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Is the continuity of externalizing psychopathology the same in adolescents and middle–aged adults? A test of the externalizing spectrum's developmental coherence

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Abstract

Externalizing psychopathology (EXT) is a framework for understanding diagnostic comorbidity and etiology of antisocial and substance-use behaviors. EXT indicates continuity in adulthood but the structure of adolescent EXT is less clear. This report examines whether adolescent EXT is trait-like, as has been found with adults, or categorical. We use tests of measurement invariance to determine how diagnostic indicators of EXT differ in adolescents compared to adults. The EXT measures employed were DSM-IIIR diagnoses of adult antisocial behavior, conduct disorder, and alcohol, marijuana, and drug dependence. Latent trait, latent class, and hybrid models were fit to two separate data sets: 2,769 seventeen-year-old adolescents and 2,619 adults from the Minnesota Twin Family Study. The best model in both samples was a single-trait LT model. Parameters from the adolescent and adult models were equivalent for all disorders except alcohol dependence. It appears that EXT in adolescence can be accurately represented by a single-trait model, and the measurement properties of EXT are similar during these time periods with the exception of alcohol dependence.

Externalizing Psychopathology (EXT) is a construct that aims to articulate mechanisms underlying the higher than chance rates of co-occurrence among a set of common psychological disorders (e.g. conduct disorder; antisocial personality disorder; substance use disorders), personality traits (e.g. impulsivity; aggression), and problem behaviors (e.g. risky sexual behavior, criminality, precocious substance misuse). Support for the EXT construct comes from evidence that measures of EXT correlate cross-sectionally among adolescents (e.g., Achenbach & Edelbrock, 1984; Disney et al., 1999; Armstrong & Costello, 2002) and adults (e.g., Kendler et al., 1997; Krueger et al., 2002; Krueger et al., 2005) and longitudinally within individuals (e.g., Lynskey & Fergusson, 1995; McGue et al., 2001; Elkins et al., 2006; Hicks et al., 2007). Based on large twin and family studies, measures of EXT can be usefully modeled as a single continuous dimension of vulnerability that is largely inherited (Slutske et al., 1998; Young et al., 2000; McGue et al., 2001; Krueger et al., 2002; Kendler et al., 2003; Hicks et al., 2004; Bornovalova et al., 2010). This model of EXT accommodates the diverse manifestations of EXT-related indicators (substance use, criminality, etc.) across development as the transaction of (A) an inherited liability of EXT risk with (B) pathogenic characteristics of the environment (opportunity to use substances, deviant peers, poor parental monitoring).

The externalizing spectrum (EXT) presents an alternative conceptualization of traditional psychiatric phenotypes. The DSM-IV's classification system is categorical, and the limitations of this system in the face of comorbidity have been addressed elsewhere (Krueger et al., 2005; Kendler et al. 2003; Meehl, 2001). Indeed, a movement toward continuous (versus categorical) phenotypes designed to accommodate phenomena such as comorbidity has now been observed in a variety of areas of mental health research. For example, the U.S. National Institute of Mental Health is working to identify Research

Domain Criteria (RDoC) as phenotypic alternatives to traditional DSM categories, and these targets are explicitly conceptualized as continuous psychobiological dimensions of variation that cut across traditional categories (Sanislow et al., 2010). In addition, DSM-5 is likely to contain continuous cross-cutting constructs to facilitate measurement-based intervention (Helzer et al., 2008). Such changes have potentially notable impacts on clinical care, research design, and policy initiatives. The planned merger of the National Institute of Alcohol Abuse and Alcoholism (NIAAA) with the National Institute of Drug Abuse (NIDA) is just one example of a notable change in policy and funding mechanisms in substance use disorder research, due in part to recognition of comorbidity between the disorders (National Institute of Health, 2010; p. 9). Models of comorbidity, such as those tested in the present report, will become increasingly relevant and informative as nosological systems and funding mechanisms undergo these types of changes. In addition, it is important to understand the applicability and comparability of these models across the life span, including as they may apply to adults who have largely passed through the age of risk for developing these disorders and to adolescents in the period of highest risk for their development.

As a hierarchical, organizing construct, EXT may also help to unify findings from descriptive psychopathology and epidemiology with other fields of research, such as psychiatric genetics or clinical neuroscience. Conceptualizing and measuring EXT as a continuous phenotypic trait is advantageous for genetic research for a variety reasons. First, latent traits, relative to discrete diagnostic entities, offer increased statistical power, for example, to detect genetic/environmental associations or to make predictions (Almasy and Blangero, 2000; Grove, 1991). Second, external correlates may be relevant only for shared etiological processes, as opposed to specific etiology. For example, Dick et al et al. (2007) showed that the *GABRA2* and *CHRM2* genes may promote a general vulnerability towards developing EXT disorders rather than any specific EXT disorder. Also, the P300 amplitude reduction, a neuroelectric phenomenon measured in the event-related potential, is associated with familial risk for a variety of specific EXT indicators (Carlson et al., 1999; Iacono, 2002). The general EXT vulnerability appears to mediate the association between P300 amplitude reduction and specific EXT disorders and behaviors (Patrick et al., 2006), which suggests that the P300 may represent an objectively-assessed neurobiological indicator of latent risk for EXT generally (i.e., the shared etiology), and not for any specific disorder. Findings such as these highlight the importance of elucidating the properties of adolescent EXT. When measured premorbidly (prior to onset of substance use and delinquent behavior), EXT could be a potentially advantageous target phenotype for clinical intervention and translational research.

