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Age-of-onset or Behavioral Sub-types? A Prospective Comparison of Two Approaches to Characterizing the Heterogeneity within Antisocial Behavior

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Abstract

There are two common approaches to sub-typing the well-documented heterogeneity within antisocial behavior: age-of-onset (i.e., childhood-onset versus adolescence-onset; see Moffitt, 1993) and behavioral (i.e., physical aggression versus non-aggressive rule-breaking). These approaches appear to be associated, such that aggression is more characteristic of childhood-onset antisocial behavior whereas rule-breaking is linked to both child- and adolescence-onset antisocial behavior. However, it remains unclear which approach, if either, better explains the heterogeneity within antisocial behavior. We examined this question in a prospective sample of male twins, assessed at the ages of 11, 14, 17, and 24 years. Although the age-of-onset subtypes predicted adult antisocial behavior in the expected direction when analyzed alone, this association dissipated once we controlled for aggression and rule-breaking. Such findings suggest that the behavioral sub-types of antisocial behavior may be a stronger predictor of later antisocial outcomes than is their age-of-onset.

Keywords

antisocial behavior; age-of-onset; aggression; rule-breaking

Antisocial behavior describes actions and attitudes that violate societal norms and the personal or property rights of others. Though typical examples prior to adulthood include running away, vandalism, hurting animals, setting fires, theft, and bullying, the specific manifestation varies markedly from individual to individual (Lahey & Waldman, 2003; Loeber & Stouthamer-Loeber, 1998; Offord & Bennett, 1994; White, Bates, & Buyske, 2001). This behavioral heterogeneity has important long-term consequences, with adult outcomes ranging from conventional, crime-free lifestyles to multiple stints in the prison system (Lahey & Waldman, 2003; Lynam, 1996).

To better understand this heterogeneity, researchers have long advocated the parsing of antisocial behavior into conceptually meaningful dimensions. Moffitt (1993) proposed a dual taxonomy model which is perhaps the most influential approach to characterizing antisocial behavior. She initially theorized that there were two primary subtypes of antisocial behavior that could be distinguished primarily by age-of-onset (Moffitt, 1993). Childhood-

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onset or life-course persistent antisocial behavior was postulated to represent a relatively rare (5–10%), severe, persistent, and often violent condition that began early in life and culminated in negative adult outcomes (e.g., incarceration). Adolescence-onset or adolescence-limited antisocial behavior, by contrast, was thought to represent a relatively normative and transient increase in largely non-violent antisocial behavior that emerged alongside puberty in otherwise healthy adolescents and dissipated as they incorporated themselves into adult roles. Consistent with these typologies, life-course persistent antisocial behavior was thought to stem from neuropsychological and biological risks and their interaction with criminogenic environments, whereas adolescence-limited antisocial behavior was postulated to stem from the "maturity gap" (i.e., a period of development in which societal constraints limit the freedom of otherwise biologically mature individuals) and affiliation with antisocial peers.

Though subsequent research has broadly supported this dual taxonomy (see Moffitt, 2003), and particularly the predictions associated with the life-course persistent group, recent evidence suggests that the adolescence-limited group may have been "under-pathologized" in the original theory (e.g., Roisman et al., 2010). Follow-up studies conducted in early adulthood revealed that adolescence-onset delinquents had not, as was originally proposed, desisted from antisocial behavior and become psychologically healthy (Moffitt, Caspi, Harrington, & Milne, 2002). Instead, they continued to commit low-level crimes such as property offenses, and reported problems with mental health and substance abuse/dependence (Moffitt et al., 2002; Nagin, Farrington, & Moffitt, 1995). They also evidenced an impulsive personality style (i.e., unconventional, spontaneous, and sensation-seeking) (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996), a known risk factor for later antisocial behavior (Krueger, 1999). Similarly, although earlier work did find evidence of neuropsychological and cognitive differences between childhood- and adolescence-onset cases (as reviewed in Moffitt, 2003), more recent psychophysiological and fMRI research (Fairchild, van Goozen, Calder, Stollery, & Goodyer, 2009; Fairchild et al., 2008; Haltigan et al., 2011; Passamonti et al., 2010) found that both child- and adolescent-onset subtypes evidenced marked (and in fact, statistically equivalent) impairments on peripheral physiological dysfunction (e.g., blunted cortisol levels), neurophysiological abnormalities, and neuropsychological deficits. Should the latter findings ultimately prove to be the more replicable ones, it would argue against the claims that adolescence-limited antisocial behavior is limited to adolescence, and moreover, that it is etiologically distinct from life-course persistent antisocial behavior. In other words, "adolescence-limited" antisocial behavior may be quantitatively, rather than qualitatively, distinct from life-course persistent antisocial behavior (Walters, 2010).

Behavioral sub-types of Antisocial Behavior

Age-of-onset is not the only way to characterize the heterogeneity within antisocial behavior, however. Research has also focused on behavioral sub-types of antisocial behavior: namely, physically aggressive (e.g., physically attacking others, bullying) and non-aggressive rule-breaking (e.g., truancy, theft, vandalism) behaviors. Factor analytic studies support this distinction, with meta-analytic results indicating that there are at least two moderately correlated antisocial factors: an "overt" or physically aggressive/oppositional factor and a "covert" or non-aggressive/rule-breaking factor (Frick et al., 1993; Loeber & Schmaling, 1985). Indeed, this general pattern has been observed in both empirically-derived behavioral rating scales, such as Achenbach's Child Behavior Checklist (CBCL), and in factor analyses of conduct disorder and oppositional defiant disorder symptoms (Tackett, Krueger, Iacono, & McGue, 2005; Tackett, Krueger, Sawyer, & Graetz, 2003).

