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_____ _____

Lu Dong Date

The Underlying Structure of Psychopathic Traits in Children

By

Lu Dong

Master of Arts

Psychology

Irwin D. Waldman, Ph.D.
Advisor

Scott O. Lilienfeld, Ph.D.
Committee Member

Donna Maney, Ph.D.
Committee Member

Accepted:

Lisa A. Tedesco, Ph.D.
Dean of the James T. Laney School of Graduate Studies

Date

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Lu Dong

B.A., B.S., Peking University, 2008

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Abstract

The Underlying Structure of Psychopathic Traits in Children

By Lu Dong

Despite growing research on psychopathic traits among children and adolescents in recent years, the factor structure of commonly used measures of psychopathic traits in youth is still unresolved, as is the external validity of these factors. The present thesis investigated the factor structure of psychopathic traits in children as well as the relations between psychopathic trait dimensions and reactive and proactive aggression. Across zygosity (monozygotic vs. dizygotic twins), sample type (clinic-referred vs. controls), and sex (boys vs. girls), the three-factor model (i.e., Narcissism, Callous-Unemotional traits, and Impulsivity) was shown to be the best-fitting model of psychopathic traits in children as measures by the Antisocial Process Screening Device (APSD). Factorial invariance testing suggested excellent psychometric properties of the APSD. The external validity of the three-factor model was supported by the differential associations between the three psychopathic trait dimensions and reactive and proactive aggression. The present thesis strongly supports the validity and robustness of the three-factor model of psychopathic traits in children as well as the generalizability of the APSD across samples.

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The Underlying Structure of Psychopathic Traits in Children

Psychopathy is an important clinical and forensic construct with a long history. Although it has been initially studied in adults, the downward extension of the psychopathy construct to youth has become a rapidly growing research domain with implications for better understanding the development of psychopathy. However, studying psychopathy in children is not without concerns over its legitimacy. Further, the field of child psychopathy is still struggling with unresolved measurement issues. This thesis aims to examine the underlying dimensions of psychopathic traits in children and their relations with aggression, in order to address some fundamental issues in the assessment of psychopathic traits in children.

The Conceptualization of Psychopathy within a Historical Perspective

The psychopathy construct has a long and evolving history. In early 1800's, the French physician Phillipe Pinel first recognized psychopathy as a mental condition that he described as "insanity without delirium" (Millon, Simonsen, & Birket-Smith, 1998). Around the same time, the American psychiatrist Benjamin Rush further emphasized the "the moral alienation of the mind" in psychopathy and stated that this lack of morality was primarily congenital (Millon, et al., 1998). Psychopathy has since been closely associated with social condemnations and moral judgments (Arrigo & Shipley, 2001). Over the years, the concept of psychopathy has shifted from an overly inclusive construct encompassing all "mental irregularities" to a much narrower set of personality traits and behaviors that are most socially devastating (Arrigo & Shipley, 2001).

Among the historical accounts of psychopathy, Cleckley's classic work *Mask of Sanity* (1941) is perhaps the most influential for the contemporary conceptualizations of psychopathy. Cleckley stated that psychopathic individuals are *interpersonally* egocentric,

superficial, and manipulative; *affectively* callous with a lack of empathy, guilt, or remorse; and *behaviorally* impulsive, irresponsible, and prone to criminality (Hart & Hare, 1997). In Cleckley's view, involvement in criminal activities is *not* a core feature of psychopathy. In fact, Cleckley (1941) differentiated psychopaths that engaged in illegal activities from their non-offending counterparts, who could be successful in certain careers that return great material goods. Both criminal and non-criminal psychopaths, however, appear to benefit from the primary characteristics of psychopathy (e.g., emotional detachment, lack of remorse or guilt, superficial charm, glibness) in pursuing their desires without experiencing negative feelings (Arrigo & Shipley, 2001).

Operationalization and Measurement of Psychopathy

Several diagnostic and research tools have been developed to measure psychopathy. The Diagnostic Statistical Manual of Mental Disorders (DSM) attempted to capture the psychopathy construct under the name of Sociopathic Personality Disorder (SPD) in earlier editions (1952, 1968), and Antisocial Personality Disorder (ASPD) in DSM-III (1980) and DSM-IV (1994). SPD retained the psychopathic personality traits (e.g., callous, impulsivity, cannot learn from experience) but was criticized for lacking specific diagnostic criteria (Hare, 1996). ASPD, on the other hand, does provide explicit diagnostic criteria and an improved diagnostic reliability by focusing on the behavioral characteristics (particularly the delinquent and criminal behaviors) instead of the psychopathic personality traits that many believed to be the core of psychopathy (Arrigo & Shipley, 2001; Hare, 1996; Lilienfeld, 1994). Some critics argued that ASPD has limited validity in capturing the psychopathy construct (Hare, 1998). The relation between ASPD and psychopathy remains perplexing to researchers and clinicians in the field (Arrigo & Shipley, 2001).

Contrary to the behaviorally-based approach in the DSM, several researchers have undertaken a personality-based approach to measure psychopathy (Lilienfeld, 1994, 1998). Hare developed a semi-structured interview, the *Psychopathy Checklist* (PCL; Hare, 1980) and its subsequent revision, the PCL-R (Hare, 1991, 2003), as an operationalization of Cleckley's (1941) concept of psychopathy being a constellation of personality traits (Lilienfeld, 1998). The PCL-R has been used in various settings (e.g., legal, clinical, research) and its psychometric properties are well supported (see Hare & Neumann, 2006). Nonetheless, despite the emphasis on the personality characteristics, the inclusion of antisocial behavior in the PCL-R still provokes debate, as some researchers argue that criminality is a behavioral consequence rather than a core feature of psychopathy (Cooke & Michie, 2001). The self-report measure, *Psychopathic Personality Inventory* (PPI; Lilienfeld & Andrews, 1996), offers an alternative operationalization of Cleckley's definition which does *not* explicitly assess antisocial behavior. Nonetheless, the PCL-R remains the most empirically supported measure and is regarded by many as the "gold standard" for the assessment of psychopathy (Acheson, Payne, & Olmi, 2005; Lynam & Gudonis, 2005)

Downward Extension of Psychopathy to Youth

Despite historical construct drifts and present conceptual controversies, researchers and clinicians, past and present, tend to agree that psychopathy is essentially heritable and has its roots in childhood (Arrigo & Shipley, 2001). The concept of child psychopathy appeared in the work of Cleckley (1941) and McCords (1964). However, it is not until the last two decades that researchers have begun examining the early manifestations of psychopathy in children and the development of psychopathy (Lynam & Gudonis, 2005). In an effort to disentangle the heterogeneity within Conduct Disorder, several researchers have identified emerging psychopathy-like traits in a subgroup of children with conduct problems

(Forth, Hart, & Hare, 1990; Frick, O'Brien, Wootton, & McBurnett, 1994; Lynam, 1996). A few measures were developed to assess the precursors to psychopathy in youth, including the Hare Psychopathy Checklist: Youth Version (PCL:YV; Forth, Kosson, & Hare, 2003), the Antisocial Process Screening Device (APSD; Frick & Hare, 2001), the Child Psychopathy Scale (CPS; Lynam, 1997) , the Youth Psychopathic traits Inventory (YPI; Andershed, Gustafson, Kerr, & Stattin, 2002), among others. These measures are all derivatives of the PCL-R, with different ways of adapting the items to be age-appropriate to youth (Kotler & McMahon, 2010).

Growing evidence supports the construct validity of child psychopathy and the measures used to assess it. Firstly, the factor structures of child psychopathy across measures are generally consistent with those found in adult psychopathy, although some specific differences between the factor structures of child and adult psychopathy (e.g., narcissism being highly correlated with impulsivity in children but with callous-unemotional traits in adults) require careful examination (Kotler & McMahon, 2010). Secondly, it appears that measures of child psychopathy tap traits and behaviors that resemble adult psychopathy, based on the examination of patterns of relations between these measures and theoretically relevant constructs such as temperament, psychopathology, and conduct problems (Kotler & McMahon, 2010). Furthermore, these psychopathy-like traits can be identified in children as early as age 6 (Dadds, Fraser, Frost, & Hawes, 2005) and in both community and clinical samples (e.g., Frick, Bodin, & Barry, 2000).

The usefulness of psychopathic traits in children is also implied in the literature. In particular, Callous-Unemotional traits (CU; i.e., lack of guilt, lack of empathy, cold-heartedness) seem to be most important in subtyping antisocial youth, such that the presence of CU traits designates a distinct and more severe subgroup of children with conduct

problems (e.g., Christian, Frick, Hill, Tyler, & Frazer, 1997; Frick, Cornell, Barry, Bodin, & Dane, 2003). CU traits are also associated with aggression (both concurrently and prospectively), poor treatment outcome (Frick & Dickens, 2006), and temperament characteristics such as fearlessness (Barry et al., 2000) and sensation seeking (Frick, et al., 1994). It is thus hypothesized that the presence of psychopathic traits may place children with conduct problems on a distinct etiological pathway (White & Frick, 2010). Other psychopathic traits (e.g., Narcissism, Impulsivity), though relatively less studied, were also found to be strongly associated with conduct problems (Frick, et al., 1994).

While considerable construct validity of the child psychopathy measures has been suggested in empirical studies, some legitimate concerns over the downward extension of psychopathy to youth have been raised. These concerns include the appropriateness of studying psychopathy in youth and the risk of misusing the pejorative label psychopathy (Petrila & Skeem, 2003; Seagrave & Grisso, 2002), given that adult psychopathic offenders are regarded as essentially "untreatable" and require long-term institutionalization (Skeem, Monahan, & Mulvey, 2002). Further, the factor structure of adult psychopathy is still under debate, which makes the downward translation of the construct even more questionable.

Apart from the conceptual issues in child psychopathy, several important measurement issues have not been resolved. The factor structures of child psychopathy are not stable both within and across measures. Though several factor models are present in the literature, there is no strong basis for which model best represents the underlying structure of psychopathic traits in children (Kotler & McMahon, 2010). Relative to the adult psychopathy literature, there is also a paucity of studies examining the psychometric properties of child psychopathy measures in clinical, community, and forensic samples. Given that most child and adult psychopathy studies have focused on males and Caucasian-

Americans, the generalizability of the construct and measures of psychopathy to females and individuals from other culture and ethnic backgrounds is much less investigated.

The Present Thesis

As discussed above, there have been advances and concerns in studying psychopathic traits in children. Although this line of research seems promising and necessary in understanding the development of psychopathy and antisocial behavior, researchers should be particularly cautious in labeling any child as being "psychopathic". More importantly, fundamental measurement issues need to be addressed to facilitate further investigations. The primary goal of the present thesis is to better establish the internal and external validity of a commonly used child psychopathy measure (i.e., APSD) by examining its factor structure and its relations with aggression, a theoretically important correlate of psychopathy, across both clinically-referred and non-referred samples and several relevant subsamples. This thesis includes two distinct but related studies. In the first study, confirmatory factor analyses were conducted to determine the best-fitting factor model for APSD and factorial invariance was tested across several samples/ subsamples to examine the generalizability of APSD. In the second study, the relations between APSD dimensions from the best-fitting factor model and reactive and proactive aggression were investigated to further support the construct validity of the best-fitting model for the underlying structure of the APSD.

