secondary factors and the individual disorders and traits. Furthermore, both models were factorially invariant, indicating that there was no significant difference in the factor structure of mental disorders between AAs and EAs in PNC. **Conclusions:** This study provides evidence that

the hierarchical factor structure of mental disorders may be racial-ethnically robust. This finding has implications for etiological and epidemiological studies focused on racial-ethnic subgroup comparisons, particularly with respect to identifying similarities and differences in prevalence rates or sociodemographic risk factors for mental disorders.

Keywords: cross-cultural differences, psychopathology, general factor, internalizing, externalizing

Abbreviations: AA: African American; EA: European American

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Several issues have been raised with regard to our current taxonomy of mental disorders as defined in the *Diagnostic and Statistical Manual of Mental Disorders, 5th Edition* (American Psychiatric Association, 2013). One issue is that discrete mental disorders tend to exhibit excessive co-occurrence with one another. For instance, studies featuring large community samples have shown that at least one-third of individuals diagnosed with a major depressive disorder were also diagnosed with an anxiety disorder, and around half of individuals diagnosed with any psychiatric disorder were likely to be diagnosed with another disorder (Beekman et al., 2000; Kessler, Chiu, Demler, & Walters, 2005). This calls into question whether mental disorders may be more optimally defined within a hierarchical framework that transcends the categorical boundaries of the *DSM-5* (Kotov et al., 2017; Lahey et al., 2017; O'Donovan & Owen, 2016).

Another issue is that there are well-documented (but poorly understood reasons for) racial-ethnic differences in the prevalence of mental disorders in the United States (Anderson & Mayes, 2010; Breslau et al., 2005). Previous studies that have examined racial-ethnic differences in the prevalence rates of mental disorders have largely focused on the role of socio-cultural factors, including (but not limited to) poverty, exposure to violence, racial discrimination and mental health stigma (Alang, 2016; Alegría et al., 2008; Liu, Mustanski, Dick, Bolland, & Kertes, 2017). However, few studies have considered whether prevalence rate differences

between racial-ethnic subgroups might also reflect differences in the way that mental disorders co-occur at the hierarchical level.

The importance of precisely clarifying the structure of mental disorders across racial-ethnic lines stems from its theoretical and clinical implications. The excessive comorbidity found in certain patient populations complicates our search for etiologic mechanisms (Newman, Moffitt, Caspi, & Silva, 1998; O'Donovan & Owen, 2016; Smoller et al., 2018). This slows progress towards developing better and more tailored intervention and treatments for mental disorders. Furthermore, African Americans (AA) make up the second largest racial-ethnic subgroup in the United States (13.4% of the U.S. population; U.S. Census Bureau, 2018). Yet, they are rarely the primary focus of epidemiological psychiatric studies (although see Breslau et al., 2005 and Harris, Edlund, & Larson, 2005 for a few notable exceptions). The burden of mental illness is not exclusive to one group, and there is currently a pressing need to generalize our models of mental illness to underrepresented populations such as AAs. The current study aims to address a critical gap in an emerging literature by systematically comparing the factor structure of mental disorders in a large sample of AA and EA youths.

Epidemiological studies have shown that the hierarchical factor model of mental disorders may be invariant to age and sex (Carragher et al., 2015; de Jonge et al., 2018; Eaton et al., 2012; Lahey et al., 2017; Patalay et al., 2015), as well as across reporters and over time (Lahey et al., 2017; Snyder, Young, & Hankin, 2017). However, there is inconclusive and limited evidence that this factor structure might also be invariant to racial-ethnic status (e.g.,

Eaton et al., 2013; Liu et al., 2017). For example, Eaton and colleagues (2013) examined adult data from the National Epidemiological Survey on Alcohol and Related Conditions (NESARC; $N=43,093$), which included EAs ($n=24,507$), AAs ($n=8,245$), Hispanic ($n=8,308$), Asian/Pacific Islanders ($n=1,332$) and American Indian/Alaska Natives ($n=701$). The researchers found that the mental disorders best fit a two-factor model (i.e., internalizing and externalizing), with no observed differences in the factor structure between racial-ethnic subgroups (Eaton et al., 2013). However, the researchers did not model a general factor, which is known to account for a significant proportion of the covariation among the disorders of the *DSM* across studies and populations (Kotov et al., 2017; Lahey et al., 2017; O'Donovan & Owen, 2016). Furthermore, NESARC featured an excessive amount of age heterogeneity, and only certain age groups (i.e., young adults) were oversampled (Grant & Dawson, 2006).