Aggression and rule-breaking also appear to have markedly different developmental trajectories (Tremblay, 2010). The peak frequency in physical aggression is between ages 2 and 4, with the majority of preschoolers engaging in some form of physical aggression (Côté, Vaillancourt, Barker, Nagin, & Tremblay, 2007; Tremblay, 2010). Mean frequencies of physically aggressive behaviors then steadily decrease from early childhood through adolescence (Stranger, Achenbach, & Verhulst, 1997; Tremblay, 2003), with the exception of a very small group (roughly 5% of individuals, almost exclusively males) who continue to aggress at relatively high rates (Tremblay, 2003). The status and property violations that comprise the rule-breaking sub-type, by contrast, are relatively rare during childhood, and increase dramatically over the course of adolescence (Barker et al., 2007; Bongers, Koot, van der Ende, & Verhulst, 2004), only to decrease again in adulthood. As summarized by Tremblay (2010), these clear developmental differences “make it hard to understand why diagnostic categories, developmental theories, etiological studies, and studies meant to test preventive and corrective interventions aggregate physical violence and theft assessments” (p. 351).

Perhaps not surprisingly then, there is now a burgeoning literature supporting personological and cognitive distinctions between physically aggressive and rule-breaking forms of antisocial behavior. Emotion dysfunction appear to be particularly characteristic of physical aggression (Burt & Donnellan, 2008; Burt & Larson, 2007; DeMarte, 2008; Pardini, Lochman, & Frick, 2003), whereas impulsivity appears to be more strongly associated with rule-breaking (Burt & Donnellan, 2008; DeMarte, 2008). Other studies have suggested that verbal IQ and executive functions are negatively related to the frequency of aggression but are either unrelated or positively related to the frequency of rule-breaking (Barker et al., 2007; Barker et al., submitted for publication; Hancock, Tapscott, & Hoaken, 2010).

There is also clear evidence of etiological distinctions between the two behavioral sub-types. Physical aggression is more heritable than is rule-breaking (i.e., genetic influences accounted for 65% versus 48% of the variance, respectively, in a recent meta-analysis; Burt, 2009). Rule-breaking, by contrast, is influenced more by the shared environment than is aggression (i.e., 18% versus 5% of the variance, respectively) (Burt, 2009; Tackett et al., 2005). Consistent with these findings, aggression and rule-breaking also appear to stem from different etiological processes. A family study of 273 clinically-referred probands with Attention Deficit Hyperactivity Disorder and their first-degree biological relatives (n=807) found strong evidence for specificity in the familial aggregation of overt (aggressive) and covert (rule-breaking) Conduct Disorder symptom dimensions (Monuteaux, Fitzmaurice, Blacker, Buka, & Biederman, 2004). Recent work has further suggested that associations with particular candidate genes (namely, $5HT_{2A}$ His452Tyr, DAT1, and COMT) vary across physical aggression and rule-breaking (Burt & Mikolajewski, 2008; Monuteaux, Biederman, Doyle, Mick, & Faraone, 2009), such that $5HT_{2A}$ His452Tyr, DAT1 are uniquely associated with rule-breaking, and COMT is uniquely associated with aggression. In short, there is accumulating evidence that physical aggression and non-aggressive, rule-breaking constitute separable but correlated subtypes of antisocial behavior.

Interestingly, the aggressive/non-aggressive distinction appears to more or less map on to Moffitt’s dual taxonomy (see especially Moffitt, 2003). The median age-of-onset of aggressive behaviors is earlier than that of non-aggressive but delinquent behaviors (Lahey, Loeber, Quay, Frick, & Grimm, 1992). Moreover, as compared to those whose antisocial behavior began in adolescence, those whose onset was prior to age 10 exhibited higher rates of aggressive behaviors in particular (Lahey et al., 1998). In sum, pathological physical aggression appears to be largely characteristic of those with childhood-onset antisocial behavior. Rule-breaking, by contrast, is observed in both child and adolescence-onset antisocial youth; however, because most antisocial adolescents initiated these behaviors after

childhood, and adolescence-onset youth clearly outnumber childhood-onset youth, rule-breaking is thus effectively linked more to adolescence-onset than childhood-onset antisocial behavior.

Despite this well-described link between the behavioral sub-types and the age-of-onset sub-types (Moffitt, 2003; Tremblay, 2010), it is as yet unclear which approach, if either, better predicts antisocial adult outcomes. In particular, extant research (Lahey et al., 1998; Moffitt, 2003) has explicitly assumed that age-of-onset is the more clinically relevant predictor, whereas aggression and rule-breaking are simply a convenient way of indexing the age-of-onset taxonomy. It is equally plausible, however, that the behavioral sub-types themselves are in fact the stronger predictor of adult outcomes, whereas age-of-onset is simply an alternate way of tapping these distinctive behaviors. In other words, the more important issue may be the particular manifestation of antisocial behavior as opposed to the age at which the behaviors began.

We know of only one study that has empirically examined this question to date (Burt & Hopwood, 2010). Burt and Hopwood (2010) evaluated how aggression, rule-breaking, and age-of-onset independently predicted two common correlates of antisocial behavior, anger and alcohol dependency (as measured continuously), in a cross-sectional sample of 1,726 adults in treatment for alcoholism. Analyses revealed that those with childhood-onset Conduct Disorder evidenced far higher rates of aggression and rule-breaking (and particularly aggression) than those with adolescence-onset Conduct Disorder or those without Conduct Disorder, results that are in keeping with the more severe levels of antisocial behavior thought to characterize those with a childhood-onset (Moffitt, 1993). Age-of-onset was also predictive of anger and alcohol dependency, such that those with earlier ages-of-onset evidenced higher rates of these outcomes. Once aggression, rule-breaking, and age-of-onset were simultaneously entered into a regression equation, however, age-of-onset no longer predicted anger or alcohol dependency. By contrast, rule-breaking continued to predict alcohol dependency, while anger was predicted by both aggression and rule-breaking.