Study 1: Confirmatory Factor Analyses and Factorial Invariance Testing of the Antisocial Process Screening Device

In his classic work *The Mask of Sanity*, Cleckley (1941) described the psychopath as a superficially charming and intelligent person absent of emotions and guilt, as well as

irresponsible and manipulative of others. Following Cleckley's work, substantial efforts have been placed on various approaches to the conceptualization, assessment, and development of psychopathy. Although there is still considerable debate over the defining features of psychopathy, especially with regard to the importance of antisocial behavior, one overarching theme regarding the contemporary conceptualization of psychopathy concerns its multifaceted nature comprising several personality traits and specific behavioral manifestations (Krueger, 2007).

Following different perspectives on the conceptualization and operationalization of psychopathy, several competing factor models have been proposed and examined in the literature. One prevalent definition proposed by Hare (1996) emphasizes both the personality characteristics of psychopathy, including affective and interpersonal deficits (e.g., egocentricity, impulsivity, lack of empathy/ guilt/ remorse, irresponsibility, shallow emotions) and the persistent socially deviant behaviors such as antisocial behavior and criminality (e.g., Hare, 1996). Emanating from this definition was the development of the *Psychopathy Checklist-Revised* measure (Hare, 1991, 2003), a semi-structured clinical interview regarded by many as the "gold standard" for adult psychopathy (Acheson, et al., 2005). Most of the evidence for the validity of the PCL-R was based on Caucasian male offenders and its underlying factor structure has been a point of controversy.

The PCL-R was traditionally represented as having two distinct but correlated factors: Factor 1 -- Interpersonal/ Affective, which primarily assesses personality features such as superficial charm, lack of remorse, shallow emotion, and callousness; and Factor 2 -- Socially Deviant Behaviors, which consist of behavioral features including impulsivity, irresponsibility, and delinquency (Harpur, Hakstian, & Hare, 1988; Hart & Hare, 1989). Cooke & Michie (2001) challenged the traditional two-factor model by proposing a three-

factor model (i.e., Interpersonal, Affective, and Lifestyle), deemphasizing the antisocial behavior features. Subsequent studies have supported the good fit of this model (Forth, et al., 2003; R. L. Jackson, Rogers, Neumann, & Lambert, 2002; Skeem, Mulvey, & Grisso, 2003; Weaver, Mayer, Van Nort, & Tristan, 2006). More recently, Hare (2003) proposed a four-factor model for the PCL-R, adding the socially deviant behaviors to Cooke and Michie's three-factor model. The first two factors remained the Interpersonal and Affective, whereas the behavioral factor from the two-factor model was divided into two subscales: Lifestyle and Antisocial. This four-factor model also has been supported empirically (Hare & Neumann, 2006; Neumann, Kosson, Forth, & Hare, 2006)

Until recently, the majority of research on psychopathy has focused on adults, particularly on forensic populations. In the past two decades, there has been an increasing interested in studying psychopathic tendencies in youth. Although the applicability of the construct of psychopathy to youth is controversial, this line of research began with attempting to explain heterogeneity in Conduct Disorder in children. Frick et al. (1994) developed a parent and teacher rating scale that was adapted from the *PCL-R* to measure psychopathic traits in children, which was later published as the *Antisocial Process Screening Device* (APSD; Frick & Hare, 2001).

The APSD has been widely used in research for understanding the nature of psychopathic traits in children and adolescents. Nonetheless, evidence for the construct validity of APSD is still not firmly established. Similar to the adult psychopathy literature, a two-factor model was first suggested for APSD. Frick et al. (1994) identified a two-factor model of psychopathic tendencies among clinic-referred children via Principal Components Analysis (PCA). This two-factor model resembles what has been found in the adult literature regarding the PCL-R, as the first factor was labeled Impulsivity/ Conduct

Problems (I/CP) and the second factor was labeled Callous/ Unemotional (CU) and contains items similar to those associated with psychopathic personality features in adults (e.g., lack of guilt, shallow emotion). Interestingly, unlike the findings with adults, items related to narcissism loaded on the I/CP rather than the CU factor.

Frick, Bodin, & Barry (2000) extended the clinic-referred sample and recruited a community sample to further test the structure of psychopathy in children. Both two- and three-factor models were justified in the two samples based on PCA. The new two-factor model was similar to the one extracted by Frick et al. (1994), whereas in the three-factor model the I/CP factor was separated into two factors, namely Narcissism and Impulsivity, and the CU factor remained intact. In addition, Confirmatory Factor Analyses (CFAs) were conducted in the clinic-referred sample, testing a one-factor model as well as the two- and three-factor nested models. The results showed that both the two- and three-factor models fit the data equally well and no statistical support was found for one of these models over the other. However, the three-factor model was preferred due to the theoretical interest in studying the Narcissism and Impulsivity constructs separately in children (2000; 1994).

Following Frick et al. (2000; 1994), several studies have tried to replicate and validate these factor structures in various samples. Evidence regarding the best fitting factor structure of the APSD is inconsistent across studies, depending on different sample characteristics including sample type (i.e., clinic-referred versus non-referred samples), sex, age (i.e., pre-adolescent children versus adolescents), means of administration (parent or teacher ratings versus self-reports), and ethnicity. The two-factor model has been supported in clinic-referred children (Fite, Greening, Stoppelbein, & Fabiano, 2009; Frick, et al., 2000), whereas the three-factor model has been favored in non-referred community children (Dadds, et al., 2005) and incarcerate adolescents (Vitacco, Rogers, & Neumann, 2003).

In summary, although the APSD is widely used in research, its construct validity is still at an early stage and several important issues need to be addressed. First, the factor structure of psychopathic tendencies in youth, especially in pre-adolescent children, needs further investigation. At present, there is no sufficient evidence regarding which factor model fits significantly better than others in school-age children. One problem is that some studies only tested one proposed factor structure, which prevents determining which model best captures the underlying structure of the APSD in a particular population. Second, some studies have conducted CFAs using estimation methods that may cause bias in the evaluation of model fit. Specifically, treating categorical variables as continuous in CFA may result in underestimation of the relations among the items as well as biased test statistics, parameter estimates, and standard errors (Brown, 2006).

Third, it is not known whether the factor structure of the APSD is consistent across populations, and whether the psychopathy dimensions measure the same latent traits in distinct groups, such as boys and girls, and different ethnicities, sample types, and cultures. These are important issues in measure development which can be tested through multiple-group CFA and a series of structural and measurement invariance models (Reise, Widaman, & Pugh, 1993). Unfortunately, structural and measurement invariance is often assumed but rarely tested for psychopathy measures such as the APSD.

The Present Study and Hypotheses

In the current study we attempt to answer the following questions: (1) which factor model best represents the underlying structure of child psychopathic traits as measured by the APSD? (2) Are the APSD items' factor loadings, variances, and covariances invariant across different samples and subgroups? (3) Are there true group differences in the level of the latent psychopathic traits after adjusting for measurement errors and any invariance?

Based on the previous literature, we hypothesize that (1) in the clinic-referred sample, both the two- and three-factor correlated models will fit well; (2) in the non-referred controls and the community sample, the three-factor model will fit significantly better than the two-factor model; (3) structural and measurement invariance will hold across zygosity, suggesting that the APSD performs identically in both MZ and DZ twins; (4) there will be partial measurement invariance across sex and sample type (clinic-referred children versus non-referred controls), whereas structural invariance will not hold across sex and sample type, thus indicating some degree of population heterogeneity in the measurement characteristics of the APSD across populations.

Method

Participants and procedure

The present study consists of two samples. The community twin sample (*Sample 1*) comprised 846 twin pairs from the Georgia Twin Registry, a population-based twin registry of 4 to 17 year-old twins (*Mean* = 10.6 years, *SD* = 3.2 years), with 49% males, 82% European Americans, 11% African Americans, 1% Hispanic Americans, and 6% mixed/other ethnicity. The sample consisted of 392 (46%) monozygotic (MZ) and 454 (54%) dizygotic (DZ) twin pairs. Twins were recruited using the following procedures. In 1992 to 1993, 5,620 parents of twins born between 1980 and 1991 in the state of Georgia were contacted via mail according to the state birth records. Of these families, 1,567 twin families joined the Georgia Twin Registry, among which 846 families provided complete ratings on the psychopathology and psychopathy measures. The zygosity of the twins was determined based on parent reports of twins' physical similarity using a 9-item scale previously validated against DNA polymorphisms ((Bonnelykke, Hauge, Holm, Kristoffersen, & Gurtler, 1989).

The combined sample (*Sample 2*) comprised a group of clinic-referred children and their siblings (*N*=350) from 213 families and a group of non-referred children (*N*=153) from 77 families that serve as controls. The clinic-referred children and their siblings included 228 boys (65%) and 122 girls (35%), with 78% Caucasian Americans, 10% African-Americans, 2% Hispanic Americans, 1% Asian Americans, and 9% mixed/ other ethnicity. The mean age for this group was 10.7 (*SD*=3.6) years. The control sample included 67 boys (44%) and 84 girls (56%), with predominantly (92%) Caucasian-American ethnicity and mean age 13.7 (*SD*=2.5) years. The clinic-referred children and their siblings were recruited through the Center for Learning and Attention Deficit Disorders (CLADD) at Emory University in Atlanta, Georgia and through private psychiatric practices in Tucson, Arizona. The controls were sampled from both the Georgia Twin Registry and the general population in the Arizona site.

Measures

Psychopathic traits were assessed using the APSD, which is a 3-point, 20-item scale rated by primary caregivers or teachers to screen for antisocial characteristics and processes in youth. Three domains are thought to underlie the APSD items, namely Narcissism (e.g., 'brags excessively'), CU (e.g., 'is concerned about others' feelings', reversely coded), and Impulsivity (e.g., 'acts without thinking'). In the present study, we used the previous version of this measure (i.e., the Psychopathy Screening Device) rated by the child's mother. Each item is rated on a 0-4 scale, with 0 meaning not at all like the child and 4 meaning describing the child very well.

Data analyses

We conducted a series of confirmatory factor analyses contrasting alternative models for the underlying structure of the APSD in the community twin sample (*Sample 1*) as well as

the combined sample of clinic-referred children and twin controls (*Sample 2*). The CFA framework allows for imposing constraints on the measurement model and for the direct comparison of alternative models. Another advantage of CFA is that all aspects of measurement and structural invariance in a factor model can be tested within the CFA framework. In the present study, we first determined the best-fitting model for APSD in both samples and in each individual subgroup. Specifically, within each sample and subgroup we tested a one-factor model corresponding to general psychopathy, the traditional two-factor model (Frick, et al., 1994) comprising Impulsivity/ Conduct Problems (I/CP) and Callous/ Unemotional (CU) factors, and the three-factor model (Frick, et al., 2000) comprising Callous-Unemotional, Narcissism, and Impulsivity dimensions within a set of hierarchically nested models. In a set of hierarchically nested models, the models include increasing numbers of constraints (i.e., parameters that are fixed to be equal to each other or equal to some value, such as 0), and the fit of the more restricted model (i.e., the nested model) is statistically compared to the fit of the less restricted model (i.e., the parent model). The viability of these constraints is examined using a χ^2 difference test with the null hypothesis of equal fit for both models and the number of degrees of freedom equal to the number of constraints imposed in the more restricted model. A non-significant χ^2 difference test suggests that the fit of the more restricted model is not significantly worse than the fit of the less restricted model and thus the more restricted model is favored (Brown, 2006; Schermelleh-Engel & Moorehouse, 2003).