The first goal of this report is to model the structure of EXT using a community representative sample of 17-year-old adolescents. Although adolescence is a critical developmental period for indicators of EXT (e.g., onset of substance use), most of the research that has contributed to our understanding of EXT has examined adult samples. For example, an important foundation of the continuous trait model of EXT in adults is that the covariance among common EXT indicators, such as adult antisocial behavior (AB; i.e., antisocial personality disorder without the requirement of pre-existing conduct disorder), conduct disorder (CD), alcohol dependence (AD), marijuana dependence (MD), and drug dependence (DD), is better accounted for by a continuous trait rather than by categorical models of comorbidity (Krueger et al., 2005; Markon & Krueger, 2005; Grove & Vrieze, 2010). To our knowledge, no study to date has examined the “dimensional or categorical” nature of adolescent EXT. The structure of adolescent EXT may shed light on the developmental pathways that give rise to adult manifestations of externalizing behaviors. The few studies that have modeled adolescent EXT comorbidity have assumed, at the outset, that EXT is composed of one or more continuous dimensions and then compared the fit of

models varying solely in the number of dimensions (Lahey et al. 2008; Farmer et al., 2009), without examining the possibility that a class structure might characterize adolescent EXT. Others have fit latent class models or latent trait models to a narrow subset of EXT indicators (e.g. symptoms of conduct disorder), but did not compared the relative fit of these two types of models (Nock et al., 2006; Odgers et al., 2007; Gelhorn et al., 2009). One study compared the relative fit of categorical and continuous latent variable models, and presented evidence for better fit for a continuous model, but the indicators were checklist items, as opposed to formal psychiatric diagnoses (Walton, Ormel, & Krueger, 2011). Thus, this report aims to examine whether comorbidity among adolescent EXT disorders is better accounted for by a uni-dimensional structure, as was found for adults by Krueger et al. (2005), or by alternative (e.g. categorical) models. This was accomplished by comparing the relative fit of categorical and continuous latent variable models, including latent trait models (also known as item response theory; Embretson & Reise, 2000), latent class models (Heinen, 1996), and factor mixture models (which contain aspects of both latent trait and latent class models (Muthen & Shedden, 1999; Muthen, 2006; Muthen & Asparouhov, 2006). For comparability with previous research, including Krueger et al. (2005) who modeled the very same parent sample considered here, we consider the same putative measures of EXT: AB, CD, AD, MD, and DD.

The second goal of this report is to examine differences in how indicators of EXT (i.e., the diagnoses) function in the adolescent sample compared to the adult sample. While measures of EXT show a developmental continuity (Lynskey & Fergusson, 1995; McGue et al., 2001; Elkins et al., 2006; Hicks et al., 2007), they may not provide the same information for adolescents as adults. For example, precocious adolescent substance use is a risk factor for later substance dependence, and conduct disorder is a risk factor and diagnostic necessity for adulthood antisocial personality disorder. However, there are many reasons to suspect that adolescent EXT is not simply a “younger” version of adult EXT; we list three. First, for example, some indicators of antisocial personality disorder were designed to capture psychosocial impairment specific to adulthood (e.g. quitting a job irresponsibly, a symptom of antisocial personality disorder, has clear implications for adults and less so for adolescents). Second, some EXT-related behaviors show transient increases in frequency and severity during adolescence (e.g., delinquency; Moffitt, 1993). Third, childhood and adolescent-onset EXT behaviors are associated with an elevated genetic risk for EXT (e.g., particularly early onset of conduct disorder is associated with greater genetic risk (Taylor et al., 2000)). Similarly, “EXT disorders” with a later, adult onset may be associated with pathogenic processes that are unrelated to EXT (e.g., a subtype of alcoholics has been characterized by predominate anxiety/depression, relatively late age of onset, and less genetic risk for alcoholism (Cloninger, 1987; Windle & Scheidt, 2004)).

Measurement differences between adolescents and adults would suggest that the manifestations of an underlying EXT liability are developmentally or generationally sensitive, perhaps due to pathogenic processes (i.e. environmental, cultural, and/or biological risk factors) that emerge or dissipate across development or between generations. Such differences may also be the target of future research to help identify the pathogenic processes. To test for any such differences we used latent trait analysis, combined with tests of measurement invariance, to determine whether AB, CD, AD, MD, and DD function differently between a sample of adolescent 17-year-olds and their parents.

Method

Participants

Data for this study comes from the Minnesota Twin Family Study (MTFS), an epidemiological and longitudinal study of twins born in the state of Minnesota and their

parents. The recruitment procedures and exclusion criteria have been described elsewhere (Iacono & McGue, 2002). Twins were identified by public birth records and then their parents and the twins were contacted by phone or mail and invited to participate. Exclusion criteria were based on feasibility of completing a thorough day long assessment (e.g., living more than a day's drive away or a mental or physical handicap in either twin excluded potential participants). The evaluation included psychiatric history, psychophysiological assessment, substance use habits, academic achievement, cognitive function, and personality. Every three years, participants were invited back to participate in follow-up assessments. When unable to visit in person, participants underwent parts of the assessment (e.g., the diagnostic assessment) by phone. Participants received modest honoraria for their assessments and written assent or consent was obtained from all participants, including the parents of minor children.