The current study compares factor models of *DSM* disorders and psychiatric traits between AAs and EAs in a population of youths ($N=8,235$) aged 8 to 21 from the Philadelphia Neurocognitive Cohort (PNC). We employed a multigroup confirmatory factor analysis (CFA) to test for factorial invariance between AAs and EAs. We also conducted follow up tests of criterion validity to establish that the racial-ethnically stratified latent factors are meaningful indicators of risk. Based on the findings reported by others (e.g., Eaton et al., 2013; Liu et al., 2017), we hypothesized factorial invariance to emerge with respect to our factor models for AAs and EAs in a younger population.

Method

Participants

Participants were from PNC, a population-based dataset of children, adolescents and young adults that consisted of assessments on mental disorders and medical history as well as assays involving neuroimaging, genetics, and neurocognition. The data was primarily collected from November 2009 to December 2011 from participants recruited in the greater Philadelphia, Pennsylvania area ranging from 8 to 21 years of age. Additional details of this study are detailed extensively elsewhere (e.g., Calkins et al., 2015 and Satterthwaite et al., 2016). In total, we analyzed data on 3,088 AA and 5,147 EA youths in PNC. Informed consent/assent was obtained from all PNC participants (Satterthwaite et al., 2016).

Diagnoses

The “GOASSESS” is a semi-structured, computerized clinical interview modified from the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) (Kaufman et al., 1997). The GOASSESS was administered to the caregivers or legal guardians (i.e., collaterals) among PNC participants aged 8 to 10, participants and collaterals who were ages 11 to 17, and the participants themselves if they were between the ages of 18 to 21. In order to minimize reporter inconsistency between the age groups, we prioritized data from collaterals for participants between the ages of 8 to 17 and self-report data for those older than 18. Trained interviewers who administered the GOASSESS assessed major depressive disorder (MDD), manic episodes (MAN), generalized anxiety disorder (GAD), social anxiety disorder (SOC), separation anxiety disorder (SEP), specific phobias (PHB), agoraphobia (AGR), anorexia (AN),

post-traumatic stress disorder (PTD), obsessive compulsive disorder (OCD), attention-deficit/hyperactivity disorder (ADD), oppositional defiant disorder (ODD), conduct disorder (CD), panic disorder (PAN), and psychosis (PSY). Diagnoses were coded based on having a sufficient number of symptoms, age of onset, duration, frequency, and evidence of distress or impairment according to *DSM-5* criteria, to the extent that data were available.

Analytic Plan

All analyses were conducted in Mplus 7.4 (Muthén & Muthén, 2015). We tested multidimensional and bifactor confirmatory factor analytic (CFA) models based on theory and empirical precedents. In the multidimensional models, we tested the model fit for: 1) a unidimensional model, where all disorders loaded onto a single latent factor, 2) a correlated two-factor model (i.e., internalizing vs. externalizing) (Eaton et al., 2013), 3) a correlated three-factor model (i.e., fear vs. distress vs. externalizing) (Krueger & Markon, 2006), 4) a correlated three factor model with a thought dimension (i.e., internalizing, externalizing, thought) (Caspi et al., 2014; Krueger et al., 2018), 5) a bifactor, two-factor model (Caspi et al., 2014; Laceulle, Vollebergh, & Ormel, 2015; Lahey et al., 2018), 6) a bifactor, three-factor model (i.e., distress, fear, externalizing) (Calkins et al., 2015), and 7) a bifactor, three-factor model with a thought dimension (i.e., internalizing, externalizing, thought). Based on recent critiques of the bifactor model, we also fit a bifactor “S-1” model (Eid, Geiser, Koch, & Heene, 2017) (see supplemental materials for figures, tables, and results). Traits were specified in the two-factor model accordingly: internalizing (PTD, MAN, MDD, AN, GAD, AGR, SEP, PAN, PHB, SOC, OCD)

and externalizing (ODD, CD, ADHD, PSY). Trait specifications in the three-factor model were: distress (PTD, MAN, MDD, AN, GAD), fear (AGR, SEP, PAN, PHB, SOC, OCD), and externalizing (ODD, CD, ADD and PSY). Traits were specified in the three-factor model accordingly: internalizing (PTD, MDD, AN, GAD, AGR, SEP, PAN, PHB, SOC), externalizing (ODD, CD, ADHD) and thought (OCD, MAN, PSY).