Factorial invariance (i.e. measurement and structural invariance) of the APSD items under the best-fitting model was next tested using multiple-group CFA. The analyses were conducted proceeding from the least constrained model to the fully constrained model. We first examined measurement invariance by testing the equivalence across groups of the

measurement parameters (i.e., factor loadings, residual variances, and item thresholds). Second, structural invariance was tested by equating across groups the structural parameters (i.e., factor variances, factor correlations, and factor means). The least constrained model (i.e., the congeneric model), which tests the presence of an equal factor pattern (i.e., all of the items loading on the same factors) across groups, freely estimated all of the parameters separately for both groups, although some constraints were imposed for model identification (e.g., item thresholds were equated across groups, factor variances were fixed at 1 in both groups, item residual variances and factor means were fixed at 1 and 0, respectively, in one group and were freely estimated in the other group). Successive constraints equating the factor loadings and item residual variances were used to test aspects of measurement invariance in turn, whereas equating the factor variances, factor correlations, and factor means were used to test aspects of structural invariance in turn. These alternative models were evaluated by directly comparing the more restricted model to a less restricted well-fitting model using the same χ^2 difference tests described above. Supplemental fit indices (described below) were also used to aid in the selection among alternative models.

All data analysis were performed using Mplus 6.0 (Muthén & Muthén, 1998-2010). As recommended in the MPlus manual and in the relevant statistical literature (e.g., & Curran, 2004), a weighted least squares (WLSMV) estimator was used to accommodate ordinal data (i.e. APSD items are assessed on an ordinal scale) using a diagonal weight matrix (with standard errors and mean- and variance-adjust chi-square test statistic; Muthén & Muthén, 1998-2010). The *difftest* option was used to perform the χ^2 difference test to contrast alternative models. Given that both samples consist of siblings or twins that are nested within families, the *cluster* option was used to account for the presence of multiple children within families. For the multiple group analyses, *the theta parameterization* was used under

WLSMV estimation, resulting in the residual variances of all APSD items being fixed at one in one group and are freely estimated in the second group.

For the evaluation of model fit, we reported the following goodness-of-fit indices: the χ^2 test statistic and its associated degrees-of-freedom (*df*) and *p* value, the Tucker-Lewis index (TLI), and the root mean square error of approximation (RMSEA). Assessment of the adequacy of model fit was based on guidelines suggested in the literature: TLI ≥ 0.95 for excellent fit (Hu & Bentler, 1999) and values between 0.90 - 0.95 for acceptable model fit (Bentler, 1990); RMSEA ≤ 0.08 for adequate fit and ≤ 0.05 for good model fit (Browne & Cudeck, 1993). Although χ^2 values are reported in the present study, they were not used for model evaluation because the χ^2 is inflated in large samples resulting in models being rejected even when the difference between the observed and predicted variance-covariance matrices is not significant (Brown, 2006). Model fit was determined by the combination of all fit indices reported, as each individual fit index has its limitations and no consensus has been reached regarding use of a single fit index to evaluate the adequacy of model fit.

Results

Consistency across zygosity: monozygotic twins (MZ) vs. dizygotic twins (DZ)

As is shown in Table 1, although the two-factor correlated and three-factor correlated models have acceptable fit for both MZ and DZ twins separately as well as in the community twin sample as a whole, the 3-factor model is preferred as it showed better fit as indicated by higher TLI values and lower RMSEA values. In contrast, the one-factor, two-factor orthogonal, and three-factor orthogonal models fit the data poorly, with TLIs all below 0.800 and RMSEA above or around 0.080. Further comparisons of the nested models using χ^2 difference test, as shown in Table 2, also suggest that the three-factor

correlated model is the best-fitting model in MZ and DZ twins both separately as well as combined.

Since the three-factor correlated model was the best-fitting model relative to the other four alternative models, we adopted this as a baseline model to test for measurement and structural invariance across MZ and DZ twins. As shown in Table 3, all the factor loadings (χ^2_{diff}= 17, df = 18, p= .525) and residual variances (χ^2_{diff} = 15, df = 18, p = .650) were equatable across zygosity. Using the most constrained measurement model, structural invariance was tested with a series of equality constraints on the factor variances, correlations, and means across groups. Results show that both the factor variances (χ^2_{diff} = 1, df = 3, p = .705) and factor correlations (χ^2_{diff} = 2, df = 3, p = .503) were equatable across zygosity. In contrast, the factor means were equatable across zygosity only for CU (χ^2_{diff} = 3, df = 1, p = .106). The most restricted model (reported in detail in Table 4), in which all factor loadings (and thresholds, which were fixed by default), item residual variances, factor variances, and factor correlations were constrained across zygosity, showed excellent model fit (TLI = 0.962, RMSEA = 0.035).

Consistency across sample type: clinic-referred vs. non-referred children

Table 1 also presents the results from CFAs comparing alternative models in different sample types (clinic-referred vs. controls). Similar to the findings across zygosity, although the two-factor correlated and the three-factor correlated models showed adequate fit, the three-factor model fit best in both the combined sample as well as in the clinic-referred and non-referred controls separately. Comparison of the nested models, shown in Table 2, also suggested that the three-factor correlated model fit better than all the alternative models (all $p < .001$) in the clinic-referred, non-referred, and the combined samples.

Tests of measurement and structural invariance (shown in Table 3) were again conducted using the best-fitting model (i.e., the three-factor correlated model) across sample type as a baseline. The equality of factor loadings across the samples was tested by comparing Model 2, in which factor loadings were constrained to be equal across samples, with the baseline model (i.e., the congeneric model, Model 1, in which all parameters were freely estimated in both samples (except for the item thresholds, which were equated across samples by default). Results of the χ^2 difference test (Model 2 vs. 1: $\chi^2_{diff} = 34$, $df = 18$, $p = .012$) suggest that constraining all factor loadings across samples could be rejected. Partial measurement invariance was then explored to determine the source of non-equivalence and the results showed that the factor loadings for all APSD items except for item 5 ('emotions seem shallow', on the Narcissism factor) were equatable (as in Model 2') across samples without significantly worsening the model fit (Model 2' vs. 1: $\chi^2_{diff} = 22$, $df = 17$, $p = .178$). The test of equal item residual variances also was rejected (Model 3 vs. 2': $\chi^2_{diff} = 69$, $df = 17$, $p < .001$), suggesting significantly different item error variances in clinic-referred and control children.

Based on the partially invariant measurement model, structural invariance across sample type was next tested. Results of these tests suggested that all of the factor variances were equivalent across the two samples (Model 4 vs. 2': $\chi^2_{diff} = 4$, $df = 3$, $p = .279$), whereas only the correlation between Narcissism and Impulsivity could be equated across sample type (Model 5' vs. 2': $\chi^2_{diff} = 1$, $df = 1$, $p = .248$). Further, the clinic-referred sample was significantly higher in the mean levels of all three psychopathic factors (i.e., 0.665 for Narcissism, 0.798 for CU, and 0.756 for Impulsivity, all $p < .05$) than those of the non-referred control sample (i.e., in which all three factor means were fixed at zero). Adequate model fit (Model 5': TLI = 0.950, RMSEA = 0.055) was found for the model in which all

factor loadings (except for item 5), factor variances, and the factor correlation between Narcissism and Impulsivity were constrained across sample type.

Consistency across sex: boys vs. girls

Table 1 presents the results of CFAs across sex in both *Sample 1* (community twin sample) and *Sample 2* (combined sample of clinic-referred and controls). Consistent with the findings of CFAs comparing alterative factor models across zygosity and sample type, the three-factor correlated model fit best across sex in both the community twin and the combined samples, as shown in Table 1 and 2.

Tests of measurement invariance were conducted across sex for both samples, as presented in Table 3. Although the factor loadings could not be all equated across sex in either sample (present the relevant test results), the majority of factor loadings (Model 2' vs. 1: $\Delta\chi^2_{diff} = 20$, $df = 17$, $p = .285$ for *Sample 1*; $\Delta\chi^2_{diff} = 18$, $df = 16$, $p = .339$ for *Sample 2*) could be equated across sex, as could the item residual variances (Model 3 vs. 2': $\chi^2_{diff} = 21$, $df = 17$, $p = .246$ for *Sample 1*; $\chi^2_{diff} = 23$, $df = 16$, $p = .122$ for *Sample 2*). As also shown in Table 4, items that were not equatable across sex include: item 14 ('acts charming', loading on Narcissism) for *Sample 1* (the community twin sample), and item 5 ('emotions seems shallow', loading on Narcissism) and item 13 ('engaged in risky activities', loading on Impulsivity) for *Sample 2* (clinic-referred and controls).

Tests of structural invariance by sex suggested similar findings for both samples, specifically that factor variances (Model 4 vs. 2': $\chi^2_{diff} = 2$, $df = 3$, $p = .501$ for *Sample 1*; $\chi^2_{diff} = 2$, $df = 3$, $p = .519$ for *Sample 2*) and factor correlations (Model 5 vs. 2': $\chi^2_{diff} = 1$, $df = 3$, $p = .725$ for *Sample 1*; $\chi^2_{diff} = 2$, $df = 3$, $p = .483$ for *Sample 2*) were equable across sex, while factor means could only be equated for Narcissism (Model 6' vs. 5: $\chi^2_{diff} = 4$, $df = 1$, $p = 0.061$ for *Sample 1*; $\chi^2_{diff} = 2$, $df = 1$, $p = 0.148$ for *Sample 2*). In both samples, the means of CU

were significantly lower (all $p < .05$) in girls than those in boys (in whom the means were fixed = 0; -0.418 for *Sample 1*; -0.199 for *Sample 2*) and Impulsivity (-0.456 for *Sample 1*; -0.349 for *Sample 2*). The most restricted well-fitting model (i.e., Model 6', in which all equatable measurement and structural parameters were constrained) showed excellent model fit (TLI = 0.954, RMSEA = 0.038 for *Sample 1*; TLI = 0.968, RMSEA = 0.047 for *Sample 2*).

Factor loadings and correlations for the APSD

Table 4 shows the factor loadings and correlations of the most restricted models from each comparison (i.e., by zygosity, sample type, and sex). For Narcissism, item 5 ('emotions seem shallow') had the highest loading in most subgroups (MZs, DZs, boys in *Sample 1*, non-referred controls, and girls in *Sample 2*); item 14 ('can be charming') had the highest loading in the remaining subgroups (girls in *Sample 1*, clinic-referred children, and boys in *Sample 2*). Consistent across samples/ subgroups, item 18 ('concerned about feelings of others', reversely coded) and item 4 ('acts without thinking') had the highest loadings on CU and Impulsivity, respectively. As for factor correlations, Narcissism and Impulsivity were most highly correlated (0.863-0.879), whereas CU and Impulsivity were least correlated (0.384-0.680) in all samples/ subgroups.

Discussion

Despite increasing research on the downward extension of psychopathy to youth, inconsistent empirical evidence regarding the underlying structure of psychopathic traits in youth both within and across measures in various samples pose considerable measurement issues in the child psychopathy literature. In an attempt to solve this problem, the present study examined the psychometric properties of the most frequently studied measure of child psychopathic traits, the APSD, by testing competing alternative factor models as well as

measurement and structural invariance across multiple samples (i.e., clinic-referred vs. non-referred) and subgroups (i.e., sex, zygosity) of children using CFAs.