Such findings suggest that behavioral sub-types may serve as better predictors of antisocial behavior outcomes than age-of-onset. Constructive replications are certainly needed before any firm conclusions can be drawn, however. The latter point is particularly salient here, as the use of retrospective, cross-sectional data (as in Burt & Hopwood, 2010) reveals little about the longitudinal utility of aggression and rule-breaking as compared to age-of-onset. It thus remains possible that age-of-onset outperforms aggression and rule-breaking when assessed prior to adulthood or longitudinally across childhood, adolescence, and adulthood. Moreover, the well-known difficulties with retrospective recall (Henry, Moffitt, Caspi, Langley, & Silva, 1994; Kahneman, Paul, & Tversky, 1982) may be even more pronounced in the Burt and Hopwood (2010) sample as a result of their alcohol abuse history, a condition with known cognitive consequences. There is thus a clear need to replicate these findings in a prospective sample. Second, the results of Burt & Hopwood (2010) apply to the correlates of anger and alcohol dependency rather than to adult antisocial behavior per se. There is thus a need to contrast the predictive power of the age-of-onset and behavioral sub-types as they relate to antisocial behavior in adulthood. The goal of the current study was to do just this, evaluating whether the behavioral dimensions of physical aggression and rule-breaking have additional predictive utility over and above of Moffitt's (1993) age-of onset taxonomy in a sample of male twins assessed at ages 11, 14, 17, and 24.

Methods

PARTICIPANTS

The sample was drawn from participants in the ongoing and longitudinal Minnesota Twin Family Study (MTFS). Detailed information regarding the design, recruitment procedures, and participation rates of the MTFS has been provided elsewhere (Iacono, Carlson, Taylor, Elkins, & McGue, 1999). The age 11 intake sample of same-sex male twins consisted of 755 reared-together twins (67% monozygotic). Twins were assessed again approximately every 3–4 years, with the most recent assessment occurring at age 24. For the current study, we will focus on the age 11, 14, 17, and 24 assessments. The retention rate was quite good, with a total of 697 (93%), 614 (82%), and 633 (85%) twins completing the age 14, age 17, and age 24 assessments, respectively. Those who completed at least one of the three follow-up assessments (n=740; 98%) reported equivalent number of Conduct Disorder symptoms at age 11 (overall, and after partitioned into aggressive and rule-breaking components as described below) as compared to those who participated only at intake ($p \geq .80$; Cohen's d effect sizes range from .03 to .07).

MEASURES

The current study made use of several indices of antisocial behavior, each of which are described below in more detail: 1) age 11 and 14 diagnoses of Conduct Disorder (CD; or the criterion A symptoms of Antisocial Personality Disorder (ASPD)), 2) age 17 symptom count variables of CD partitioned into aggressive and rule-breaking symptom dimensions, and 3) an age 24 symptom count of the adult or criterion C symptoms of ASPD (referred to as Adult Antisocial Behavior herein; AAB). The latter was used as our outcome variable. The former were all used as predictor variables (as described below). To adjust for positive skew, all symptom count variables were log-transformed prior to analysis.

Participants were assessed for lifetime DSM-III-R (the current manual at the onset of the study) CD at their age 11 and 17 visits, and lifetime DSM-IV AAB at their age 24 visit. At their age 14 visit, they were assessed for DSM-III-R CD symptoms present since their age 11 assessment. Participants' mothers reported on lifetime DSM-III-R CD at their children's age 11 assessment, and CD present since age 11 at their children's age 14 assessment. Mothers reported simultaneously on symptom presence in both twins, while twins reported only on themselves. Both informants also reported on the age-of-onset for each endorsed symptom of CD.

All assessments were conducted in-person by trained bachelors- and masters-level interviewers. Within a given family, each family member was interviewed by a different interviewer. At ages 11 and 14, the Diagnostic Interview for Children and Adolescents-Revised (DICA-R) (Reich, 2000; Welner, Reich, Herjanic, Jung, & Amado, 1987) was administered to the twins and their mothers. At ages 17 and 24, the Structured Clinical Interview for personality disorders (SCID-II) (Spitzer, Williams, Gibbon, & First, 1987) was administered to the twins. Supplementary probes and questions were added to all interviews to ensure complete coverage of each symptom and to ensure CD symptom assessments were comparable across the DICA-R and SCID-II interviews. Of the 13 possible symptoms of DSM-III-R CD, only symptom 9 ("has forced someone into sexual activity with him or her") was not assessed.

Following the interview, a clinical case conference was held in which the evidence for every symptom was discussed by at least two advanced clinical psychology doctoral students (neither of whom conducted the clinical interview). The inter-rater reliability of the consensus process was good, with a kappa of 0.79 for diagnoses of CD. Although AAB does not constitute a DSM diagnosis (i.e., ASPD diagnoses require at least three Criterion A

symptoms of Conduct Disorder as well), if either 3 or 4 symptoms are used to define a "diagnosis" of AAB, the kappa reliability exceeds .78.

Computer algorithms were used to sum the number of CD and AAB symptoms, respectively, and to create diagnoses of CD. At ages 11 and 14, we used a "best estimate" approach to assigning symptoms, such that a symptom was considered present if it was endorsed by either the mother or the child. Symptoms that were endorsed by both mother and child were counted as only one symptom. At all ages, symptoms judged to be definitely present (i.e., they were clinically significant in both severity and frequency by at least one informant) were counted as one full symptom. Symptoms judged to be probably present (i.e., they were clinically significant in either severity or frequency, but not both) were counted as half of a symptom. Only full symptoms were considered when determining diagnoses of CD. Diagnoses were considered "definite" if the participant met full criteria for at least three symptoms of CD. Individuals who met full criteria for only two symptoms were considered "probable" cases of CD.