In the bifactor models, the same trait specifications and latent factors were estimated, but each disorder was also allowed to have a positive loading on a “general” factor (Reise, Morizot, & Hays, 2007). Here, the general and specific factors are also specified as being orthogonal to each other. Factor reliability indices were also computed (see Table S2). For all models, we specified a theta parameterization and the weighted least squares (WLSMV) estimator in the models with diagnoses as indicators. Optimal model fit was determined based on several goodness-of-fit indices, including comparative fit index (CFI), Tucker-Lewis index (TLI), and the root mean squared error of approximation (RMSEA). CFI and TLI values greater than .95 and RMSEA values less than .06 generally indicate “good” model fit (Hu & Bentler, 1999).

Once the optimal model for each racial-ethnic subgroup was selected, we then conducted a multigroup CFA on both the best fitting multidimensional model and bifactor model. In multigroup CFA, both racial-ethnic groups were entered simultaneously to test for the presence or absence of factorial invariance. First, we tested an *unconstrained* model, where factor loadings and item thresholds were freely estimated within groups. Next, we tested a *constrained* model, where factor loadings and item thresholds were constrained to be equal across groups. Factor

loadings represent (standardized) associations between each disorder and the latent factor. Item thresholds represent the change in latent factor value required for an item (i.e., a specific mental disorder) to move from one level (i.e., no diagnosis) to the next level (i.e., diagnosis). Tests of factorial invariance directly determine whether certain mental disorders have the same relation to the latent factor in one group as in another (e.g., is depression related to the internalizing latent factor the same way in AAs as in EAs?). If constraining both factor loadings and item thresholds to be equal does not significantly affect the model fit, invariance of the model is supported. A difference in a CFI value of less than .01 indicates no change in model fit after adding equality constraints over factor loadings and item thresholds (Cheung & Rensvold, 2002). In addition, we calculated Gamma Hat and McDonald's Noncentrality Index (NCI) to further evaluate change of model fit (Cheung & Rensvold, 2002; Rodriguez, Reise, & Haviland, 2016). A difference value of .001 and .02 was used as the cut-off for Gamma Hat and NCI, respectively. Sex was entered as a covariate in all our analyses, consistent with previous CFA studies using this dataset (e.g., Calkins et al., 2015).

Secondary Data Analysis

Secondary analyses were performed to examine whether the latent factors that we derived in our best fitting models were associated with neurocognitive outcomes, given the prior and consistent evidence that neurocognitive functioning is disrupted among individuals with psychopathology on a phenotypic level (Pont, Rhee, Corley, Hewitt, & Friedman, 2019). Partial correlations between the latent factor scores (from each of the best fitting multidimensional and

bifactor models) and neurocognitive functioning indices were tested separately for both AAs and EAs. Neurocognitive functioning was measured from Computerized Neurocognitive Battery (CNB) and the Wide Range Achievement Test (WRAT). Full details of the CNB and WRAT are provided in the supplemental materials (see Supplemental Methods). Sex and age were entered as covariates in the partial correlations. We did not conduct tests of invariance in our secondary analyses, given that AAs and EAs share can still share the same underlying hierarchical structure for psychopathology, but are likely to be impacted differently by environmental factors in their expression (Alang, 2016; Keyes, 2009; Meyer, 2003). Furthermore, the current investigation is primarily focused on addressing the question of whether the structure of psychopathology is invariant between AAs and EAs.

Results

Descriptive Statistics

Prevalence rates and sample characteristics are presented in Table 1. Group differences between AAs and EAs were observed for sex ($\chi^2=15.039$, $p=.001$, Cramer's $V=.060$), but not for age ($t(8228)=1.505$, $p=.132$, Cohen's $d=.035$). Group differences were also observed for ODD, CD, AGR, PHB, PTD and PSY, where AAs exhibited higher prevalence rates for these disorders than EAs (Table 1). Consistent with prior epidemiological studies of adult samples, group differences were observed for PAN, SEP, GAD and MDD, where AAs exhibited lower prevalence rates for these disorders than EAs (Breslau et al., 2005; Harris et al., 2005). Notably

however, the effect size of these differences was consistently small (i.e., Cramer's $V < .15$) for all disorders.

Within Group CFA

Model fit indices for multidimensional and bifactor models are presented in Table 2. From the multidimensional models, the three-factor model (specifying a thought dimension) demonstrated the best fit to the data for both AAs and EAs (AA: CFI=.961, TLI=.953, RMSEA=.020; EA: CFI=.961, TLI=.953, RMSEA=.017). As expected, all factor loadings of the disorder on the latent factors were consistently significant and positive for both AAs and EAs (see FigS1 and FigS2).