Measures—Participants were assessed for lifetime diagnostic history based on the Diagnostic and Statistical Manual 3rd Edition-Revised (DSM-IIIR; American Psychiatric Association, 1997) criteria during an in-person interview with a trained interviewer using modified versions of commonly used semi-structured interviews. Trained graduate students and research assistants reviewed each case during a diagnostic case conference to verify symptom presence or absence.

Participants under the age of 18 were assessed for lifetime presence of CD, AD, MD, and DD using the Diagnostic Interview for Children and Adolescents (*DICA-R*; Reich & Welner, 1988). We also interviewed a biological parent, usually the mother, about the mental health of their offspring. We used a "best-estimate" approach (Leckman et al., 1982) in which a symptom is considered present when either the child or parent endorsed the symptom. Participants over the age of 18 were assessed for CD and AB using a modified version of the SCID II (Spitzer et al., 1987) and for AD, MD, and DD using a modified version of the expanded Substance Abuse Module (SAM; Robins et al, 1987) of the Composite International Diagnostic Interview (CIDI; Robins et al., 1988). For purposes of this report, participants were coded 1 for a positive lifetime history of a disorder and 0 for a negative lifetime history of a disorder. DD positive status was determined by the presence of any illicit drug dependence disorder excluding MD (i.e. cocaine, heroin, amphetamines, and prescription medication). AB was assessed as the presence, since age 15, of four or more of the 10 DSM-IIIR Criterion C symptoms of antisocial personality disorder, without the prerequisite diagnosis of Conduct Disorder.

Diagnostic Status—Data were available for 2769 twins (52% female). Lifetime diagnostic status was determined from diagnostic data up until about age 17–18 years-old. 192 adolescents (8%) were missing the age 17 assessment but had participated in the age 11 or age 14 assessment. We were in the fortunate position of having some prior (albeit imperfect) information about these participants' lifetime diagnostic status at age 17, because they had already been assessed at age 11 and 14. Thus, instead of ignoring this additional information, we used it, and derived a lifetime diagnostic status from the earlier assessments for these individuals, thus optimizing both the information yield and the sample size. To insure that this decision did not bias our results, we re-analyzed the data after excluding these adolescents with missing age-17 data and found equivalent results. Average age in the adolescent sample was 17.54 (SD = 1.32). The youngest adolescent was 10 and the oldest 20. Biological parents were assessed at intake (N = 2619; 53% female), and thus there was no effect of attrition. The parent's average age at time of their diagnostic assessment was 45.17 (SD = 5.5). The youngest parent was 28 and the oldest 71.

Disorder base rates in the adolescent sample were .05, .13, .08, .06, and .01 for AB, CD, AD, MD, and DD. Corresponding rates in the parent sample were .06, .10, .22, .06, and .01.

The rate of alcohol dependence in the parents, while high relative to that of the adolescents, is consistent with previous epidemiological findings in individuals of the same age and generation as the parents in the present sample (Anthony et al., 1994; see Holdcraft & Iacono, 2004, for a discussion of these rates). Nine per cent of adolescents were missing measures of AB. AB by definition does not include behavior prior to age 15, and thus cannot be derived from age-14 or age-11 assessments and so were set as missing in all present analyses. Rates of CD, AD, MD, and DD diagnoses did not significantly differ between those missing and not missing an AB diagnosis. In the parents, one subject was missing AB; four subjects were missing CD; four were missing AD; fifteen were missing MD; and one was missing DD.

Model Comparison Analyses

LT, LC, and factor mixture models were fit to the adolescent diagnostic data and compared for fit, similar to the method reported in Krueger et al. (2005). In a separate analysis, the same kinds of models were fit to the parent diagnostic data and compared for fit, because Krueger et al. (2005) did not consider factor mixture models in their analyses. We only used data from the biological parents of the children (i.e., we excluded diagnostic data from step-parents). This was necessary to keep within-family relationships (which had to be accounted for to obtain accurate between-group results) consistent across families.

Latent Trait (LT) models posit continuous latent variables (i.e., factors) linked to categorical manifest variables (i.e., disorders). There are no classes in LT analysis, and everyone in the population is assumed to be arrayed along a continuous latent trait, or factor. Latent class analysis (LC; Lazarsfeld & Henry, 1968), on the other hand, assumes that the population is composed of discrete classes and that covariation of manifest variables arises solely from the admixture of classes (i.e., variation within classes is entirely random). A third type of model, factor mixture models, has not been utilized in studies that examine the dimensionality of EXT diagnoses.

Factor mixture models can be understood as a hybrid model containing features of both latent trait models and latent class models. Factor mixture modeling posits latent classes but, unlike LC analysis, also allows for variability within classes, further positing that within-class variability can be modeled using a LT model. For example, a two-class, single-factor, factor mixture model could capture a discrete class of externalizers with manifestations of serious antisocial behaviors and problematic substance use which are often but not always comorbid, the extent of comorbidity directly estimated by a LT model with a single dimension within the externalizing class. The second class may represent non-externalizers, who infrequently engage in antisociality or experiment with substances and rarely meet diagnostic criteria. That is, there is also a single dimension within this class, to account for low-severity externalizing-type behaviors such as drinking far too heavily once or twice but otherwise having no notable problems with alcohol. The factor mixture model is designed to identify distinct classes such as these and model a latent factor within each class, to capture intra-group variability in comorbidity. An assumption of LC analysis is that once class membership is accounted for, the manifest variables are rendered independent (Heinen, 1996). In other words, LC analysis may provide poor fit to the data if there are intra-group individual differences, or if there exists only one class. Evidence that the factor mixture model is superior to the factor model in accounting for the structure of EXT would indicate that class structure is a useful way to characterize externalizing disorders, but that significant intraclass variability remains after class membership is estimated. Evidence that a class model fits best, whether by LC analysis or factor mixture modeling, may suggest that discrete disease mechanisms exist, and these discrete mechanisms give rise to observed comorbidity. This result would be in contrast to a single-trait LT model (e.g., Krueger et al.,

2005), where pathological behavior is understood to be continuously distributed in the entire population.