Aggression (AGG) and rule-breaking (RB) scales—Scales representing lifetime AGG and RB (see Table 1 for a list of items) were constructed to be consistent both with previously-defined CD item subsets (Hopwood et al., 2009;Tackett et al., 2003) and with frequently-used and well-validated questionnaire measures of these constructs (i.e., the Achenbach Child Behavior Checklist and Adult Self-Report; Achenbach & Rescorla, 2001,2003). To ensure that DSM-III-R symptoms of CD could be meaningfully partitioned into AGG and RB symptoms in this way, we conducted a confirmatory factor-analysis with weighted least squares mean and variance adjusted (WLSMV) as implemented in Mplus 6.0 using the age 17 data. We compared the fit of the two-factor (X^2 = 58.372 on 53 *df*, p = .28; TLI = .992; CFI = .993; RMSEA = .013) and one-factor (X^2 = 69.509 on 54 *df*, p = .08; TLI = .981; CFI = .976; RMSEA = .022) models. Although the two factors were correlated .839, the one-factor model provided both a subjectively worse overall absolute fit to the data, as well as a significantly worse fit relative to the two-factor model (ΔX^2 = 9.121 on 1 *df*, p < .01). This suggests that symptoms of CD can be meaningfully divided into AGG and RB symptom dimensions. AGG factor loadings ranged from .49–.86 (the average loading was .70), and RB factor loadings ranged from .53–.87 (the average loading was .74). The resultant AGG and RB symptom count variables were correlated .36 at age 11 and .45 at age 17 (note that these observed correlations are, as one would expect, lower than that between the AGG and RB latent factors).

Age-of-onset—We made use of CD diagnostic status at ages 11 and 14, along with reported symptom age-of-onset, to construct our age-of-onset variable. Childhood-onset, life-course persistent CD (hereafter referred to as LCP) was operationalized as a probable or definite diagnosis of CD at both ages 11 and 14, and the presence of at least one symptom of CD by age 10. Nearly 9% of the total sample (n=67; 8.8%) met the persistence criterion (i.e., CD present at ages 11 and 14). Of these 67 cases, the vast majority (n=60) also reported that at least one CD symptom had been present by age 10 (indeed, 56 reported that at least 2 symptoms had been present by age 10). These 60 participants (or 7.9% of the sample) thus comprised the LCP group. Adolescence-onset or adolescence-limited CD (hereafter referred to as AL) was operationalized as a probable or definite diagnosis of CD at age 14 only (i.e., a probable or definite CD diagnosis was not present at age 11). Those 7 individuals with persistent CD who denied that symptoms were present by age 10 were also included in the AL group. The AL group thus consisted of 134 participants (or 17.7% of the sample). The remainder of the participants were coded as "no CD" (74.3%, n=561).

Of note, the final group primarily contained individuals who never met criteria for definite or probable CD (91%). However, a small portion of individuals assigned to the no CD group

(n=52) had a probable or definite diagnosis of CD at age 11 that had remitted by the age of 14. Although the finding that 46% of childhood-onset youth had "recovered" is inconsistent with Moffitt's (1993) original theory, empirical studies have actually indicated that a pattern of "recovery" in 40–70% of childhood-onset youth is the norm (Barker & Maughan, 2009; Barker, Oliver, & Maughan, 2010; Nagin & Tremblay, 1999; van Domburgh, Loeber, Bezemer, Stallings, & Stouthamer-Loeber, 2009). To maximize statistical power and the representativeness of our data, these childhood-limited cases were included in our primary analyses. We also re-ran our analyses without those cases to ensure that they did not unduly influence our findings (they did not).

ANALYSES

We first compared mean symptom counts of AGG and RB at ages 11 and 17, as well as the AAB symptom count at age 24, across age-of-onset groupings. This allowed us to evaluate whether, as expected, LCP youth evidenced higher rates of antisocial behavior as compared to those with AL or no CD. For our final analyses, we conducted a progressive series of moderated regressions with AAB at age 24 as our dependent variable. All regression analyses were conducted using Multilevel Linear Modeling (MLM) in SPSS 18.0 (Norušis, 2007) to account for the non-independence of observations within families while maximizing statistical power. In particular, because twins are nested within families, our data have a two-level structure with the adolescent as the lower-level unit and the family as the upper-level unit. Predictor variables could conceivably be measured at either the child-level (e.g., AGG symptom count) or the family-level.

To test for age-of-onset group differences in a meaningful way (and avoid assumptions of linearity), it was necessary to employ contrast codes for the regression analyses (J. Cohen, Cohen, West, & Aiken, 2003). For the first contrast code, no CD was coded as a 2 and LCP and AL cases were both coded as −1. For the second contrast code, no CD was coded a 0, AL was coded −1, and LCP was coded as 1. When both contrast codes are in the model, the coefficient for the first contrast code tests whether those with CD (be it AL or LCP) differ from those without CD. The second contrast code (again, when both codes are in the model), tests whether LCP youth differ from AL youth.

In the first model, we entered both contrast codes as fixed effect predictors of AAB at age 24. In the second model, we added the age 17 lifetime AGG and RB symptoms counts as well. In this way, we hoped to determine whether age-of-onset (as represented by contrast code 2) continued to predict AAB at age 24 once AGG and RB were included in the model (and vice versa), as well as clarifying whether AGG and RB yield additional predictive value over and above the age-of-onset taxonomy.

Results

As expected under Moffitt's (1993) taxonomy, all variables varied according to age-of-onset (see Table 2). LCP youth evidenced far higher rates of RB and AGG symptoms at age 11 than did those with AL and those without CD (Cohen's *d* standardized effect sizes ranged from 1.45 – 1.91, indicating that the LCP mean was at the 92–97% of the AL and no CD groups (J. Cohen, 1988)). By age 17, these differences between LCP and AL youth had (not surprisingly) diminished, but were still small to moderate in magnitude (d = .24 – .55). Comparison of RB and AGG across those in the AL and no CD groups revealed potent differences at age 17 (d = .68–1.02), but somewhat smaller differences at age 11 (d = .40–41). Moreover, the general pattern of differences observed at age 17 persisted up through age 24. LCP men reported significantly more symptoms of AAB at age 24 than did AL men (d = .45), whereas AL men continued to report significantly more symptoms of AAB than did men in the no CD group (d = .70). The percentage of individuals meeting full diagnostic

criteria for ASPD (i.e., as defined via a definite diagnosis of CD at 11 and/or 14 and a "definite diagnosis" or AAB at age 24) also varied significantly across the age-of-onset groups, with 54.2% of those in the LCP qualifying for a formal diagnosis of ASPD at age 24 as compared to 15.5% of AL youth and 0.5% of no CD youth (the latter was a function of 2 of the childhood-only cases). In short, our age-of-onset groupings appear to largely tap the age-of-onset taxonomy postulated by Moffitt (1993; 2003), with the important caveat that "AL" youth continued to exhibit some antisocial behavior up through early adulthood (albeit significantly less so than the LCP youth).