In our bifactor model tests, the bifactor two-factor model was a superior fit to the data (AA: CFI=.985, TLI=.979, RMSEA=.013; EA: CFI=.980, TLI=.972, RMSEA=.014). However, in line with our multidimensional three-factor model, the bifactor three-factor model with a thought dimension conveyed a similarly excellent fit to the data (AA: CFI=.975, TLI=.965, RMSEA=.017; EA: CFI=.976, TLI=.967, RMSEA=.015). For theoretical reasons, we compared the three-factor model with the bifactor three-factor factor model to aid our interpretations of the factor loadings. With respect to the latter model, all loadings onto the general factor were statistically significant for both groups (Fig1 and Fig2). However, trait loading patterns differed with respect to their loadings on to the secondary specific factors. Accounting for the general factor significantly attenuated trait loadings onto the secondary factors in both AAs and EAs. For instance, PTD (AA: loading=.102, sd=.062; EA: loading=-.046, sd=.063), AN (AA:

loading=.171, sd=.109; EA: loading=-.140, sd=.109) and PAN (AA: loading=-.227, sd=.170; EA: loading=.180, sd=.102) did not significantly load onto internalizing factor for either group. MAN (AA: loading=.129, sd=.194; EA: loading=.178, sd=.151), OCD (AA: loading=-.099, sd=.103; EA: loading=.078, sd=.064), and PSY (AA: loading=.370, sd=.285; EA: loading=.428, sd=.221) did not significantly load onto thought factor for either group. MDD significantly loaded onto internalizing factor for AAs but not EAs (AA: loading=.212, sd=.065; EA: loading=-.083, sd=.068). Finally, ADD significantly loaded onto externalizing factor for EAs but not AAs (AA: loading=.015, sd=.103; EA: loading=.230, sd=.075).

Multigroup CFA

First, we tested for factorial invariance for the three-factor multidimensional model with a thought dimension (i.e., internalizing, externalizing, and thought). EA and AA groups were entered in the same model simultaneously (see TableS1). In the *unconstrained* model, factor loadings and item thresholds were freely estimated in both racial-ethnic groups. The *unconstrained* model showed excellent model fit (CFI=.961, TLI=.953, RMSEA=.018, Gamma Hat=.996, McDonald's NCI=.984). In the *constrained* model, factor loadings and item thresholds were held constant across both groups. The *constrained* model also showed excellent model fit (CFI=.959, TLI=.954, RMSEA=.016, Gamma Hat=.995, McDonald's NCI=.983). Thus, results suggested factorial invariance of the three-factor multidimensional model with a thought dimension, as the difference in CFI, Gamma Hat, and McDonald's NCI was less than .01, .001, and .02 (Cheung & Rensvold, 2002).

Factorial invariance was then tested for the bifactor three-factor model with the thought dimension. EAs and AAs were entered in the same model simultaneously (see TableS1). The *unconstrained* model showed excellent model fit (CFI=.976, TLI=.966, RMSEA=.016, Gamma Hat=.997, McDonald's NCI=.990). The *constrained* model was also an excellent fit to the data (CFI=.972, TLI=.966, RMSEA=.016, Gamma Hat=.997, McDonald's NCI=.989). Thus, the bifactor three-factor model was factorially invariant between AAs and EAs.

Secondary Data Analysis

In the multidimensional model, AAs did not differ on their internalizing ($t(8233)=-1.157$, $p=.247$, $d=-.027$), externalizing ($t(8233)=-.960$, $p=.337$, $d=.021$) or thought factor scores ($t(8233)=-1.696$, $p=.090$, $d=.038$) relative to EAs (see TableS2). In comparison, in the bifactor models, AAs had lower general factor scores ($t(8233)=-5.508$, $p<.001$, $d=-.125$), but higher internalizing, externalizing and thought factor scores ($t(8233)=17.325$, $p<.001$, $d=.386$; $t(8233)=8.895$, $p<.001$, $d=.200$ and $t(8233)=5.454$, $p<.001$, $d=.125$, respectively), relative to their EA peers.