For convenience, short descriptions of the models are provided in Table 1. All models were estimated with a maximum likelihood estimator in Mplus version 6 (Muthen & Muthen, 1995–2010) with full information maximum likelihood estimation to deal with incidental missing values. These sets of models are non-nested but can be compared using likelihood statistics that that account for model complexity. Two statistics have become widely used for model selection: the Akaike Information Criterion (AIC; Akaike, 1974) and the Bayesian Information Criterion (BIC; Schwarz, 1978). In general, BIC involves a higher penalty for model complexity when compared with AIC. The two statistics derive from widely different considerations regarding the pursuit of a model most likely to be the population model (Kuha, 2004) and often can disagree. Reconciling disagreement between AIC and BIC is impossible (Yang, 2005) but agreement between the statistics provides increasing support for a chosen model, if only because it broadens the available statistical assumption set.

After the two best models are selected (one each in the parents and adolescents) they can be compared to one another for equivalence. This comparison is conducted through tests of measurement invariance, described next.

Analysis of Measurement Invariance

Testing for measurement invariance involves testing whether model parameters are equivalent across groups. Here there are two groups: adolescents and parents. Strong measurement invariance was determined via a two-group (adolescents and parents) simultaneous LT analysis. Strong measurement invariance exists when model parameters (such as loadings and thresholds of the LT model) are equivalent across groups (Widaman et al., 2010; Meredith, 1993). Loadings in the LT model are regression slopes in a regression of the diagnosis onto the latent trait. Thresholds refer to the assumption in LT models that a binary item is assumed to be, in actuality, normally distributed. It is equivalent to the notion of threshold in tetrachoric and polychoric correlations, and represents a superficial difference between LT and item response theory models (Takane & de Leeuw, 1987).

Parameter estimates and tests of measurement invariance were conducted using the MLR estimator in Mplus and a likelihood ratio test (because in this case models were nested). BIC was also considered for these tests, and with our sample size and model comparisons was equivalent to a likelihood ratio test with significance threshold of $p = .003$, far below conventional levels of significance. AIC was not considered for these tests, because it was equivalent to a likelihood ratio test with significance threshold of $p = .16$, above conventional levels of significance. The BIC thus simply represents a more stringent likelihood ratio test when models are nested, at least for our models and sample size. *p*-value comparisons such as these are uninformative when models are not nested, and one must use AIC and BIC on the basis of their statistical properties (e.g., as described in Kuha, 2004). A test of measurement invariance was computed for each parameter by calculating a likelihood ratio test between the full model (with all loadings and thresholds estimated) and a model constrained to have a single between-group parameter fixed to be equal. For example, to test for invariance in AB's loading, the loading parameter for AB was constrained to be equal between the adolescent and adult models, with all other parameters freely estimated. Since the parent and adolescent samples are correlated (by both heredity and shared environment), the standard test of measurement invariance is inaccurate, as it assumes the two groups are independent. To correct for non-independence we used the cluster option in Mplus, using family as the clustering variable. This option adjusts standard errors within each group accounting for groupwise correlation.

Results

The tetrachoric correlation matrix, for both the youths and parents, are presented in Table 2. All correlations are highly significant. Within adolescents the correlations range from .6 to .8. In parents they range from .44 to .81. While parent correlations are more variable (especially for DD), both patterns suggest the possibility of a well-fitting one factor model. Correlations in the adolescent sample are generally higher than in the parent sample, at rates ranging from .1 to .3, suggesting higher rates of comorbidity among the adolescents. Note, however, that the larger discrepancies are present among disorders with lower base rates, and the resulting estimates and comparisons are less stable. The full diagnostic contingency table is displayed in Table 3, giving the rates of comorbidity for each combination of disorder and diagnostic status. Rates of disorder, and disorder combinations, are relatively similar between the adolescents and adults, with the exception that parents have higher rates of an AD-only diagnosis and lower rates of a CD-only diagnosis. Our externalizing sample (persons who met criteria for at least one externalizing disorder) comprised 1243 individuals, representing 24.2% of our total of 5128 participants. Fully 19.4% of adolescents and 28.9% of parents had at least one externalizing disorder. For those with an externalizing diagnosis, comorbidity was common, with 38.7% of adolescents and 32.4% of adults having two or more disorders.