The CFAs suggested that the three-factor correlated model fit better than all alternative models, including the two-factor model, in each subgroup examined (i.e., MZ twins, DZ twins, clinic-referred children, non-referred children, males, and females), as well as in both *Samples 1* and *2*. Although the two-factor correlated model showed adequate fit in this study and in much of the literature, the $\Delta\chi^2$ difference test comparing the two- and three-factor correlated models favored the three-factor model in both samples and all subgroups.

Tests of measurement invariance suggested full measurement invariance across zygosity, and partial measurement invariance across sample type (clinic-referred vs. non-referred children) and sex (boys vs. girls). In the context of full or partial measurement invariance, structural invariance was subsequently tested in each multiple-group comparison. Factor variances were all equatable across all group comparisons, whereas factor correlations were also all equatable across zygosity and sex. In contrast, only the correlation between Narcissism and Impulsivity was equatable across clinic-referred and non-referred children. True group differences (i.e., not due to measurement errors) in the mean level of the latent psychopathic traits were found for all comparisons. Specifically, the factor means were substantially higher in clinic-referred than in non-referred children for all three APSD factors, significantly higher in males than in females for the CU and Impulsivity factors, and slightly higher in DZ than in MZ twins for Impulsivity.

The current findings should make substantial contributions to resolving the inconsistent findings on the APSD factor structure in the extant literature. We found robust evidence favoring the three-factor model over the competing two-factor model as the best-fitting model for the underlying structure of the APSD, which provides a conclusive

resolution of the inconsistent findings of APSD factor structures in the literature. This finding is compelling because it holds for MZ and DZ twins, both clinic-referred and non-referred community children, and boys and girls, indicating that the factor structure of the APSD shows excellent stability and consistency across samples and subgroups based on various characteristics. Furthermore, the strong support for the three-factor model of psychopathic traits in children also suggests considerable similarity between the factor structure of psychopathy in children, adolescents, and adults, as the three-factor model for the APSD in children parallels the three-factor model that has been established for the PCL: Youth Version in adolescents (Forth, et al., 2003; Neumann, et al., 2006; Sevecke, Pukrop, Kosson, & Krischer, 2009), and the PCL-R in adults (Cooke & Michie, 2001; R. L. Jackson, et al., 2002; Skeem, et al., 2003; Weaver, et al., 2006).

With the three-factor model fitting best, our findings strongly support the distinction between Narcissism and Impulsivity factors. Previously, Frick et al. (2000) advocated for distinguishing Narcissism and Impulsivity based on research interest in studying these traits separately in children. Others argued for combining Narcissism and Impulsivity based on consistently high correlations between the two factors across all studies (Fite, et al., 2009). These two factors were also highly correlated in our analyses (i.e., r's consistently around 0.87) with comparable magnitude across all samples and subgroups, suggesting that the two factors shared a substantial amount of variance. However, the comparison of nested models demonstrated that the three-factor solution fit better than the two-factor solution, even accounting for model complexity. Thus, although highly correlated, Narcissism and Impulsivity factors appear to be statistically distinguishable. Nonetheless, subsequent examination of the differential associations of Narcissism, Impulsivity, and CU with relevant correlates is necessary to confirm this conclusion based on external validity.

Our study is the first to investigate measurement and structural invariance of the APSD across clinical and community samples as well as in boys and girls. The present results provide good evidence for the psychometric properties of the APSD. Satisfying the assumptions of equivalent measurement is crucial for establishing the generalizability of a measure (Meredith, 1993; Widaman & Reise, 1997) and our results show robust support for the generalizability of the APSD across different populations. The establishment of congeneric measurement in all samples and subgroups demonstrated that the same factor structure (i.e., the three-correlated factor model) underlies psychopathic traits in children across MZ and DZ twins, clinic-referred and community samples, and boys and girls. Partial measurement invariance was found in the comparisons of the item factor loadings and residual variances across sample type and sex, suggesting that the relations between observed scores on the APSD items and the latent psychopathy dimensions is equivalent across groups for the vast majority of APSD items. Based on these results, we conclude that the APSD, developed in clinical samples of predominantly boys of European ancestry, can be generalized to nonreferred population-based samples of children and to girls. Due to the nature of our samples, specifically the relatively small numbers of individuals of non-European ethnic ancestry, generalizability of the APSD factor structure and measurement invariance across ethnic groups could not be tested but should be investigated in future studies.

The establishment of equal factor variances and correlations (except for factor correlations across sample type) in the multiple-group comparisons indicates that the distributions of and the relations among the latent psychopathy factors are similar across subgroups. Lack of invariance in factor means across subgroups indicates true differences in the mean levels of APSD, free from any measurement error, which has important clinical

implications. For example, we found that boys have higher means than girls for CU and Impulsivity in both samples, which is consistent with prior findings that boys have higher mean scores than girls on the APSD factors (i.e., Narcissism, CU, and Impulsivity). This is also consistent with findings of sex difference in psychopathy in adults. Sex differences in the PCL-R have been consistently reported such that higher percentages of males scored above 30 points than females in criminals and forensic psychiatric population (Bolt, Hare, Vitale, & Newman, 2004), despite the similarity of the factor structures across sex in adults. Although a cut-off score was not recommended for the APSD for various reasons (Frick, et al., 2000; Frick & Hare, 2001), higher means of psychopathic traits in boys than in girls suggest higher levels of psychopathy in boys if the APSD was used to create clinical groups.

Strengths and limitations

The present study has several methodological strengths. First, the use of CFA approach is more advantageous than exploratory factor analysis or principal components analysis used in previous studies, because CFAs enable the testing and comparison of model fits of highly constrained a priori factor models. Second, we directly contrasted competing factor models (e.g., two- versus three- factor models) proposed in the literature using formal statistical procedure, which yielded compelling evidence for the best-fitting model containing three correlated factors. Moreover, the same best-fitting factor structure was statistically supported in two independent samples and six different subgroups, making this finding much more robust and generalizable than previous factor structures reported in the extant literature. Finally, measurement and structural invariance were formally tested and largely supported across the different samples and groups, providing a high level of generalizability of the APSD across different populations.

Although several important group comparisons for APSD were investigated, the generalizability of APSD across ethnic groups was not examined due to both our clinic-referred and community samples being predominantly European-American. Racial/ethnic generalizability is a critical piece of validity evidence for any psychopathy measures given that the majority of studies in the assessment of psychopathy, for both adults and children, are based on Caucasian males. In adults, despite some evidence showing ethnic-group differences between Caucasian- and African-Americans in the manifestation and correlates of psychopathy (Doninger & Kosson, 2001; Newman, Schmitt, & Voss, 1997), little difference was found for the factor structure and few differences for item functioning in the examination of racial biases for the PCL-R (Cooke, Kosson, & Michie, 2001). In children, little research has examined whether there is ethnic/racial bias with the APSD specifically. Therefore, future studies should examine potential ethnic group differences in the factor structure and measurement and structural invariance of the APSD in more diverse samples of children.

Future studies should also incorporate teacher ratings of the APSD in addition to parent ratings, on which the present study relied entirely. Inclusion of information from both sets of raters may result in a more comprehensive profile of children's psychopathic tendencies, as parents may be biased informants even though they usually are the most knowledgeable about the child (Carter, Godoy, Marakovitz, & Briggs-Gowan, 2009). Along these lines, it has been recommended that a combined score on the APSD be obtained from both teacher's and parents' ratings (Frick & Hare, 2001). In addition, future studies should extend the nomological network of the APSD by examining correlations between each of the three APSD factors and external variables including different dimensions of aggression (e.g., reactive and proactive), other relevant personality traits (e.g., conscientiousness,

neuroticism, agreeableness), and aspects of internalizing and externalizing psychopathology. It is especially important to test whether psychopathy-related Narcissism and Impulsivity are differentially related to external variables, in order to further support the validity of making such distinctions in children.

Study 2: Psychopathic Trait Dimensions are Differentially Associated with Reactive and Proactive Aggression in Youth

Psychopathy is a condition characterized by affective and interpersonal features such as superficial charm, egocentricity, callousness, lack of guilt, and shallow emotions, as well as behavioral maladaptations such as persistent social deviance, impulsive actions, and irresponsibility (Hare, 1996). Although research has focused primarily on psychopathy in adults (see Patrick, 2006), there is a growing literature on the etiology and development of psychopathy in children and adolescents (Salekin & Lynam, 2010). Accordingly, the valid characterization of psychopathic tendencies in children has assumed increasing importance. Although some researchers have attempted to establish the external validity of the component dimensions underpinning psychopathy by means of correlations with relevant criteria such as psychopathology (e.g., externalizing and internalizing disorders) and aggression, this research is still in its early stages (Johnstone & Cooke, 2004). In the present study, we investigate the relation between psychopathic traits in children and two theoretically important correlates of psychopathy, namely, reactive and proactive aggression.

The question of whether aggression or antisocial behavior is a core characteristic of psychopathy is controversial (Cooke, Michie, & Hart, 2006; Hare & Neumann, 2006; Lilienfeld, 1994; Skeem & Cooke, 2010). The traditional two-factor model of the Psychopathy Checklist-Revised (PCL-R; Hare, 1991; Harpur, et al., 1988), a commonly used

interview-based measure of psychopathy in adults, includes Interpersonal/ Affective and Socially-Deviant Lifestyle factors (Hare et al., 1990). In contrast, a more recent three-factor model includes Affective, Interpersonal, and Irresponsible/Impulsivity factors, and de-emphasizes aggression (Cooke & Michie, 2001). A most recent four-factor model has added back in Antisocial Behaviors as the fourth factor (Hare & Neumann, 2006), while the other three dimensions remain unchanged.

The underlying structure of psychopathic traits in children similarly remains unclear. Following factor models in adult psychopathy, a two-factor model consisting of Impulsivity/ Conduct Problem (I/CP) and Callous-Unemotional traits (CU; Frick, et al., 1994) (CU) and a three-factor model consisting of Narcissism, CU, and Impulsivity (Frick, et al., 2000) were proposed for the Antisocial Process Screening Device (APSD; Frick & Hare, 2001), a measure of psychopathic tendencies in childhood. Empirical evidence for the best-fitting factor model has not been consistent across samples (e.g., clinic-referred, inpatient, community) or age groups (e.g., preschool, pre-adolescent children, and adolescents). There also is no strong theoretical basis for choosing among these competing factor models (Kotler & McMahon, 2010). The two-factor model is more parsimonious than the three-factor model and appears to fit better in clinic-referred children (Fite, et al., 2009; Frick, et al., 2000), although other studies have found the three-factor model to fit better among younger children (4-9 years old; Dadds, et al., 2005) and incarcerated adolescent samples (Vitacco, et al., 2003). Although empirical support for separating Narcissism and Impulsivity was not entirely convincing, Frick et al. (2000) recommended the three-factor model based on advances in the assessment of psychopathy in adults that favored three- and four-factor models, as well as an interest in distinguishing these traits in youth. Nonetheless, the

question of whether three factors are differentially associated with theoretically relevant external correlates better than two factors remains unresolved.

In previous studies, differential associations have been found between APSD subscales and measures of external constructs such as features of DSM-IV disorders (e.g., attention deficit hyperactivity disorder, conduct disorder, oppositional defiant disorder; Frick, et al., 2000; Sevecke & Kosson, 2010) and personality dimensions such those from the Five Factor Model (Lynam, 2010). In this study, we evaluate the external validity of the three-factor model of APSD by examining associations between the three APSD dimensions and reactive and proactive aggression. We adopt the three-factor model because it is more widely accepted in the literature and fits better than the two-factor model in our samples in a set of confirmatory factor analyses (CFA) conducted previously (Dong & Waldman, 2010 June).