In the multidimensional model, internalizing, externalizing and thought factor scores were weakly or uncorrelated with measures of neurocognitive functioning for both AAs and EAs (see TableS4). In contrast, latent factor scores from the bifactor three-factor model demonstrated consistently negative correlations between the general, internalizing, externalizing and thought factor and the indices of neurocognitive functioning for both AAs and EAs, as expected (see TableS5).

Discussion

This study showed that a hierarchical factor structure of mental disorders is racial-ethnically invariant when compared between a large sample of AA and EA youths in the PNC dataset. The general factor accounted for a significant amount of the covariation between the mental disorders, over and above the contribution of the secondary specific dimensions. Our findings, along with the previous studies, have several important implications.

First, our findings provide additional evidence that mental disorders, including hierarchical conceptualizations of them, may be robust to “ethnic bias” (Eaton et al., 2013). In a recent meta-analysis, group differences in symptom counts and rates of diagnoses between EA and non-EA individuals were found to be small across several mental disorders (Causadias et al., 2018). These results were detected after having accounted for developmental period (i.e., childhood or adolescence), age, sex, years of education, and the type of measure of psychopathology (i.e., interview or questionnaire) (Causadias et al., 2018). Similarly, small effect sizes were detected with respect to racial-ethnic group differences in the prevalence rates of the mental disorders in the PNC sample. Overall, these findings converge on an emerging notion that mental disorders may be more *similar* than they are *different* across racial-ethnic groups with respect to their presentations and prevalence rates, although it is also important to note that small effect sizes do preclude the presence of meaningful group differences (Rosenthal, 1990).

Second, we replicated the evidence (Caspi et al., 2014; Lahey et al., 2017) that the general factor accounted for a large portion of the variance between several discrete disorders of the DSM in both racial-ethnic subgroups, over and above contributions from the secondary factors. In the bifactor three-factor model, for example, several of the disorders that had previously loaded onto the secondary factors no longer significantly loaded onto these dimensions after the general factor was taken into account. Moreover, our secondary analyses showed that the general and secondary factors from bifactor three-factor models were negatively correlated with neurocognitive functions, whereas latent factor scores from the multidimensional model (which did not feature a general factor) were weakly or not significantly correlated with these outcomes. Still, we note that the *precise* structure of mental disorders, especially with regard to the presence or absence of secondary factors like internalizing, externalizing, thought, fear and/distress dimensions, likely varies from population to population. Yet, the replicability of the general factor has been demonstrated across samples and now, across racial-ethnic subgroups. For instance, some researchers have conceptualized the general factor as reflecting negative affectivity or trait neuroticism, given its transdiagnostic associations (e.g., Caspi & Moffitt, 2018).

Third, hierarchical characterizations of psychopathology can potentially unravel more meaningful and precise risk and protective factors in a given population (Eaton et al., 2013; Morrison, Martinez, Hilton, & Li, 2018). That is, instead of focusing on discrete, categorical diagnoses that vary in prevalence across populations, latent factor scores can be examined

instead, reflecting each person's risk on a broader dimension of psychopathology that is statistically comparable across racial-ethnic groups (de Jonge et al., 2018; Eaton et al., 2013). However, establishing that the hierarchical factor structure of mental disorders is racially-ethnically invariant does not mean that the causes of psychopathology are the same between racial-ethnic subgroups. For example, racial discrimination (Liu et al., 2017), socialization and identity development (Smith, Reynolds, Fincham, & Beach, 2016) may contribute to racial-ethnic differences in the prevalence rates of psychopathology. Thorough tests of these hypotheses are beyond the scope of this investigation; future studies that focus on broad-band psychiatric constructs may be informative for understanding the differences that exist across racial-ethnic lines.

Some study limitations are worth noting. For example, the demographics of the PNC participants may not reflect the general population, including the prevalence rates of mental disorders that we observed. Furthermore, some disorders featured very low prevalence rates. This may be because of the abbreviated nature of the GOASSESS, which may have lacked sensitivity in detecting less common mental disorders. Future studies that feature clinically enriched samples and more rigorous clinical assessments might help to elucidate the contribution of the general and secondary factors on low prevalence mental disorders. An additional limitation is that we could not rule out the possibility of reporter effects or interviewer bias during the administration of the GOASSESS, both of which may have contributed to racial-ethnic differences in the prevalence rates of the disorders. While reporter effects have been

shown to play minimal role in population-based studies of the general factor (Snyder et al., 2017), rigorous studies examining the possibility interviewer bias are currently limited, unfortunately.