The results of our primary MLM regression analyses are presented in Table 3. We begin with Model 1. As seen there, the presence of a formal diagnosis of CD significantly predicted AAB at age 24 (note that the direction of this effect is appropriately negatively signed since no CD was coded 2 while LCP and AL were both coded −1). The age-of-onset and persistence of that CD diagnosis provided additional predictive information over the presence/absence of CD, such that LCP youth evidenced significantly more AAB at age 24 than did AL youth. Importantly, however, when AGG and RB symptom counts at age 17 were also added to the model (see Model 2a), age-of-onset no longer predicted AAB at age 24 whereas AGG and RB each independently predicted adult antisocial behavior. Such results suggest that the AGG and RB symptom dimensions offer incremental validity over and above both a diagnosis of CD and the age-of-onset taxonomy.

To confirm that our results were not a function of the fact the age 17 symptom count variables were more proximal to the age 24 outcome variable than was age-of-onset (which was operationalized using age 11 and age 14 CD diagnoses), we repeated analyses using age 11 AGG and RB symptom counts (see Model 2b). As in Model 2a, age-of-onset no longer predicted AAB at age 24 when AGG and RB symptom counts at age 11 were also added to the model. RB at 11, by contrast, continued to predict AAB at age 24 (although AGG did not). In short, the above results cannot be solely attributed to the shorter time lag between the symptom count assessments and adult antisocial behavior.

To also confirm that the results were not driven primarily by the additional information on severity contained in symptoms counts as compared to diagnoses, we trichotomized the AGG and RB symptom counts (i.e., 0 symptoms = "0", 0.5–1 symptoms = "1", and 1.5 or more symptoms = "2") at ages 17 and 11 and re-ran our final model. The results of these analyses are presented under Models 2c and 2d, respectively. As seen there, the RB parameter estimates (and at age 17, the AGG estimate) remained significant predictors of AAB at age 24, indicating that RB and to a lesser extent AGG outperform age-of-onset in their prediction of adult antisocial behavior even when AGG and RB symptom counts are collapsed into trichotomized variables. Such results suggest that the improved predictive power of AGG and RB over age-of-onset is not accounted for solely by the additional severity information contained in symptom counts as compared to diagnoses. Rather, the aggressive versus non-aggressive behavioral manifestation of antisocial behavior appears to predict adult antisocial behavior better than the age-of-onset of CD in general.

Yet another consideration relates to the fact that our age-of-onset groupings were assessed using a best-estimate informant approach whereas our outcome variable was self-report only. This approach benefits from the known advantages of multi-informant assessments of childhood externalizing psychopathology (Achenbach, McConaughy, & Howell, 1987). However, children and adolescents are often highly motivated to conceal antisocial acts from their parents and other authority figures, and to the extent that they are successful in doing so, are likely to have first-hand knowledge of particular clinically-meaningful acts that their parents do not. It may thus be the case that our use of a best-estimate approach obscured rather than illuminated the respective role of AGG, RB, and age-of-onset. We

therefore recomputed our age-of-onset groupings using probable and definite cases of self-reported CD at ages 11 and 14 along with self-reported age-of-onset. More than 3% of the sample (n=27) met the persistence criterion. Of these, 23 (3%) also reported that at least one CD symptom had been present by age 10. AL was again operationalized as a probable or definite diagnosis of CD at age 14 but not at age 11. Those 4 individuals with persistent CD who denied that symptoms were present by age 10 were also included in the AL group (total n = 113 participants; 15% of the sample). The MLM analyses were then re-run using these redefined age-of-onset groupings.

Results are presented in Table 4. As seen there, the presence of a formal diagnosis of CD significantly predicted AAB at age 24, as did the age-of-onset of that CD diagnosis. However, when AGG and RB symptom counts at ages 17 and 11 (see Models 2a and 2b, respectively) were added to the model, age-of-onset no longer predicted AAB at age 24. To again ensure that these results were solely not a function of the severity information contained in symptom counts as compared to diagnoses, we trichotomized AGG and RB and ran Models 2c and 2d. As before, RB (and at age 17, AGG) continued to predict age 24 antisocial behavior, whereas age-of-onset did not. In short, the results of these supplementary analyses collectively suggest that the results reported above are not a function of our use of a best-estimate approach.

Supplemental Analyses

We first sought to confirm that our results persisted to maternal informant-reports as well. We thus recomputed our age-of-onset groupings using probable and definite maternal-reported diagnoses at ages 11 and 14 (all persistent cases evidenced at least one symptom by age 10), and re-ran Model 2a. Age-of-onset again did not predict AAB at age 24 (fixed effect estimate (SE) = .03 (.06), p=.66). By contrast, RB and AGG continued to predict adult antisocial behavior (fixed effect estimates (SE) = .34 (.04), p<.001 for RB; .17 (.05), p<.001 for AGG). The results reported above thus do not appear to be a function of the specific informants used to form our age-of-onset groupings.