We focus on aggression because of its theoretical importance as a correlate of psychopathy (Skeem & Cooke, 2010). Like psychopathy, aggression can best be viewed as multifaceted given mounting evidence suggesting that reactive and proactive aggression differ in important ways. Reactive aggression refers to emotionally provoked behaviors stemming from anger or frustration, and perceived rejection or other threats to oneself, whereas proactive aggression refers to goal-directed, unprovoked, and largely unemotional aggression (Dodge & Coie, 1987; Vitaro, Gendreau, Tremblay, & Oligny, 1998). Historically, this distinction has been labeled in different ways and is also referred to as hostile (reactive, retaliatory, impulsive, or affective) versus instrumental (premediated, opportunistic) aggression. These distinctions derive largely from early ethological observations and models of aggressive behaviors in both humans and nonhuman animals (Eichelman, 1992). Although concerns have been raised regarding the validity and clinical utility of the reactive-

proactive aggression distinction (Bushman & Anderson, 2001), and despite the substantial correlation often found between these two factors, reactive and proactive aggression are readily distinguishable and contribute to separable subgroups of aggressive individuals (Brendgen, Vitaro, Tremblay, & Lavoie, 2001; Miller & Lynam, 2006; Vitaro, Brendgen, & Tremblay, 2002; Vitaro, et al., 1998). In addition, CFAs suggest that a two-factor model better represents the underlying structure of aggression than does a single factor model (Poulin & Boivin, 2000).

Understanding the relations between psychopathic traits and aggression is important in clarifying conceptual issues in psychopathy, as well as in contributing to evidence for the external validity of child psychopathy dimensions. As mentioned above, in the adult psychopathy literature, there is considerable debate over the centrality of aggression and antisocial behavior in psychopathy. Because one important use of the psychopathy construct is in its prediction of future aggression and violence (Salekin, Rogers, & Sewell, 1996), including antisocial features in psychopathy measures could blur the distinction between the two (Skeem & Cooke, 2010). One potential benefit of using the APSD is that its items are less contaminated by aggression than are most other psychopathy measures (e.g., PCL-R).

Previous studies have suggested the existence of significant associations between psychopathy and aggression in youth and adults. In children, several investigators have reported positive correlations between overall aggression and psychopathy subscales such as I/CP (Frick, et al., 1994) and CU (Christian, et al., 1997). In contrast, studies examining the relationship between overall psychopathy scores and reactive and proactive aggression suggested that global psychopathy was either significantly related to both reactive and proactive aggression (Kimonis, Frick, Fazekas, & Loney, 2006), or related only to proactive

aggression (Raine et al., 2006). These findings mirror those from the adult literature, some of which indicate that compared with non-psychopaths, psychopaths are more likely to use instrumental than reactive aggression (Cornell et al., 1996; Porter & Woodworth, 2007; Woodworth & Porter, 2002).

Prior studies have revealed associations between psychopathy dimensions (e.g., Narcissism and CU) and reactive and proactive aggression, although findings have been mixed. Narcissism was positively associated with both reactive and proactive aggression among aggressive children (Barry, Grafeman, Adler, & Pickard, 2007; Barry et al., 2007) and adults (Baumeister, Bushman, & Campbell, 2000; Bushman & Baumeister, 1998; Reidy, Foster, & Zeichner, 2010) in some studies, whereas in others it was associated only with reactive aggression (Bettencourt, Talley, Benjamin, & Valetine, 2006). CU was associated with higher levels of both reactive and proactive aggression among children with conduct problems (Frick, et al., 2003) but was uniquely associated with proactive aggression in preschoolers (Kimonis, et al., 2006).

In summary, although preliminary findings suggest associations between psychopathic traits and aggression, most studies have relied either on total scores on psychopathy, aggression, or both, or studied the association between only one component of psychopathy and reactive versus proactive aggression. These methodological approaches do not elucidate potentially important differences in the relations between the multiple dimensions of psychopathy and the two forms of aggression. In addition, because moderate-to-high correlations have been reported between the Impulsivity and Narcissism facets of psychopathy (e.g., $r = 0.66$; Frick, et al., 2000) as well as between reactive and proactive aggression (e.g., $r = 0.70$ in a meta-analysis; Polman, Orobio de Castro, Koops, van Boxtel, & Merk, 2007), the use of simple regression models, as used in most studies,

poses potential problems of multicollinarity and may fail to capture the unique variance attributable to each of the psychopathy and aggression dimensions. Finally, little attention has been paid to establishing the external validity of the APSD Impulsivity factor. As a consequence, its implications for childhood psychopathy, as well as its potential differences from the Narcissism factor (with which it is combined in the two factor model of child psychopathy) are unclear.

The Present Study and Hypotheses

In contrast to previous investigations, the present study analyzed the relations between the three psychopathic trait dimensions and the two forms of aggression in children using a structural equation modeling (SEM) framework, which is advantageous for several reasons. First, it allows for the substantial correlations between reactive and proactive aggression and among the three psychopathy factors. Second, instead of using summary composite or factor scores for each APSD factor obtained from CFA, we conducted the analyses of external validity within the same SEM model in which the best-fitting measurement model for APSD (i.e., the three-factor model) was specified. Third, given that we are examining the relations with the latent psychopathy dimensions, we are able to estimate these relations free of measurement error. Fourth, we conducted these analyses in both clinic-referred and non-referred samples, both simultaneously in a multiple group analysis and subsequently in each group separately, allowing us to statistically compare the replicability of the relations between the psychopathic traits and reactive and proactive aggression across both samples. Fifth, the statistical power to detect differential relations of reactive and proactive aggression with the three psychopathy dimensions is increased when these two samples are pooled, by constraining the equivalent measurement and structural parameters of the psychopathy measure across sample. Our analyses thus will provide a

stronger test of the external validity of the three-factor model of psychopathic traits in children than has heretofore been available.

Based on the current literature and theories of psychopathy and aggression, we hypothesize that: (1) Narcissism will be positively related to both reactive and proactive aggression, but that the magnitude of the association will be stronger for reactive than proactive aggression; (2) CU will be positively related to proactive aggression but not reactive aggression; and (3) Impulsivity will be positively related to reactive aggression but negatively related to proactive aggression.

Method

Participants and procedures

Clinic-referred sample. Participants (N = 382) were recruited from the Center for Learning and Attention Deficit Disorders (CLADD) at the Emory University School of Medicine in Atlanta, Georgia and from private practices in Tucson, Arizona. Both sites provided the referred children with assessment, treatment, or both, for childhood disruptive behavior disorders, including Attention Deficit Hyperactivity Disorder (ADHD), Oppositional Defiant Disorder (ODD), and Conduct Disorder (CD), as well as Learning Disorders (LD). The final sample size, which consisted of participants with complete parent-ratings of psychopathic traits and aggression, was 350. The sample comprised 65% males (N = 228) and 35% girls (N = 122). The age range was 4-18 years old (M = 10.7, SD = 3.6). The ethnicity was 78% Caucasian American, 10% African American, 2% Hispanic, 1% Asian, and 9% mixed ethnicity. The exclusion criteria for the study included diagnosis of autistic disorder, traumatic brain injury, or neurological conditions (e.g., epilepsy), as well as IQ < 75.

Community sample. Participants were 838 twins pairs recruited as part of the Georgia Twin Registry (GTR), a community-based twin sample aged 4 to 17 years old (M = 10.6 years, SD = 3.2 years). The GTR was initiated in 1992-1993, during which 1,567 of the 5,620 contacted twin families joined the registry and returned demographic and zygosity information. Of these 1,567 families to whom a second set of questionnaires (including measures used for the present study) were sent, 885 families returned the questionnaires, 838 of which were complete. Of these 838 twin pairs, 1,579 individual twins had complete ratings on both psychopathic traits and aggression and were included in the final analyses. Forty nine percent of the sample was male and 82% was Caucasian, 11% African American, 1% Hispanic, and 6% mixed ethnicity. The sample consisted of 389 monozygotic twin pairs (MZ; 46%) and 449 dizygotic twin pairs (DZ; 54%). The composition of the DZ pairs was 115 (26%) same-sex males, 119 (26%) same-sex females, and 215 (48%) opposite-sex pairs. Twin zygosity (MZ vs. DZ twin status) was determined from parental reports of the physical similarity of the twins. Questionnaire measures of zygosity have been found to be highly accurate in ascertaining actual zygosity when compared with DNA polymorphisms (R. W. Jackson, Sneider, Davis, & Treiber, 2001).

Measures

The measures described here were administered to the parents of the children in both the clinic-referred and non-referred twin samples.

Reactive-Proactive Aggression Scale. We used a 12-item rating scale of aggression developed by Dodge and Coie (1987), which was originally a teacher-rating scale. Of these 12 items, three measure reactive aggression (i.e., when teased, strikes back; blames others in fights; overreacts angrily to accidents) and three measure proactive aggression (i.e., uses physical force to dominate; gets others to gang up on a peer; threatens and bullies

others). The remaining six items were not included in this two-factor model of reactive vs. proactive aggression (Dodge & Coie, 1987). The internal consistency of the subscales was supported by high alpha coefficients (clinic-referred sample: 0.82 and 0.78; community sample: 0.78 and 0.72, for reactive and proactive aggression respectively).

Antisocial Process Screening Device (APSD). The APSD is the first major measure to apply the multidimensional construct of psychopathy to youth. It is a 20-item 3-point rating scale adapted from the PCL-R to detect psychopathic tendencies in children (Frick & Hare, 2001). In the present study, we obtained parent (mother and/or father) ratings of the APSD on a 5-point scale (from "not at all" to "very well" based on how descriptive they are of the child). Based on previous analyses of the present samples (Dong & Waldman, 2010 June) as well as recommendations in the literature (Frick, et al., 2000), we adopted three subscales for APSD: Narcissism (7 items), CU (6 items), and Impulsivity (5 items). Moderate-to-high internal consistencies were found for clinic-referred (0.80, 0.63, and 0.72) and community samples (0.84, 0.61, and 0.78) for these three subscales, respectively.

Data analyses

All analyses were performed using SEM in Mplus 6 (Muthén & Muthén, 1998-2010). A diagonally weighted least squares (WLSMV) estimator was used to deal with ordinal variables (i.e., APSD items), as recommended in the literature (Flora & Curran, 2004). The *cluster* option was used to account for the nesting of the data, given that both clinic-referred and community samples comprised siblings or twin pairs within families. The *difftest* option under WLSMV was used to compare hierarchically nested models using a χ^2 difference test. Model fits were evaluated using several fit indices: (1) the χ^2 value and its associated degrees of freedom (*df*) and *p* value[1]; (2) The Tucker-Lewis index (TLI), for which values between

0.90 - 0.95 indicate acceptable fit (Bentler, 1990), and values ≥ 0.95 indicate excellent fit (Hu & Bentler, 1999); and (3) Root Mean Square Error of Approximation (RMSEA), for which values ≤ 0.08 indicate adequate fit, and values ≤ 0.05 indicate good fit (Browne & Cudek, 1993). The three APSD factors (Narcissism, CU, and Impulsivity) served as correlated dependent variables and reactive and proactive aggression served as correlated predictor variables. Sex, age, age^2, sex*age, and sex*age^2 were entered as covariates and only those covariates that were at least marginally significant ($p < .10$) and their constituent lower-order terms remained in the final model. Reactive and proactive aggression scores were obtained by summing the relevant item scores for each dimension.