To help determine the appropriate number of factors for the factor models, scree plots and parallel analyses were conducted on the diagnostic data. The five eigenvalues of the adolescent correlation matrix were 2.27, .88, .69, .60, and .56. Eigenvalues for the parent correlation matrix were 1.97, .89, .78, .71, and .65. Parallel analysis of the tetrachoric correlation matrices, carried out with the *R* package *random.polychor.pa*, also suggested single factor solutions for both the adolescents and parents (not shown for lack of space). In addition, likelihood ratio tests favored one-trait versus two-trait models in both the adolescent and parent samples. LT models, LC models, and factor mixture models were then fit to the adolescent data and compared for fit. AIC and BIC for the adolescent data are presented in Table 4. The single trait model was the best fitting model, as judged both by the AIC and the BIC. The factor mixture model was not better than the second-best model according to AIC and BIC. The agreement between the AIC and the BIC provides consistent evidence for the one factor model over the other models under consideration. Model fitting in the parent sample proceeded similarly and we found the best fitting parent model to be a single latent trait as well, replicating the results of Krueger et al. (2005). The factor mixture model was the second-best fitting model in the parents.

To determine if our results would also be obtained using symptom counts as diagnostic variables (as opposed to binary diagnoses), we fit multiple threshold models. These models are the same as the models fit the to the binary diagnoses but contain additional threshold parameters to accommodate the additional scale points in a symptom count variable (e.g., modeling a count of 0,1,2 requires two thresholds to distinguish the three scale points, whereas modeling 0,1 requires only a single threshold). The rank ordering of the fit for these models was identical to that reported in table 4 for binary diagnoses, albeit the two class, one trait model did not converge in spite of implementing thousands of random starts. This is not unexpected, given the computational complexity involved in fitting this model, and similar issues emerged with the closely related three class, one trait model fit to binary diagnoses; see Table 4. These findings provide reassurance that the latent trait model is the better fitting model, regardless of whether symptom counts or diagnoses are used as the manifest indicator variables.

We conducted tests of measurement invariance between the adolescent and parent single-trait models to better understand the structure among the EXT indicators during adolescence

and how it might differ from the structure for adults. Table 5 gives parameter estimates for the single factor model in both groups, as well as the likelihood ratio test of measurement invariance. Non-invariance was significant for AD in both loading and threshold parameters. AD loads less highly onto externalizing in the parent than adolescent sample, and thus offers less information about an adult's amount of externalizing relative to an adolescent's. Adolescents are also less likely to be diagnosed with AD for the same level of EXT, due to the higher threshold for adolescents. AB, CD, MD, and DD each give similar estimates of EXT in children and adults; they show no group differences. Based on this pattern of results, the loading and threshold parameters for AD was re-estimated and made free to vary in the adolescent and parent sample; the parameters for CD, MD, DD, and AB were re-estimated as fixed to be equal between parents and adolescents. Model parameters for this final and full model are listed in Table 6. This model fit better than the same model with all parameters allowed to freely vary across groups (likelihood ratio was 24.35 on 8 degrees of freedom, $p = .002$). Results from the BIC were equivalent to those from the likelihood ratio test; AIC was not considered for reasons described above in the methods. By squaring the reported standardized loadings, one can compute the variance in the observed diagnosis accounted for by the latent trait. On average, the common factor accounted for 69% of variance in the diagnoses in the youths, and 64% in the parents. In the combined sample, the factor accounted for 77% of variance in AB, 48% in CD, 69% in MD, and 79% in DD. The factor accounted for 72% of variance in AD in the adolescents and .46 in the parents.

The final model has several interesting characteristics. First, a trait elevation of externalizing is associated with meeting criteria for AB, MD, DD, and Adolescent AD. Second, CD and parental AD only moderately loaded on the externalizing trait. Their etiology appears to be more influenced by non-externalizing-related factors than AB, MD, DD, and Adolescent AD. AB, MD, and DD are associated with higher levels of externalizing, consistent with the generally greater social deviance of these behaviors relative to CD and AD.

Discussion

The present study examined the structure of adolescent EXT by modeling the covariance among AB, CD, AD, MD, and DD in a large representative sample of 2769 17-year-old adolescents. This is the first study, to our knowledge, that examined "dimensional vs. categorical" frameworks of adolescent EXT diagnoses. Diagnostic data were collected as part of a comprehensive assessment by trained research assistants using common structured interviews. We fit to the data a single factor model; 2, 3, 4, and 5 class models; and a 2-class, 1-factor, factor mixture model. We found that the single factor model provided the best fit according to the AIC and BIC model fit statistics. Agreement of AIC and BIC gives broad evidence that the single-factor model describes comorbidity between these disorders in a useful and coherent manner relative to other models under consideration (Kuha, 2004). The implication that EXT is more parsimoniously accounted for by a single factor model than by class models successfully replicates and extends to adolescents the work of others who examined adult populations (e.g., Krueger et al., 2005; Markon & Krueger, 2006). In addition, although we did not find that it provided the best fit to the data, this is the first report to our knowledge to examine the structure of EXT diagnoses using factor mixture modeling.

Measurement invariance across repeated observations is a prerequisite for further longitudinal modeling of repeated observations (e.g., growth curves, panel designs, Markov simplexes, latent change models; McArdle, 2009). Findings of measurement invariance across time gives strong evidence that the same construct (e.g., EXT) is being consistently measured at each time point. That is, it helps ensure that any changes from time one to time two are changes in the same construct, rather than an artifact of accidentally measuring

entirely different constructs at time one versus time two (Widaman et al., 2010). Our results reveal marked similarity between the adolescent and adult latent trait models. Strong measurement invariance (Meredith, 1993; Widaman et al., 2010) held for AB, CD, MD, and DD. If replicated in a longitudinal dataset, the existence of measurement invariance of EXT across adolescent and adult development would allow for further growth modeling of EXT from the late teen years to middle adulthood.