We next sought to determine whether our results would generalize to a conceptually-related but distinctive set of externalizing behaviors. In particular, although the DSM symptoms of CD are largely distinct from those of AAB, the fact that both CD and AAB symptoms are included in diagnoses of ASPD does raise the possibility of predictor-criterion overlap (i.e., symptoms of antisocial behavior predicting later symptoms of antisocial behavior). We would argue that this is apriori unlikely to account for the results presented above, both because age-of-onset is also based on the presence or absence of CD symptoms, but also because the various CD symptom counts were correlated .44 or less with the age 24 AAB symptom count (results that are consistent with prior research; Lahey, Loeber, Burke, & Applegate, 2005). Nonetheless, it was worth addressing this issue empirically. We therefore evaluated whether our Model 2a results applied to adult substance use problems (defined as a sum of Alcohol Dependence and Cannabis Dependence symptom counts at age 24). Age-of-onset did not significantly predict adult substance use problems (fixed effect estimate (SE) = .08 (.05), p=.11), whereas age 17 RB did (fixed effect estimates (SE) = .24 (.06), p<.001 for RB; .06 (.06), ns for AGG), results that are fully consistent with those of Burt & Hopwood (2010). As those with substance use problems at age 24 may have already commenced their substance use by age 17, however, we also re-ran Model 2a using the age 11 AGG and RB symptom counts. Our core conclusions were unchanged. Age-of-onset did not emerge as a significant predictor of adult substance use problems (fixed effect estimate (SE) = .001 (.05), p=.98), whereas RB and AGG did (fixed effect estimates (SE) = .26 (.07), p<.001 for RB; .14 (.06), p<.05 for AGG). In short, the behavioral sub-types also appear to outperform age-of-onset when examining distinct but related forms of externalizing psychopathology.

For our final series of checks, we sought to confirm that our results were not dependent on some of the other criteria used to define our age-of-onset groupings. We thus re-ran Model 2a using several different definitions of LCP and AL. First, we dropped the childhood-only cases from the analyses altogether. As before, age-of-onset did not appear to predict AAB at age 24 (fixed effect estimate (SE) = .06 (.04), p=.11). By contrast, RB and AGG continued to predict adult antisocial behavior (fixed effect estimates (SE) = .28 (.05), p<.001 for RB; .15 (.05), p=.001 for AGG). Dropping the child-only participants from the sample thus did not influence our core findings.

We next redefined our best-estimate LCP group to include those youth with childhood-onset CD that did not persist through adolescence (these child-only participants were originally included in the no CD group). This decrease in the severity of our LCP group served to increase the size of the LCP group (from n=60 to 112), but otherwise did not influence our findings. Age-of-onset again did not appear to predict AAB at age 24 (fixed effect estimate (SE) = .02 (.04), p=.67). By contrast, RB and AGG continued to predict adult antisocial behavior (fixed effect estimates (SE) = .30 (.05), p<.001 for RB; .15 (.05), p=.001 for AGG).

We then re-ran Model 2a yet again, redefining the LCP and AL groupings to include only those with definite (rather than probable and definite) best-estimate diagnoses (n = 25 and 98 for LCP and AL, respectively). Increasing the severity of our CD groups also did not appear to influence analytic results. Age-of-onset again did not appear to predict AAB at age 24 (fixed effect estimate (SE) = .04 (.07), p=.56). By contrast, RB and AGG continued to predict adult antisocial behavior (fixed effect estimates (SE) = .31 (.04), p<.001 for RB; .17 (.04), p<.001 for AGG). In sum, our findings held regardless of the specific approach used to define our age-of-onset groupings.

DISCUSSION

The goal of the current study was to evaluate whether AGG and RB have additional predictive utility over and above of Moffitt's (1993) age-of onset taxonomy. We thus evaluated how AGG, RB, and age-of-onset predicted the adult or criterion C symptoms of Antisocial Personality Disorder in a sample of male twins assessed at ages 11, 14, 17, and 24. Analyses revealed that those with persistent childhood-onset CD reported far higher rates of AGG and RB than those with adolescence-onset CD or those without CD, results that are in keeping with the more severe levels of antisocial behavior thought to characterize LCP youth (Moffitt, 1993). Age-of-onset was also predictive of AAB at age 24, such that those with earlier ages-of-onset reported higher rates of adult antisocial outcomes, and did so over and above the presence or absence of a diagnosis of CD. However, when AGG and RB were also entered into the regression equation, age-of-onset no longer predicted later antisocial behavior. Furthermore, this finding did not appear to stem solely from the increased severity information contained within symptom counts as compared to diagnoses, the specific informants used, or the age at which the behavioral sub-types were assessed. Rather, the aggressive versus non-aggressive behavioral manifestation of adolescent antisocial behavior predicted adult antisocial behavior outcomes better than did the age-of-onset of CD in general. Such findings collectively suggest that behavioral sub-types may be a better predictor of antisocial outcomes in early adulthood than is age-of-onset.

As noted, our age-of-onset results were (when analyzed alone) consistent with Moffitt's predictions (Moffitt, 1993; 2003). Moreover, our regression results were highly consistent with those of Burt and Hopwood (2010), which (to our knowledge) is the only other study explicitly comparing the predictive utility of age-of-onset to the behavioral sub-types of antisocial behavior. Burt and Hopwood (2010) also found that, when analyzed alone, age-of-onset was predictive of anger and alcohol dependency, such that those with earlier ages-of-

onset evidenced higher rates of these outcomes. However, once AGG, RB, and age-of-onset were simultaneously entered into a regression equation, age-of-onset no longer predicted anger or alcohol dependency. By contrast, RB continued to predict alcohol dependency, whereas anger was predicted by both AGG and RB.

Although the current results are thus consistent with available literature, there are several limitations that are worthy of additional consideration. First, analyses in the present study were limited to boys only, as the very low base rate of LCP in girls (i.e., 1% or less) prohibited a meaningful comparison of LCP and AL in our female sample. Future studies should seek to compare the predictive utility of age-of-onset and the behavioral sub-types in a very large sample of girls or in a clinical sample (in which we would expect a higher proportion of girls to meet LCP criteria). Next, our sample consists of twin siblings rather than singletons. Although neither the rates nor the developmental trajectories of conduct problems vary across twins and singletons (Robbers et al., 2010; Rutter & Redshaw, 1991), it would be worth ensuring that these results generalize to non-twin populations as well.