Multiple group analysis. In a previous paper (Dong & Waldman, 2010 June), we conducted multiple group CFAs that allowed for simultaneous analyses of the underlying structure of the APSD in both samples. A high degree of factorial invariance between the clinic-referred and community samples was established in these analyses. Specifically, all factor loadings (except for 5 items that were not invariant across samples), item thresholds, factor variances, and factor correlations could be equated across the samples without any significant decrement in model fit as compared with less restricted models in which these parameters were unconstrained. Hence, these constraints were incorporated in our first baseline model, in which the APSD factor means were specified to equal zero and the APSD item variances were fixed at one.

In Table 1, we present a summary of the models of interest that were contrasted against the baseline model to test our substantive hypotheses of interest. The regression coefficients for the relations of each of the psychopathy factors with the reactive and proactive aggression scales were next constrained to be equal across the clinic-referred and community twin samples. These models with increasing numbers of constraints were

compared in succession with the first baseline model using χ^2 difference tests. These analyses tested whether reactive and proactive aggression were similarly related to the psychopathy dimensions across samples, which proved to be the case for each of the 6 aggression - psychopathic trait regression coefficients. A second baseline model was then constructed in which the regression coefficients for the relations of reactive and proactive aggression with each of the psychopathy factors across samples were equated, in addition to those constraints imposed in the first baseline model. The regression coefficients for reactive and proactive aggression with each of the psychopathy dimensions were then equated in turn, and these models were compared with the second baseline model using χ^2 difference tests. These analyses tested whether the relations of each psychopathy dimension to reactive and proactive aggression were similar (i.e., were equatable) or whether they differed. These analyses were conducted using data from both samples combined to increase statistical power, as well as in each sample individually to check for the consistency of these relations across the two samples.

Results

We first tested the equatability of the regression coefficients for the relations between reactive and proactive aggression and the three APSD psychopathy dimensions (i.e., Narcissism, CU, Impulsivity) across the clinic-referred and the community twin samples. We did so to determine whether a single value could be used to characterize each of these six regression coefficients within the combined sample. We next tested whether reactive and proactive aggression were differentially related to each of the three APSD dimensions. Based on previous tests of factorial invariance between the clinic-referred and community twin samples (Dong & Waldman, 2010 June), all equatable measurement and structural parameters (e.g., factor loadings, item thresholds, factor variances, and factor correlations)

were constrained to be equal across the two samples in Baseline Model 1 and all of the subsequent models listed under *Combined sample* in Table 1. We used Baseline Model 1 to test whether each of the regression coefficients for the three APSD dimensions and reactive and proactive aggression were equatable across the two samples both separately (Models 1, 2, 4, 5, 7, 8 under Baseline Model 1) and jointly (Models 3, 6, 9 under Baseline Model 1). As shown in Table 1, results of these tests revealed that all of these regression coefficients could be equated across the clinic-referred and community samples.

Given these results, we constructed Baseline Model 2 in which all of the regression coefficients between reactive and proactive aggression and the three APSD psychopathy dimensions were constrained across the two samples, in addition to the model constraints already imposed in Baseline Model 1. We then tested whether the regression coefficients for each of the three APSD dimensions with reactive and proactive aggression could be equated (models 1-3 under Baseline Model 2 in Table 1). The results showed that none of the regression coefficients for reactive and proactive aggression with any of the three psychopathic trait dimensions was equatable (χ^2_{diff} = 5.04, df = 1, p = .025 for Narcissism; χ^2_{diff} = 6.06, df = 1, p = .014 for CU; and χ^2_{diff} = 65.24, df = 1, p < .001 for Impulsivity), indicating statistically significant differences in the association between each psychopathy dimension and reactive and proactive aggression.

We then examined whether the regression coefficients for reactive and proactive aggression were equatable in relation to the each of the three APSD dimensions in the two samples separately (see Table 1 under *Clinic-referred sample* and *Community twin sample*). In the clinic-referred sample, the relation between reactive aggression and Narcissism did not differ significantly from the relation between proactive aggression and Narcissism (χ^2_{diff} = 1.42, df = 1, p = .234), whereas for CU and Impulsivity the regression coefficients for reactive

aggression differed significantly from those for proactive aggression (χ^2_{diff}= 5.15, *df*= 1, *p* = .023 for CU; χ^2_{diff}= 18.97, *df*= 1, *p* < .001 for Impulsivity). In the community twin sample, the regression coefficients for reactive and proactive aggression in relation to Narcissism and CU did not differ significantly (χ^2_{diff}= 1.40, *df*= 1, *p* = .237 for Narcissism and χ^2_{diff}= 2.39, *df* = 1, *p* = .122 for CU), whereas these coefficients did differ for Impulsivity (χ^2_{diff}= 40.27, *df*= 1, *p* < .001).

In Table 2 we present the unstandardized regression coefficients for the associations between the three psychopathy dimensions and reactive and proactive aggression in the combined sample, as well as in the clinic-referred and community twin samples separately. In the combined sample, all six regression coefficients were significant, although Narcissism was more strongly associated with reactive than proactive aggression (*b* = 0.24, *p* < .001 and *b* = 0.18, *p* < .001, respectively), Impulsivity was much more strongly related to reactive than proactive aggression (*b* = 0.34, *p* < .001 and *b* = 0.06, *p* = .008, respectively), whereas CU was more strongly associated with proactive than reactive aggression (*b* = 0.13, *p* < .001 and *b* = 0.05, *p* < .001, respectively). In addition, as shown in Table 2, these associations in the clinic-referred and community twin samples were highly consistent with each other and with those in the combined sample. Given the high degree of similarity between the regression coefficients in the clinic-referred and community samples, the differences in significance for the tests of the equatability of the reactive and proactive aggression regression coefficients between the combined sample and the two sub-samples are most likely due to increased statistical power in the combined sample. Path diagrams showing standardized regression coefficients for the relations between the three dimensions of psychopathy and reactive and proactive aggression in the combined sample are shown in Figure 1.

Discussion

In the present study, we examined the relations between three psychopathic trait dimensions (i.e., Narcissism, CU, and Impulsivity) and two forms of aggression (i.e., reactive and proactive aggression) in two independent child samples. Pooling the two samples by constraining all the equatable parameters using SEM, we found that Narcissism was related slightly more to reactive than proactive aggression, CU was related more to proactive than reactive aggression, and Impulsivity was related substantially more to reactive than proactive aggression. Analyses conducted in each individual sample were consistent with the patterns and magnitudes of differential associations found in the combined sample.

The present findings support our hypotheses that the three psychopathic trait dimensions are associated with reactive and proactive aggression in significantly different ways and are broadly consistent with those of prior studies (e.g., Barry, et al., 2007; Kimonis, et al., 2006). The fact that Narcissism, CU, and Impulsivity differentially relate to distinct facets of aggression contributes to evidence for the external validity of the three-factor model of psychopathic traits in children. It provides evidence for the construct validity of the Impulsivity factor in particular, given the significantly stronger association between Impulsivity and reactive aggression which was robust across samples. The initial impetus for the three-factor model was the posited need to separate psychopathy-related Narcissism from Impulsivity (Frick, et al., 2000). Our findings support the legitimacy of separating Impulsivity from Narcissism as factors, dovetailing with our previous evidence for the internal validity of the three-factor model as the best-fitting model for the APSD.

More broadly, our findings indicate that exclusive reliance on total scores of child psychopathy measures in research and clinical practice may be misleading, as such reliance will obscure markedly differential relations between psychopathy subdimensions and

aggression. In addition, they suggest that investigations of the etiology of child psychopathy will need to account for the differential causal processes underpinning the three dimensions examined here.

It also is worth reiterating the methodological strengths of the present study. By using a SEM approach in lieu of standard multiple regression as used in previous studies, we were able to directly compare the differences in the strength of associations between the three psychopathic trait dimensions and the two types of aggression. Also, the present analyses allowed for the substantial correlations between reactive and proactive aggression and among the three psychopathy factors. Moreover, the present findings drew on a large community sample in addition to a clinic-referred sample. Inclusion of a normative sample enabled direct comparison with the clinical sample and avoided potential biases in obtaining high covariation between psychopathic traits and aggression.

Several limitations should be borne in mind when interpreting the results. First, due to the cross-sectional nature of this study, psychopathic traits and aggressive behaviors were assessed concurrently, so we could not test the temporal relations between psychopathic traits and reactive and proactive aggression. Psychopathic tendencies in children may precede their manifestations in the form of aggression, but prospective studies are needed to test this hypothesis. Second, we relied solely on parent reports of the level of both psychopathic traits and aggression. Future studies should obtain and combine parent-ratings, teacher-ratings, and self-reports to minimize potential rater bias. Third, we examined only two theoretically important correlates of psychopathy dimensions, namely, reactive and proactive aggression. In future research, it will be necessary to expand the nomological network surrounding childhood psychopathy to incorporate other theoretically relevant correlates, including measures of personality (e.g., behavioral activation and

inhibition), psychopathology (e.g., ADHD and CD), laboratory findings (e.g., passive avoidance learning, perceptions of facial displays of emotion), biochemical and molecular genetics findings (e.g., dopamine and serotonin metabolites and genetic variants), and brain imaging results (e.g., amygdala activation in response to threat). Such investigations should provide more extensive and theoretically informative tests of the three factor model of childhood psychopathy and clarify the differential correlates of these dimensions.

General Discussion

The purpose of the current thesis was to examine the underlying structure of psychopathic traits in children. In particular, the aims of this thesis were to better establish the internal and external validity of the three-factor model of psychopathic traits (i.e., Narcissism, Callous-unemotional traits, Impulsivity) in children. In Study 1, confirmatory factor analyses of the APSD conducted comparing five nested factor models in six subgroups / samples (i.e., MZ twins vs. DZ twins, clinic-referred vs. non-referred community children, boys vs. girls) uniformly indicated that the three-factor model had the best fit over alternative models. Using the best-fitting three-factor model, full measurement invariance was established across zygosity and partial measurement invariance was established across sample type and gender. Structural invariance was established for factor variances and correlations, but not factor means, across zygosity, sample type, and gender, reflecting true group differences in ratings of psychopathic traits. In Study 2, relations between the dimensions from the best-fitting, three-factor model and reactive and proactive aggression were examined. As expected, dimensions of psychopathic traits and aggression demonstrated differential associations, such that Narcissism was slightly more associated with reactive than proactive aggression, CU was more associated with proactive than reactive

aggression, and Impulsivity was substantially more associated with reactive than proactive aggression.

Results from both studies strongly support the construct validity of the three-factor model of psychopathic traits in children, given that the factor structure is stable across samples/ subgroups, the measurement characteristics of the majority of items are generalizable to other groups, and the three dimensions are distinguishable by their relations with relevant external variables like different forms of aggression. The three-factor model of the APSD in children is in fact consistent with a similar three-factor model found in adults for the PCL-R (Cooke & Michie, 2001).