Measurement invariance did not, however, hold for alcohol dependence (AD). Adults were more likely to be diagnosed with AD than adolescents at comparable levels of EXT. In addition, AD was a better discriminative index of EXT for adolescents relative to adults. In other words, a sample of adults selected for a history of meeting criteria for AD would contain a larger proportion of non-EXT cases than a sample of adolescents selected for a history of meeting criteria for AD. One possibility is that the parents have had more opportunity (e.g., years) than adolescents at comparable levels of EXT to develop AD. This could account for the increased frequency of AD in adults relative to adolescents as well as a decreased correlation between AD and the other disorders evaluated in this report. If new cases of AD regularly arise after the late teens (e.g., in someone's twenties or thirties), but new cases of AB, MD, or DD do not, then one might predict attenuation of the correlation between AD and these other disorders. This would result in a lower loading for AD in the parents relative to the adolescents. Although biased reporting cannot be ruled out, our findings are consistent with this notion, and that etiological factors for AD emerge in adulthood and are unrelated to a general EXT liability.

Along these lines, research has found adult-onset AD to be less associated with an inherited risk for EXT (i.e., adult-onset AD is associated with an anxious-depressed personality style rather than novelty-seeking and impulsivity, and a lower familial risk for alcoholism, etc) than adolescent-onset of AD (e.g. Cloninger, 1987; Windle & Scheidt, 2004). Perhaps, among a sample of adults selected for a history of meeting criteria for AD, the AD diagnosis becomes saturated with anxious-depressed adults. Diagnostic information, such as age of onset and course of diagnosis might have helped test this hypothesis but was unavailable for the parent sample. The possibility that risk factors for AD might emerge during adulthood has implications for the clinical assessment of EXT. For example, the optimal measurement of EXT via AD, or other alcohol use measures, might benefit from developmentally appropriate items or weightings.

It is important to point out that cohort-specific risk factors are also consistent with our findings. For example, sociocultural pressures to use alcohol were likely different for the two cohorts under study. In the United States cultural and political practices such as the three-martini lunch, lower alcohol taxes, less awareness of potentially harmful effects of alcohol (e.g., impaired driving), all could contribute to observed AD differences between parents and children. Further study of generational differences may yield insight into risk (or protective) factors for AD.

In contrast to AD, the item properties for the LT models in the adolescents and their parents were similar for AB, CD, MD, and DD (displayed in Table 5). In general, DD loads highly on at the high end of EXT, but these properties are unstable due to the low base rates of non-marijuana drug dependence in the sample (as seen in the relatively wider standard errors for parameter estimates). CD loaded generally lower on the EXT factor, in both parents and adolescents, than other disorders (except for AD). This suggests that CD is a relatively poorer measure of EXT, and echoes discussion by Moffitt (1993) claiming that some adolescent conduct disorder is normative and expressed by youths who will not develop later antisocial and/or drug use tendencies, the hallmark of adult EXT.

Limitations

There are a number of limitations to this study. Independent replication of our results is necessary to support the notion that adolescent EXT is better accounted for a dimensional model than by categorical models or factor mixture models. Our study was largely Caucasian, which reflects the population of Minnesota at the time of data collection. Replication with more ethnically diverse or multi-national samples is necessary to determine the generalizability of our findings to other populations. In addition, our study did not examine gender differences in adolescent EXT. Others have found that a mean-level gender difference in EXT during adolescence largely accounts for the gender differences in indicators of EXT, suggesting that the gender differences are effectively severity differences and that the structure of EXT is largely similar for males and females (Hicks et al. 2007; Lahey, et al. 2008).

Certain limitations of our study are the result of following the methodology described by Krueger et al (2005). For instance, by only examining the five indicators of EXT found in the Krueger et al. (2005) report, we could not examine in any detail multi-factor models. Others have included more than five indicators and found that EXT was better accounted for by two or more correlated dimensions (e.g. Farmer et al., 2009). Upon close examination, however, our results are consistent with such findings. For example, Farmer et al. found that EXT comorbidity during adolescence was modeled better with a two-factor structure. The same five diagnostic indicators that we included in our report comprised a single factor they labeled “Social Norm Violation Disorders.” A second, moderately correlated factor was made up of Oppositional Defiant Disorder (ODD) and Attention Deficit Hyperactivity Disorder (ADHD). Thus, in light of their findings, our results provide evidence that the covariation among the “Social Norm Violation Disorders” cannot be better accounted for by an admixture of latent classes (a plausible alternative hypothesis that was not examined in their study). Our results also support their finding that a dimensional model is an appropriate model to represent adolescent EXT (a fundamental and untested assumption of their study). An unresolved question is whether the addition of ODD and ADHD would have replicated the 2-factor solution of Farmer at el. (2009), replicated the single-factor solution of Krueger, et al. (2005), or supported a factor mixture model. Unfortunately, because ODD and ADHD diagnoses were not available for the parent sample, we were unable to test the comparability of ODD and ADHD in the adult and adolescent samples. Future research with a more informative sample could address this point.