In addition, because our intake assessment was conducted when the twins were roughly 11 years old, the assessment of symptom age-of-onsets during childhood was necessarily retrospective. This is problematic, since retrospective assessment of symptom age-of-onset is known to be an unreliable process (Angold, Erkanli, Costello, & Rutter, 1996; Tremblay, 2010). Although we would never contest the difficulty of retrospectively establishing the precise age-of-onset for a given symptom, the broad age-ranges (i.e., symptom presence by age 10) and relatively recent nature of the retrospective recall used here may ameliorate this concern somewhat. As circumstantial evidence for this conjecture, we note that 90–100% of those with persistent CD at ages 11 and 14 also reported a symptom onset by age 10, regardless of the informant approach used. Indeed, repeating our primary analyses using only persistence of CD across assessments to define our LCP group did not alter our results in any way (results not shown). The results reported here thus do not appear to be dependent on retrospective recall of age-of-onset. That said, this limitation does highlight a broader logistical difficulty with using age-of-onset as the primary indicator of Moffitt's taxonomy. To avoid retrospective recall, research examining the etiology or outcome of antisocial behavior effectively necessitates that researchers examine longitudinal samples in which participants are assessed from early- or mid-childhood up through adolescence or adulthood. As such samples are relatively difficult to come by (at least as compared to cross-sectional or short-term longitudinal studies), researchers interested in the development, etiology, or treatment of antisocial behavior are faced with limited sampling options. In this light, age-of-onset is perhaps not a practical or useful cut-point for many studies.

Finally, given that AGG may be particularly characteristic of LCP (and particularly during childhood), it is somewhat surprising that RB consistently emerged as the stronger individual predictor of AAB at age 24. Although it is unclear what may account for these findings, one likely possibility lies in the specific behaviors that constitute the adult or criterion C symptoms of Antisocial Personality Disorder. Symptoms assess acts such as consistent irresponsibility, impulsivity/failure to plan, and no regard for the truth, most of which fall more or less cleanly within the domain of RB. Indeed, only one symptom of AAB clearly taps physical aggression (e.g., irritable and aggressive). Given this, it may be that RB emerged as the stronger predictor because the AAB outcome is itself more closely linked to RB as compared to AGG. Consistent with this speculation, RB also emerged as the best predictor of substance use problems, both in these analyses and in those of Burt & Hopwood, 2010, a finding that likely stems from the particularly strong links between RB and substance use. Indeed, DeMarte (2008) and Burt & Donnellan (submitted) similarly found that substance use problems had little to no relationship with AGG, once the overlap with RB was controlled. Regardless, these RB "leanings" of our outcome variables could

also mean that age-of-onset effects were dampened accordingly (although age-of-onset did predict AAB at 24 in the expected directions when modeled without AGG and RB). The current results thus apply only to the outcomes examined here. It remains unclear how they would generalize to other definitions of adult antisocial behavior, or to the well-documented correlates of adult antisocial behavior (e.g., impulsivity, negative emotionality, low academic achievement, incarceration, inattention, and hyperactivity). It may well be the case that age-of-onset outperforms (or is equal to) AGG and RB in the prediction of these outcomes. Future research should seek to extend these analyses to both a broader definitions of adult antisocial behavior, as well as to other key correlates of antisocial behavior.

Conclusions

Our results have several potentially important implications. First, adolescence-onset men reported significantly more symptoms of adult antisocial behavior than did men in the no Conduct Disorder group (d = .70). Moreover, 15.5% of adolescence-onset youth met full diagnostic criteria for Antisocial Personality Disorder at age 24. The finding that some "adolescence-limited" youth continued to be antisocial up through early adulthood (albeit significantly less so than the life course persistent youth) is by and large inconsistent with Moffitt's original (1993) theory. Similarly, only 54% of those boys with childhood-onset Conduct Disorder were ultimately classified as part of the life-course persistent grouping. Although the finding that 46% of youth with childhood-onset CD desisted from antisocial behavior by adolescence is also inconsistent with the original Moffitt (1993) hypothesis, in which childhood-onset and life course persistent are essentially interchangeable, more recent work has suggested that this pattern of persistence for only 40–70% of childhood-onset youth is actually the norm (Barker & Maughan, 2009;Barker et al., 2010;Nagin & Tremblay, 1999;van Domburgh et al., 2009). Barker and Maughan (2009), for example, examined more than 7,000 children assessed from birth through age 13 as part of the longitudinal and population-based Avon Longitudinal Study of Parents and Children. Child conduct problems were assessed at ages 4, 7, 8, 10, 12 and 13 years. Roughly 9% of the sample was described as "early-onset persistent" in their conduct problems. An additional 15% of the sample, however, evidenced conduct problems during childhood that largely desisted by adolescence. So robust are these exceptions to the life-course persistent and adolescence-limited taxonomy that they have been incorporated into more recent permutations of the theory (see Moffitt, 2003). For example, the original dual taxonomy has now been amended to include a "low level chronic offenders" group as well, which consists of early-onset youth who avoid the severe life course persistent outcome but continue to chronically engage in low levels of antisocial behavior. Even so, the finding that many childhood-onset youth may "recover" whereas many adolescence-onset youth persist in their antisocial behavior dovetails nicely with more recent suggestions that life course persistent and adolescence-limited antisocial behavior are quantitatively, rather than qualitatively, different forms of antisocial behavior (Walters, 2010). Future research should continue to explore this possibility.

Building on this point, the utility of Moffitt's (1993) theory has not been systematically compared to other ways of parsing the heterogeneity within antisocial behavior. The current study sought to fill this gap in the literature, and did so with some surprising results. The age-of-onset of Conduct Disorder no longer predicted the adult or criterion C symptoms of Antisocial Personality Disorder once we controlled for overlap with physically aggressive and non-aggressive, rule-breaking symptom dimensions of Conduct Disorder. Moreover, these results persisted when controlling for both the improved predictive power of symptom counts as compared to diagnoses and the presence or absence of a Conduct Disorder diagnosis. They also persisted when operationalizing our age-of-onset groupings using more and less stringent criteria, when separately examining self-reports and maternal informant-

reports of Conduct Disorder, and when examining distinct but conceptually-related forms of externalizing psychopathology. Such results collectively imply that the age at which antisocial behavior first manifests itself may be a less important predictor of adult outcome than was originally anticipated; rather, the behavioral sub-types linked to age-of-onset appear to be the more salient predictors of antisocial behavior outcomes. One logical implication of these results is that the specific form of antisocial behavior may (at least partially) explain prior age-of-onset findings. Specifically, the presence of frequent/severe physically aggressive behaviors (rather than a childhood age-of-onset per se) may be driving the adult outcomes previously linked to life course persistent antisocial behavior. Associations with adolescence-onset antisocial behavior, by contrast, may be related to the presence of rule-breaking. In short, rather than serving as the best predictor of antisocial behavior outcomes, age-of-onset may simply be another way of indexing and differentiating between AGG and RB. Future research should continue to explore this possibility.