Conclusion

Our findings have implications for studies focused on identifying racial-ethnic differences in mental disorders. While there are important racial-ethnic differences in prevalence rates and risk factors for the individual disorders of the *DSM*, we showed that superordinate latent factors explained a significant portion of their covariation. This suggests that our mental disorders are perhaps more *similar* between racial-ethnic subgroups than they are *different*, especially when viewed from a broader, hierarchical perspective. This knowledge may be especially important as researchers continue their search for etiologic mechanisms that underlie our general liability for all mental disorders, which also may or may not be confined within the categorical boundaries of race-ethnicity.

Key points and relevance

- A hierarchical factor structure of mental disorders is well established in samples that are predominantly Caucasian.
- Few studies have examined whether this framework generalizes to other racial-ethnic subgroups, such as African Americans.
- Results indicated that a hierarchical factor structure, including one that featured a general factor and three subfactors (i.e., externalizing, internalizing, and thought) was statistically invariant between a large sample of African American and European American youths.
- Our ongoing search for etiological mechanisms may be aided by the knowledge that transdiagnostic latent factors of mental illness, which had been well established in predominantly Caucasian samples, also appear to generalize to other racial-ethnic subgroups.

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Ethical Considerations

This research meets the ethical guidelines as detailed in the Ethical Principles of Psychologists and Code of Conduct (American Psychological Association, 2010). Informed consent/assent was obtained from all PNC participants.

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Fig1. The bifactor three-factor model in AAs, with factor loadings represented for each pathway. All values are standardized, with standard errors in parentheses. Non-significant pathways are represented by dashed lines.

Fig2. The bifactor three-factor model in EAs, with factor loadings represented for each pathway. All values are standardized, with standard errors in parentheses. Non-significant pathways are represented by dashed lines.

Table1

Descriptive Statistics Comparing AAs and EAs in PNC

	Prevalence/Means (S.D./%)		χ^2 or t	df	p	Effect Size
	AA (N=3088)	EA (N=5147)				
Sex			14.260	1	<.001	$V=.060$
Male	1413 (45.758%)	2580 (50.126%)				
Female	1669 (54.048%)	2555 (49.641%)				
Unknown	6 (.194%)	12 (.002%)				
Age	13.860 (.067)	13.730 (.051)	1.505	8228	.132	$d=-.035$
Diagnosis						
ADD	46 (1.490%)	96 (1.865%)	1.392	1	.238	$V=.013$
ODD	595 (19.268%)	495 (9.617%)	155.692	1	<.001	$V=.137$
CD	266 (8.614%)	137 (2.662%)	145.643	1	<.001	$V=.133$
AGR	62 (2.008%)	63 (1.224%)	7.415	1	.006	$V=.030$
PHB	914 (29.598%)	1345 (26.132%)	11.479	1	.001	$V=.037$
PAN	7 (.2%)	44 (.8%)	12.375	1	<.001	$V=.039$
SOC	175 (5.667%)	335 (6.509%)	2.210	1	.137	$V=.016$
SEP	69 (2.234%)	184 (3.575%)	11.200	1	.001	$V=.037$

GAD	50 (1.619%)	172 (3.342%)	21.181	1	<.001	V=.051
AN	23 (.745%)	55 (1.069%)	1.825	1	.177	V=.015
MDD	251 (8.128%)	491 (9.540%)	4.518	1	.034	V=.023
MAN	12 (.389%)	17 (.330%)	.058	1	.810	V=.003
OCD	86 (2.785%)	161 (3.128%)	.667	1	.414	V=.009
PTD	451 (14.605%)	394 (7.655%)	100.489	1	<.001	V=.110
PSY	478 (15.480%)	450 (8.743%)	87.590	1	<.001	V=.103

Note: χ^2 : Chi-square statistic; *t*: t-statistic; *d*: Cohen's *d*; *V*: Cramer's *V*

Table2

Confirmatory Factor Analysis Model Fit Statistics

Models	CFI		TLI		RMSEA	
	AA	EA	AA	EA	AA	EA
Multidimensional Models						
One Factor	.912	.912	.898	.899	.029	.026
Two Factor	.954	.956	.946	.948	.021	.028
Three Factor	.957	.961	.947	.952	.021	.018
Three Factor with Thought	.961	.961	.953	.953	.020	.017
Bifactor Models						
Bifactor Two	.985	.980	.979	.972	.013	.014
Bifactor Three	.982	.978	.975	.969	.014	.014
Bifactor Three with Thought	.975	.976	.965	.967	.017	.015