Future research following from the two studies reported here should include examination of the relations between psychopathic trait dimensions and relevant personality dimensions and childhood psychopathology (e.g., anxiety, depression, attention deficit hyperactivity disorder, conduct disorder) to further establish the construct validity of the three-factor model. Behavior genetic analyses of the genetic and environmental influences underlying each of the three psychopathic trait dimensions and their covariation can also distinguish the three dimensions by revealing both common and distinct etiological processes and by explicitly testing whether the three psychopathic trait dimensions share any common etiological mechanisms. Further understanding of the development of psychopathy would emerge from longitudinal designs that would allow researchers to address issues that include whether the factor structure is stable across development, elucidating the normative developmental trajectory of psychopathic traits, and revealing both risk and protective factors associated with changes in psychopathic traits over time.

Given that Narcissism, CU, and Impulsivity are distinct dimensions of psychopathic traits in children, future studies investigating the associations between psychopathy and

other relevant constructs should avoid using global scores of psychopathy because potentially differential relations between the distinct psychopathic traits and the relevant constructs may be obscured. Studies investigating the etiology of psychopathy will also need to search for potentially distinct etiological mechanisms and processes underlying each psychopathic trait dimension. Indeed, a recent meta-analysis suggests that the most studied candidate gene polymorphisms for antisocial behavior (*MAOA-uVNTR*, *5HTTLPR*) only showed small or no effects on the broad construct of antisocial behavior (Ficks & Waldman, 2011 June). Reducing the heterogeneity in antisocial behavior by examining psychopathic traits in antisocial individuals and, more importantly, by using the individual component dimensions of psychopathy may help to find stronger associations with both etiological factors such as genetic variations and phenotypic variables such as other forms of psychopathology.

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Footnotes

[1] The χ^2 value and associated *df* were routinely reported in the results but not used in model fit evaluation.

Study 1- Table 1

Results from the Confirmatory Factor Analyses of the APSD items

By Zygosity: Sample 1 (Community twin sample)

	All twins				MZ twins				DZ twins			
Models	χ^2	*df*	TLI	RMSEA	χ^2	*df*	TLI	RMSEA	χ^2	*df*	TLI	RMSEA
1g	2043	135	0.724	0.092	862	135	0.757	0.083	1237	135	0.740	0.095
2o	1840	135	0.754	0.087	790	135	0.781	0.079	1221	135	0.744	0.094
2r	883	134	0.891	0.058	430	134	0.900	0.053	584	134	0.893	0.061
3o	4353	135	0.391	0.136	1860	135	0.424	0.128	2775	135	0.377	0.147
3r	812	132	0.900	0.055	401	132	0.908	0.051	546	132	0.900	0.059

By Sample Type: Sample 2 (Clinic-referred vs. Controls)

	Combined				Clinic-referred				Non-referred Controls			
Models	χ^2	*df*	TLI	RMSEA	χ^2	*df*	TLI	RMSEA	χ^2	*df*	TLI	RMSEA
1g	478	135	0.880	0.071	674	135	0.835	0.107	228	135	0.910	0.067
2o	711	135	0.798	0.092	697	135	0.828	0.109	582	135	0.568	0.147
2r	340	134	0.927	0.055	432	134	0.908	0.080	208	134	0.928	0.060
3o	1912	135	0.376	0.162	2008	135	0.428	0.199	920	135	0.242	0.195
3r	317	132	0.934	0.053	403	132	0.915	0.077	196	132	0.937	0.056

By Sex:

	Sample 1 (Community twin sample)								*Sample 2 (clinic-referred vs. controls)*							
	Males				Females				Males				Females			
Models	χ^2	*df*	TLI	RMSEA	χ^2	*df*	TLI	RMSEA	χ^2	*df*	TLI	RMSEA	χ^2	*df*	TLI	RMSEA
1g	1143	135	0.726	0.095	1033	135	0.736	0.088	642	135	0.823	0.116	411	135	0.870	0.101
2o	1055	135	0.750	0.091	968	135	0.755	0.085	750	135	0.785	0.128	679	135	0.745	0.142
2r	531	134	0.891	0.060	527	134	0.884	0.059	371	134	0.917	0.079	305	134	0.919	0.080
3o	2392	135	0.387	0.142	2197	135	0.393	0.134	1870	135	0.393	0.214	1426	135	0.394	0.219
3r	492	132	0.900	0.057	505	132	0.888	0.057	341	132	0.925	0.075	291	132	0.924	0.078

Notes. 1g=1 general factor model; 2o=2- factor orthogonal model; 2r=2-factor correlated model; 3o=3-factor orthogonal model; 3r=3-factor correlated model. For all chi-square values, $p<0.001$. TLI=Tucker-Lewis Index; RMSEA=Root Mean Square Error of Approximation.

Study 1-Table 2

Results from testing of nested CFA models

Sample 2: Community twin sample

Models	All twins			MZs			DZs			Male			Female		
	χ^2_{diff}	*df*	*p*	χ^2_{diff}	*df*	*p*	χ^2_{diff}	*df*	*p*	χ^2_{diff}	*df*	P	χ^2_{diff}	*df*	*p*
2r vs. 1	309	1	<.001	122	1	<.001	203	1	<.001	209	1	<.001	135	1	<.001
2r vs. 2o	157	1	<.001	67	1	<.001	104	1	<.001	93	1	<.001	78	1	<.001
3r vs. 1	503	3	<.001	200	3	<.001	324	3	<.001	317	3	<.001	224	3	<.001
3r vs. 3o	769	3	<.001	350	3	<.001	496	2	<.001	434	3	<.001	399	3	<.001
3r vs. 2r	53	2	<.001	19	2	<.001	29	2	<.001	33	2	<.001	23	2	<.001

Sample 1: Clinic-referred vs. Controls

	Combined			Clinic-referred			Controls			Male			Female		
	χ^2_{diff}	*df*	*p*	χ^2_{diff}	*df*	*p*	χ^2_{diff}	*df*	*p*	χ^2_{diff}	*df*	P	χ^2_{diff}	*df*	*p*
2r vs. 1	43	1	<.001	54	1	<.001	17	1	<.001	69	1	<.001	39	1	<.001
2r vs. 2o	83	1	<.001	50	1	<.001	91	1	<.001	66	1	<.001	67	1	<.001
3r vs. 1	74	2	<.001	100	3	<.001	36	3	<.001	121	3	<.001	68	3	<.001
3r vs. 3o	508	3	<.001	365	3	<.001	237	3	<.001	348	3	<.001	282	3	<.001
3r vs. 2r	35	2	<.001	17	2	<.001	17	2	<.001	17	2	<.001	17	2	<.001

Notes. 1g=1 general factor model, 2o=2- factor orthogonal model, 2r=2-factor correlated model, 3o=3-factor orthogonal model, 3r=3-factor correlated model. TLI=Tucker-Lewis Index; RMSEA=Root Mean Square Error of Approximation.

Study 1- Table 3

Results from testing measurement and structural invariance by zygosity and sample type

Model comparisons		χ^2	*df*	*p*	TLI	RMSEA	χ^2_{diff}	*df*	*p*
By Zygosity: Sample 1 (Community twin sample)									
Measurement invariance									
Model 1	Equal forms (congeneric model)	996	315	< .001	0.920	0.051			
Model 2 vs. 1	Equal factor loadings	861	333	<.001	0.941	0.043	17	18	0.525
Model 3 vs. 2	Equal residual variances	824	351	<.001	0.950	0.040	15	18	0.650
Structural invariance									
Model 4 vs. 3	Equal factor variances	824	351	<.001	0.950	0.040	1	3	0.705
Model 5 vs. 4	Equal factor correlations	716	354	<.001	0.962	0.032	2	3	0.503
Model 6 vs. 5	Equal factor means	706	357	<.001	0.964	0.034	9	3	0.033
Model 6' vs. 5	Equate factor mean for CU	716	355	<.001	0.962	0.035	3	1	0.106
By Sample type: Sample 2 (Clinic-referred vs. Controls)									
Measurement invariance									
Model 1	Equal forms (congeneric model)	628	311	<.001	0.933	0.064			
Model 2 vs. 1	Equal factor loadings	600	329	<.001	0.946	0.057	34	18	0.012
Model 2' vs. 1	Equal factor loadings (except item 5)	580	328	<.001	0.949	0.055	22	17	0.178
Model 3 vs. 2'	Equal residual variances*	637	345	<.001	0.944	0.058	69	17	<0.001
Structural invariance									
Model 4 vs. 2'	Equal factor variances	580	328	<.001	0.949	0.055	4	3	0.279
Model 5 vs. 2'	Equal factor correlations	600	331	<.001	0.946	0.057	17	3	0.001
Model 5' vs. 2'	Equal factor correlation (only between Nar and Imp)	578	329	<.001	0.950	0.055	1	1	0.248
Model 6 vs. 5'	Equal factor means	817	332	<.001	0.904	0.076	63	3	<0.001
By Sex: Sample 1(Community twin sample)									
Measurement invariance									
Model 1	Equal forms (congeneric model)	1081	315	<.001	0.907	0.054			
Model 2 vs. 1	Equal factor loadings	967	333	<.001	0.927	0.048	34	18	0.012
Model 2' vs. 1	Equate factor loadings (except item14)	937	332	<.001	0.930	0.047	20	17	0.285

Model 3 vs. 2'	Equal residual variances*	910	349	<.001	0.939	0.044	21	17	0.246
Structural invariance									
Model 4 vs. 2'	Equal factor variances	910	349	<.001	0.939	0.044	2	3	0.501
Model 5 vs. 2'	Equal factor correlations	785	352	<.001	0.953	0.038	1	3	0.725
Model 6 vs. 5	Equal factor means	840	355	<.001	0.948	0.040	27	3	<0.001
Model 6' vs. 5	Equate only mean of Narcissism	774	353	<.001	0.954	0.038	4	1	0.061
By Sex: Sample 2 (Clinic-referred vs. Controls)									
Measurement invariance									
Model 1	Equal forms (congeneric model)	638	315	<.001	0.940	0.065			
Model 2 vs. 1	Equal factor loadings	631	333	<.001	0.947	0.061	40	18	0.002
Model 2' vs. 1	Equal factor loadings (except item 5 and 13)	593	331	<.001	0.953	0.057	18	16	0.339
Model 3 vs. 2'	Equal residual variances*	586	347	<.001	0.959	0.054	23	16	0.122
Structural invariance									
Model 4 vs. 2'	Equal factor variances	586	347	<.001	0.959	0.054	2	3	0.519
Model 5 vs. 2'	Equal factor correlations	543	350	<.001	0.968	0.048	2	3	0.483
Model 6 vs. 5	Equal factor means	605	353	<.001	0.958	0.055	23	3	<0.001
Model 6' vs. 5	Equate only mean of Narcissism	541	351	<.001	0.968	0.047	2	1	0.148

Notes. TLI=Tucker-Lewis Index; RMSEA=Root Mean Square Error of Approximation. Models listed subsequent to the baseline were compared with a less restricted model in $\Delta\chi^2$ difference test. *Residual variances were equated except for items that the loadings were not equatable.