The present results apply only to lifetime diagnoses. Our results may have been different had we employed other strategies to define EXT, such as 12-month incident diagnoses. The use of lifetime diagnoses in this study is a reasonable starting point, as it allowed us to more easily compare our findings with previous studies that also examined lifetime rates of diagnoses (Krueger et al., 2005; Farmer et al., 2009). It also ensures that the parent data included diagnoses for behavior during the adolescent age span, and ensures that both samples were assessed during overlapping risk periods (i.e., during their adolescence). Yet, it is necessary for future work to consider that significant etiological and measurement differences most likely exist between middle-aged adults and 17-year-old adolescents that are not fully appreciated at the level of lifetime diagnosis. More sophisticated work needs to be done, perhaps by comparing scores on other, more normal-range and continuous measures (e.g., alcohol consumption), or through the development of novel instruments to assess the latent EXT construct (Krueger, Markon, Patrick, Benning, & Kramer, 2007), in order to increase variance in the measures and power to detect population differences (Grove, 1991; Markon, Chmielewski, & Miller, 2011).

Although, examining an epidemiological sample allows us to generalize our results to a larger proportion of the population, our results may have been different had we examined a

clinical sample. It is unclear if measurement properties of EXT differ between clinical and epidemiological samples. It is reasonable to expect differences in severity of EXT-related diagnoses in the clinical population (i.e., larger sample means, as well as larger variance-covariance estimates). However, it is unclear whether the pattern of comorbidity (e.g., the correlations, as opposed to covariances) among indicators of EXT is different in clinical samples. It is also possible that the proportion of shared etiology relevant to EXT is generally equivalent across community and clinical samples, such that the (standardized) factor loadings are invariant, despite the potential for mean- and variance-level severity differences. It is important to point out that the model parameters gleaned from a clinic-based sample could be tested against the parameters reported in this study, or from other epidemiological samples, but this is only a meaningful comparison if the clinic sample is described by a single latent dimension. Finding a single latent trait in a clinical sample, in addition to finding weak measurement invariance between the clinical and community samples, would be entirely consistent with, but not dispositive of, the notion that the clinical population represents the upper end of an externalizing continuum. In contrast, finding a different structural model (i.e., no single trait) or failing to find any measurement invariance would suggest that disorders segregate differently in clinical samples versus community samples.

In summary, the findings suggest that the EXT vulnerability is a useful way to describe comorbidity of externalizing disorders across generations. AB, CD, MD, and DD functioned similarly in both a parent and adolescent sample, indicating that the EXT spectrum operates similarly in both groups. An exception to this trend was AD, which was associated with less severe EXT in adults than in adolescents. In addition, AD was a poorer measure of EXT in the parents, indicating that non-EXT etiology contributes to AD in the parent sample.

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Table 1

Descriptions of Latent Trait, Class, and Factor Mixture Models of Externalizing Phenomena. Latent Variables are indirectly observed and inferred from observed measurements.

Model	Latent Variable(s)	Description
Latent Trait	Continuous	All individuals in the population are assumed to be arrayed along continua of externalizing behavior.
Latent Class	Categorical	All individuals are presumed to lie in one of *k* classes. The observed variables are assumed to be statistically independent once class membership is known. This precludes the possibility of systematic differences between individuals within a class—classes are homogenous.
Factor Mixture Model	Categorical and Continuous	All individuals are presumed to lie in one of *k* classes. The observed variables remain correlated within each class, and this residual correlation is further modeled with factor analysis. This allows for within-class systematic differences in externalizing severity.

Table 2

Tetrachoric Correlations for Adolescents and Parents. Parent correlations are reported in the upper triangle. Adolescent correlations are in the lower triangle. AB = Adult Antisocial Behavior; CD = Conduct Disorder; AD = Alcohol Dependence; MD = Marijuana Dependence; DD = Drug Dependence. All correlations are highly significant.

		Parents				
		AB	**CD**	**AD**	**MD**	**DD**
Adolescents	AB	-	.59	.64	.65	.44
	CD	.69	-	.49	.57	.52
	AD	.75	.63	-	.52	.62
	MD	.77	.62	.71	-	.81
	DD	.75	.67	.74	.84	-

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Table 3

Contingency Table of Diagnostic Status. Table is ordered first by the number of comorbid disorders. Within a value for the number of comorbid disorders (e.g., 2 disorders), the entries are ordered by frequency of observation within the adolescent sample. Ties are broken by the frequency within the parent sample. Values within the Adolescents and Parents columns are computed only for participants with no missing data. Note that the counts among adolescents with missing observations of AB are displayed. Counts for adults with missing data are not displayed for lack of space. The column of adolescents missing AB contains counts for adolescents missing a value for AB who display that particular pattern of disorder. AB = Adult Antisocial Behavior; CD = Conduct Disorder; AD = Alcohol Dependence; MD = Marijuana Dependence; DD = Drug Dependence.