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Table 1

DSM-III-R Conduct Disorder symptoms comprising the rule-breaking and aggression scales.

Rule-breaking (n=6 symptoms)	Aggression (n=6 symptoms)
Stolen without confrontation	Destroyed property
Runaway from home	Cruel to animals
Often lies	Used a weapon
Fire-setting	Initiates physical fights
Often truant	Stolen with confrontation
Broken into home/car	Cruel to people

Note. Twelve of the thirteen symptoms of DSM-III-R Conduct Disorder were partitioned into rule-breaking and physically aggressive symptom dimensions. The remaining symptom, Forced Sex, was not assessed.

Table 2

Number of symptoms by age-of-onset grouping.

Symptom count variable	No CD		Adolescence-limited (AL)		Life Course Persistent (LCP)	
	Mean	SD	Mean	SD	Mean	SD
AGG, age 11	0.36	0.62	0.59	0.63	2.02	1.29
RB, age 11	0.22	0.46	0.38	0.48	1.49	1.03
AGG, age 17	0.34	0.65	0.89	1.00	1.27	1.48
RB, age 17	0.35	0.61	1.19	1.10	2.11	1.70
AAB, age 24	1.37	1.05	2.24	1.43	3.09	1.77
ASPD diagnosis at age 24	0.5%	--	15.5%	--	54.2%	--

Note. Statistical comparisons of the means were conducted on the log-transformed data, as they better approximate normality. However, the corresponding raw symptom counts are presented in here and discussed in the text to promote ease of understanding. The percentage of individuals meeting full diagnostic criteria for ASPD (i.e., as defined via a definite diagnosis of CD at 11 and/or 14 and a "definite diagnosis" of AAB at age 24) is also presented. All possible pairings of means within a row differed significantly from one another at $p < .05$, respectively.

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Table 3

Unstandardized multilevel linear model fixed effect estimates, defining contrast codes by the pattern of combined mother-offspring "best-estimate" CD diagnoses at ages 11 and 14 and reported symptom age-of-onset.

	Model 1	Model 2a	Model 2b	Model 2c	Model 2d
	Estimate (SE)	Estimate (SE)	Estimate (SE)	Estimate (SE)	Estimate (SE)
Contrast code 1: CD diagnosis	−.13 (.02)**	−.07 (.02)**	−.10 (.02)**	−.08 (.02)**	−.11 (.02)**
Contrast code 2: Age-of-onset	.09 (.04)*	.05 (.04)	.05 (.04)	.07 (.04)~	.05 (.04)
AGG symptom count	----	.16 (.04)**	.07 (.05)	.10 (.03)**	.00 (.03)
RB symptom count	----	.29 (.04)**	.25 (.06)**	.17 (.03)**	.13 (.03)**

Note. The outcome variable is a log-transformed symptom count of the adult or criterion C ASPD symptoms. For contrast code 1, no CD was coded as a 2 and life course persistent (LCP) and adolescence-limited (AL) were both coded as −1. As a result, an increase in AAB at age 24 with a CD diagnosis would be negatively signed. For contrast code 2, no CD was coded a 0, AL was coded −1, and LCP was coded as 1. The coefficient for the first contrast code tests whether those with CD (be it AL or LCP) differ from those without CD. The second contrast code tests whether LCP youth differ from AL youth. In Model 2a, AGG and RB at age 17 are modeled as log-transformed symptom counts. In Model 2b, AGG and RB at age 11 are modeled as log-transformed symptom counts. In Model 2c, age 17 symptom counts of AGG and RB were trichotomized prior to analysis. In Model 2d, age 11 symptom counts of AGG and RB were trichotomized prior to analysis.

** $p < .01$

* $p < .05$

~ $p < .10$

Table 4

Unstandardized multilevel linear model fixed effect estimates, defining contrast codes by the pattern of youth self-reported CD diagnoses at ages 11 and 14 and reported symptom age-of-onset.

	Model 1	Model 2a	Model 2b	Model 2c	Model 2d
	Estimate (SE)	Estimate (SE)	Estimate (SE)	Estimate (SE)	Estimate (SE)
Contrast code 1: CD diagnosis	−.13 (.02)**	−.04 (.02)*	−.09 (.02)**	−.07 (.02)**	−.10 (.02)**
Contrast code 2: Age-of-onset	.11 (.06)*	.02 (.06)	−.01 (.06)	.05 (.06)	.04 (.06)
AGG symptom count	----	.16 (.05)**	.11 (.06)~	.10 (.03)**	.03 (.03)
RB symptom count	----	.31 (.04)**	.29 (.07)**	.18 (.03)**	.16 (.03)**

Note. The outcome variable is a log-transformed symptom count of the adult or criterion C ASPD symptoms. For contrast code 1, no CD was coded as a 2 and life course persistent (LCP) and adolescence-limited (AL) were both coded as −1. As a result, an increase in AAB at age 24 with a CD diagnosis would be negatively signed. For contrast code 2, no CD was coded a 0, AL was coded −1, and LCP was coded as 1. The coefficient for the first contrast code tests whether those with CD (be it AL or LCP) differ from those without CD. The second contrast code tests whether LCP youth differ from AL youth. In Model 2a, AGG and RB at age 17 are modeled as log-transformed symptom counts. In Model 2b, AGG and RB at age 11 are modeled as log-transformed symptom counts. In Model 2c, age 17 symptom counts of AGG and RB were trichotomized prior to analysis. In Model 2d, age 11 symptom counts of AGG and RB were trichotomized prior to analysis.

** $p < .01$

* $p < .05$

~ $p < .10$