Study 1- Table 4

Factor loadings of the APSD items in each sample and subgroups

APSD Items	Community twin sample (*Sample 1*)				Combined sample (*Sample 2*)			
	MZs	DZs	Male	Females	Controls	Clinic	Males*	Females
	n=780	n=901	n=825	n=856	n=153	n=350	n=295	n=206
Narcissism								
16. Thinks more important	0.741		0.737		0.661	0.830	0.788	
8. Brags excessively	0.583		0.578		0.646	0.709	0.710	
10. Uses or "cons" others	0.762		0.761		0.743	0.818	0.823	
14. Can be charming	0.791		**0.725**	**0.870**	0.676	0.835	0.823	
11. Teases others	0.655		0.665		0.508	0.727	0.676	
15. Becomes angry when corrected	0.680		0.677		0.663	0.763	0.753	
5. Emotions seem shallow	0.819		0.819		**0.924**	**0.744**	**0.705**	**0.933**
Callous-unemotional (CU)								
18. Concerned about feelings of others	0.820		0.821		0.784	0.773	0.802	
12. Feels bad or guilty	0.566		0.562		0.560	0.579	0.591	
3. Is concerned about schoolwork	0.327		0.320		0.357	0.359	0.337	
7. Keeps promises	0.698		0.694		0.711	0.735	0.734	
19. Does not show emotions	0.342		0.341		0.229	0.384	0.259	
20. Keeps the same friends	0.389		0.393		0.416	0.465	0.455	
Impulsivity								
4. Acts without thinking	0.763		0.752		0.878	0.862	0.883	
17. Does not plan ahead	0.542		0.538		0.615	0.546	0.558	
13. Engages in risky activities	0.653		0.656		0.531	0.693	**0.772**	**0.500**
1. Blames others for mistakes	0.707		0.708		0.806	0.805	0.801	
9. Gets bored easily	0.597		0.590		0.684	0.594	0.627	
Correlations								
Narcissism with CU	0.487		0.488		**0.774**	**0.505**	0.583	
Narcissism with Impulsivity	0.863		0.872		0.875		0.879	
CU with Impulsivity	0.412		0.403		**0.680**	**0.384**	0.485	

Notes. All statistics reported are standardized. The standardized factor loadings for *Clinic-referred* and *Control* samples under the *Combined sample* are all different because the item residual variances were not equatable across sample type. Bolded statistics were those not equatable across subgroups. *There were 2 missing values on sex in *Sample2*.

Study 2- Table 1

Equatability of the ggression-APSD regression coefficients across and within the clinic-referred and community twin samples

Model	$\chi 2$	*df*	TLI	RMSEA	χ^2_{diff}	*df*
Combined sample						
Baseline Model 1: Constrained equatable measurement and structural parameters across samples	1387.59***	589	0.926	0.048		
1. Reactive Aggression regression coefficient on Narcissism equated across samples	1121.95***	590	0.955	0.029	0.70	1
2. Proactive Aggression regression coefficient on Narcissism equated across samples	1122.92***	590	0.955	0.029	1.46	1
3. Reactive and Proactive Aggression regression coefficient on Narcissism both equated	1118.42***	591	0.955	0.029	1.27	2
4. Reactive Aggression regression coefficient on CU equated across samples	1120.49***	590	0.955	0.029	1.34	1
5. Proactive Aggression regression coefficient with CU equated across samples	1122.98***	590	0.955	0.029	2.27	1
6. Reactive and Proactive Aggression regression coefficient on CU both equated	1098.01***	591	0.957	0.029	0.80	2
7. Reactive Aggression regression coefficient on Impulsivity equated across samples	1122.11***	590	0.955	0.029	1.66	1
8. Proactive Aggression regression coefficient on Impulsivity equated across samples	1123.08***	590	0.955	0.029	1.09	1
9. Reactive and Proactive Aggression regression coefficient on Impulsivity both equated	1117.58***	591	0.955	0.029	1.02	2
Baseline Model 2: Constrained all equatable parameters across samples[a]	1091.81***	595	0.958	0.028		
1. Reactive and Proactive Aggression coefficients constrained equal for Narcissism	1093.45***	596	0.957	0.028	5.04*	1
2. Reactive and Proactive Aggression coefficients constrained equal for CU	1094.49***	596	0.957	0.028	6.06*	1
3. Reactive and Proactive Aggression coefficients constrained equal for Impulsivity	1102.27***	596	0.956	0.028	65.24***	1
Clinic-referred sample						
Baseline Model[b]	104.62***	35	0.950	0.074		
1. Reactive and Proactive Aggression coefficients constrained equal for Narcissism	104.56***	36	0.950	0.074	1.42	1
2. Reactive and Proactive Aggression coefficients constrained equal for CU	104.84***	36	0.950	0.074	5.15*	1

3. Reactive and Proactive Aggression coefficients constrained equal for Impulsivity	105.42***	36	0.950	0.074	**18.97*** **	1
Community twin sample						
Baseline Model[b]	455.82***	73	0.943	0.055		
1. Reactive and Proactive Aggression coefficients constrained equal for Narcissism	461.07***	74	0.943	0.055	1.40	1
2. Reactive and Proactive Aggression coefficients constrained equal for CU	460.90***	74	0.943	0.055	2.39	1
3. Reactive and Proactive Aggression coefficients constrained equal for Impulsivity	468.59***	74	0.942	0.056	**40.27*** **	1

Note. TLI = Tucker-Lewis Index; RMSEA = Root Mean Square Error of Approximation. [a] Compared to Baseline Model 1, Baseline Model 2 imposed additional constraints equating each of the regression coefficients between reactive and proactive aggression and the three APSD dimensions across the clinic-referred and community twin samples. [b] The baseline models for both the clinic-referred and community samples imposed no constraints on any of the regression coefficients for the relations between reactive and proactive aggression and the three APSD dimensions; appropriate sex and age covariates were controlled as explained in the text. Significant $\chi 2_{diff}$ values are in bold.

* $p < .05$. *** $p < .001$.

Study 2- Table 2

Association between reactive and proactive aggression and the three psychopathy dimensions across samples

	Combined sample			Clinic-referred sample			Community sample		
Model results	Narcissism	CU	Impulsivity	Narcissism	CU	Impulsivity	Narcissism	CU	Impulsivity
Reactive aggression	0.24***	0.05***	0.34***	0.23***	0.04	0.31***	0.24***	0.06***	0.34***
Proactive aggression	0.18***	0.13***	0.06**	0.17***	0.16***	0.05	0.19***	0.12***	0.07**
Sex[a]				0.23***	-2.25**	-0.53**	-0.20**	0.41¶	-0.51***
Age				0.17***	-0.50*	0.34	0.07***	0.08**	0.04*
Age^2				0.36**	0.02¶	-0.02***		-0.06**	
Sex*age				-0.02**	0.33*				
Sex*age^2					-0.01¶				

Note. All coefficients shown are unstandardized regression coefficients for the regression of Narcissism, Callous-Unemotional (CU), and Impulsivity on reactive and proactive aggression and the sex and age covariate terms. [a] The regression coefficients for the sex and age covariates were estimated separately in each sample in the multigroup analyses and thus are not presented for the combined sample. The covariates sex, age, age^2, sex*age, and sex*age^2 were tested for significance separately for Narcissism, CU, and Impulsivity prior to reactive and proactive aggression being entered into the model, and the p-values reported in this table are for the step in the analyses at which each covariate was first entered as a predictor. Only significant covariates were retained in the final model.
¶ $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$.

Figure Caption

Figure 1. Path diagram showing standardized regression coefficients between the psychopathy trait dimensions and reactive and proactive aggression

1

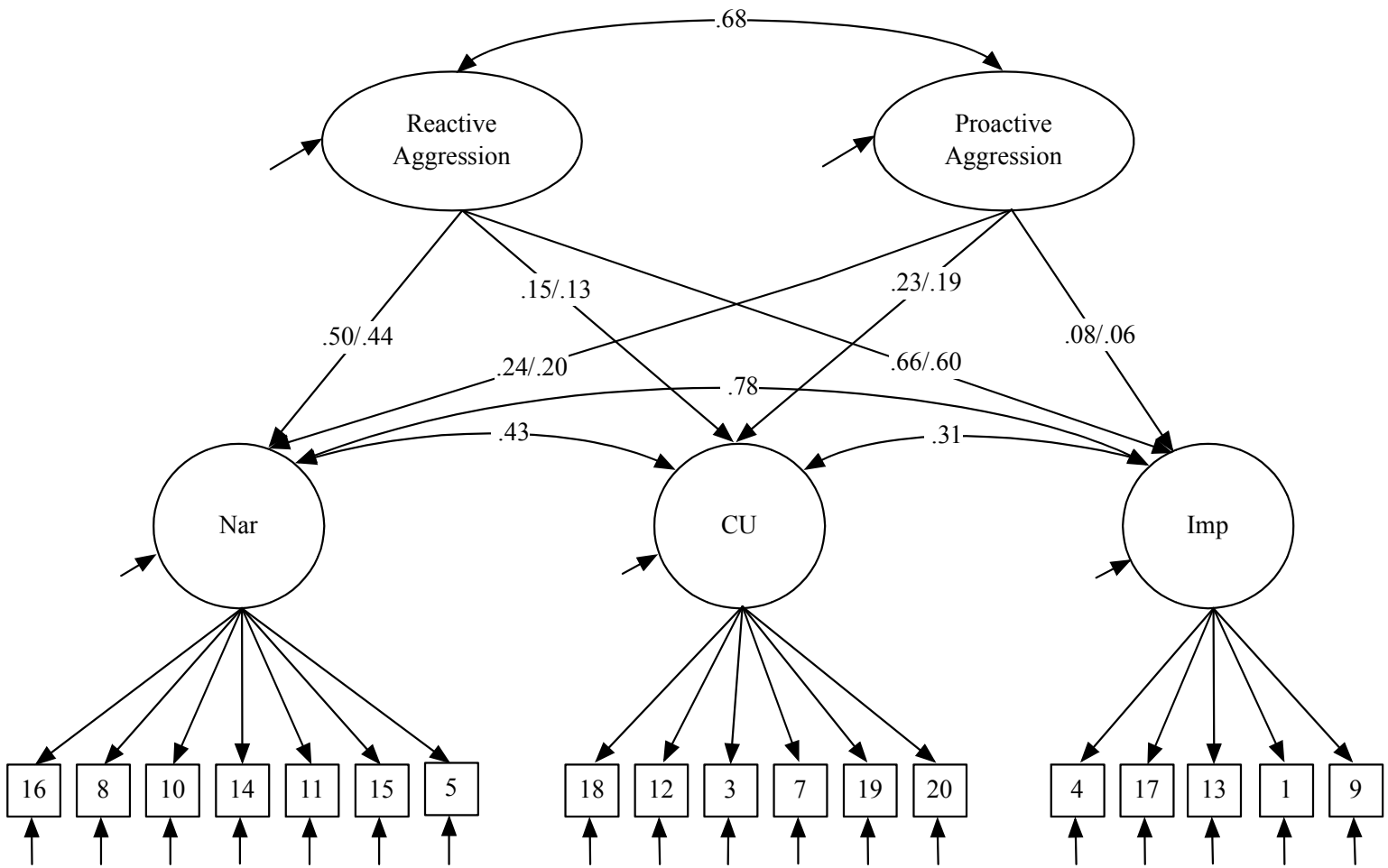

Figure 1. Path diagram showing standardized regression coefficients between the psychopathic trait dimensions and reactive and proactive aggression

Note. Nar=Narcissism, CU=Callous-unemotion, Imp=Impulsivity. All regression coefficients and correlations are standardized, coefficients for clinic-referred group is shown first followed with the twin group coefficients. The regression coefficients for the two groups are not exactly same because reactive and proactive aggression have different variances. Appropriate sex and age covariates were controlled.