	Diagnosis Present					Adolescents		Parents		Adolescents missing AB	
Number of Diagnoses	**CD**	**AD**	**MD**	**AB**	**DD**	**N**	**%**	**N**	**%**	**N**	**%**
None						2035	80.6	1850	73.2	193	79.4
One	**Y**					185	7.3	97	3.8	24	9.9
		Y				71	2.8	357	14.1	4	1.6
			Y			30	1.2	34	1.3	8	3.3
				Y		14	0.6	18	0.7	-	-
					Y	1	<0.1	2	0.1	0	0
Two	**Y**	**Y**				38	1.5	59	2.3	2	0.8
	Y		**Y**			19	0.8	9	0.4	3	1.2
	Y			**Y**		14	0.6	11	0.4	-	-
		Y	**Y**			11	0.4	30	1.2	5	2.1
			Y	**Y**		9	0.4	5	0.2	-	-
		Y		**Y**		8	0.3	38	1.5	-	-
		Y			**Y**	0	0	0	0	0	0
				Y	**Y**	0	0	0	0	-	-
			Y		**Y**	0	0	0	0	0	0
	Y				**Y**	0	0	0	0	0	0
Three	**Y**	**Y**		**Y**		20	0.8	26	1.0	-	-
	Y	**Y**	**Y**			20	0.8	14	0.6	2	0.8
	Y		**Y**	**Y**		8	0.3	5	0.2	-	-
		Y	**Y**	**Y**		6	0.2	19	0.8	-	-
	Y		**Y**		**Y**	1	<0.1	1	<0.1	0	0

	Diagnosis Present					Adolescents		Parents		Adolescents missing AB	
Number of Diagnoses	**CD**	**AD**	**MD**	**AB**	**DD**	**N**	**%**	**N**	**%**	**N**	**%**
		Y	**Y**		**Y**	1	<0.1	2	0.1	0	0
	Y			**Y**	**Y**	0	0	0	0	-	-
		Y		**Y**	**Y**	0	0	0	0	-	-
	Y	**Y**			**Y**	0	0	0	0	0	0
			Y	**Y**	**Y**	0	0	0	0	-	-
Four	**Y**	**Y**	**Y**	**Y**		26	1.0	15	0.6	-	-
		Y	**Y**	**Y**	**Y**	0	0	3	0.1	-	-
	Y	**Y**	**Y**		**Y**	0	0	2	0.1	2	0.8
	Y	**Y**		**Y**	**Y**	0	0	0	0	-	-
	Y		**Y**	**Y**	**Y**	0	0	0	0	-	-
Five	**Y**	**Y**	**Y**	**Y**	**Y**	9	0.4	5	0.2	-	-
					Totals:	2526	100%	2602	100%	243	100%

Table 4

Comparison of Factor, Class, and Factor Mixture Models in the Adolescent Sample

Model	LL	*k*	AIC	BIC
1 Trait	−2556.61	10	**5133.21**	**5192.47**
1 Class	−3038.812	5	6087.62	6117.26
2 Classes	−2572.82	11	5167.64	5232.83
3 Classes	−2552.412	17	5138.82	5239.57
4 Classes	−2549.217	23	5144.44	5280.74
5 Classes	−2548.17	29	5154.34	5326.20
2C1T	−2553.24	16	5138.22	5233.04

Note: LL is the log of the maximum likelihood estimate, *k* is the number of free model parameters, AIC is the Akaike Information Criterion, and BIC is the Bayesian Information Criterion. All models are defined fully in the text. The 1-trait model consists of a single latent factor. The class models all have categorical latent factors with varying numbers of categories. 2C1T denotes the factor mixture model with two classes, each with a single within-class trait. The 3C1T model did not converge to a local maximum despite thousands of random starts, and so results are not reported here.

Table 5

Parameter Estimates for the Two-Group Single Factor Model.

Item	Adolescents (N=2769)	Parents (N = 2619)	Likelihood Ratio Test χ^2 (*p*)
	Loadings (SE)		
AB	.90 (.02)	.87 (.03)	0.41 (.52)
CD	.74 (.03)	.66 (.04)	1.45 (.23)
AD	.84 (.03)	.70 (.03)	5.53 (.02)*
MD	.86 (.03)	.79 (.04)	1.33 (.25)
DD	.91 (.04)	.88 (.05)	0.13 (.72)
	Thresholds (SE)		
AB	1.69 (.05)	1.56 (.04)	.99 (.32)
CD	1.09 (.04)	1.28 (.03)	.33 (.57)
AD	1.39 (.04)	0.75 (.03)	33.47 (.00)*
MD	1.57 (.05)	1.57 (.04)	1.24 (.27)
DD	2.59 (.10)	2.38 (.08)	.37 (.54)

Note:

*Signficant by conventional levels. Parameters were estimated in a model that allowed all parameters to freely vary. The likelihood ratio test was conducted between the fully free model and a submodel where two parameters were constrained to be equal across groups (e.g., the loadings for AB in the parent and adolescents were constrained to be equal). AB = Adult Antisocial Behavior; CD = Conduct Disorder; AD = Alcohol Dependence; MD = Marijuana Dependence; DD = Drug Dependence. Loadings are regression slopes in a regression of the diagnosis onto the latent trait. Thresholds refer to the cutting score on a normal distribution required to yield the binary diagnostic measure. For example, the threshold on DD is 2.59 in the adolescent sample, which corresponds to the 99.5^{th} percentile of a normal distribution.

Table 6

Parameter Estimates for Final Two-Group Factor Model.

Item	Loading (SE)	Threshold (SE)
AB	.88 (.02)	1.62 (.03)
CD	.69 (.02)	1.18 (.03)
AD Parents	.68 (.03)	0.74 (.03)
AD Adolescents	.85 (.03)	1.39 (.04)
MD	.83 (.02)	1.57 (.03)
DD	.89 (.03)	2.48 (.06)

All parameters were constrained to be equal across the two groups except for Alcohol Dependence (AD), which was allowed to freely vary in the parent and adolescent groups. Loadings and thresholds are described in the text and in the caption for Table 3.