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Age of onset and the subclassification of conduct/dissocial disorder

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Abstract

Background—Conduct Disorder (CD) is a markedly heterogeneous psychiatric condition. Moffitt (1993) proposed that subclassification of CD should be according to age of onset. Our goals were to compare childhood-onset and adolescent-onset CD in terms of differences in phenotypic risk factors, genetic analyses, and factors associated with the persistence of antisocial behavior into young adulthood.

Methods—The data are from the Virginia Twin Study of Adolescent Behavioral Development (VTSABD) and Young Adult Follow-Up (YAFU). Childhood-onset CD was defined as CD beginning at or before age 11. Adolescent-onset CD was defined as having CD onset between ages 14 and 17. These subgroups were compared on ADHD, young adult antisocial behavior (ASB), family dysfunction, and parental depression. Genetic analyses compare childhood-onset and adolescent-onset CD, as well as their cooccurrence with ADHD and ASB. Finally, predictors of persistence were examined.

Results—Childhood-onset CD was significantly associated with ADHD, ASB, family dysfunction, and parental depression. Adolescent-onset CD was marginally associated with parental depression ($p = .05$) but not with any of the other risk factors. Univariate genetic models showed that both childhood-onset and adolescent-onset CD involve a large genetic liability accounting for 62% and 65% of the variance, respectively. A common genetic factor (as well as an ADHD-specific factor) accounted for the cooccurrence of childhood-onset CD and ADHD. The cooccurrence of childhood-onset CD and ASB are reflected by a common genetic factor with genetic specific effects on ASB. There was no etiological link between adolescent-onset CD and either ADHD or ASB. Both ADHD and family dysfunction were significantly associated with the persistence of antisocial behavior into young adulthood.

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Conclusions—Phenotypic findings differentiated between childhood-onset and adolescent-onset CD. ADHD and family dysfunction predicted persistence of antisocial behavior into young adulthood.

Keywords

Age of onset; conduct disorder; persistence into adult life

Introduction

It has long been observed that there is considerable variability in antisocial behavior and this might represent meaningful heterogeneity in varieties or patterns (Hindelang, Hirschi, & Weis, 1981). For many years, people had hoped that the type of crime committed would serve to delineate valid subgroups but this has not proved to constitute a helpful approach (Rutter, Giller, & Hagell, 1998). Something in the nature of a break-through was provided by Moffitt's (1993) proposal that a key differentiation should be according to age of onset. Moffitt (2003) summarized the basic distinction as that between what she termed 'life-course-persistent' (LCP) antisocial behavior and 'adolescence-limited' (AL) antisocial behavior. The former (LCP) was considered to have its origins in neurodevelopmental processes that are evident in childhood but with antisocial behavior worsening thereafter. Although based on neuropsychological deficits and/or hyperactivity, it is thought to be exacerbated by a high risk social environment (as indexed by inadequate parenting, family conflict, and poverty). Thus, the pattern was considered uncommon, persistent, and pathological. The pattern was found to be much more common in males than females (Moffitt, Caspi, Rutter, & Silva, 2001).

By contrast, AL antisocial behavior was thought to have its origin in social processes; beginning in adolescence, and followed by desistance in early adult life. It was common, relatively transient, and near-normative. It was thought to arise as a result of a 'maturity gap' and to be much influenced by delinquent peer group pressures. By comparison with the LCP group, it was considered to be less heritable and not associated with the neurodevelopmental features associated with the LCP variety. The LCP-AL differentiation has led to a wealth of high quality research and it has informed thinking about classification issues. Indeed it constitutes the main basis for the American Psychiatric Association's (APA, 2013) DSM-5.

It is necessary to appreciate that this is not synonymous with the LCP-AL distinctions, which emphasize course as much as age of onset. Obviously, a diagnostic classification cannot be based on an outcome that is unknown at the time when a diagnosis must be made. On the other hand, any testing of the age of onset subclassification would have to include adult outcome as one of the possible key validating criteria.

On the whole, research from groups all over the world has largely supported the LCP-AL distinction (Odgers et al., 2008; Rutter et al., 1998). However, three main modifications seem to be required. First, as shown earlier by Robins (1966, 1978), and confirmed by Odgers et al. (2008), about half of those with an childhood-onset do not continue with their antisocial behavior into adult life – pointing to the need to recognize a substantial childhood-limited (CL) group. Second, AL antisocial behavior sometimes constitutes a precursor of

more severe social malfunction (Odgers et al., 2007). Third, the AL group differed from nonantisocial youth in showing both individual and family adversity, albeit less than that associated with LCP (Roisman, Monahan, Campbell, Steinberg, & Cauffman, 2010).

In addition, there are varieties of antisocial behavior not encompassed by the LCP, AL, and CL distinctions. Most particularly, they do not include the infrequent adult-onset antisocial behavior (Elander, Rutter, Simonoff, & Pickles, 2000; Elander, Simonoff, Pickles, Holmshaw, & Rutter, 2000), antisocial behavior (especially violence) that develops only after the onset of psychosis (Hodgins, Mednick, Brennan, Schulzinger, & Engberg, 1996; Hodgins, Viding, & Plodowski, 2009), or substance abuse (Brook, Whiteman, Finch, & Cohen, 1996), or sexual offences (Barbaree & Marshall, 2008), and antisocial behavior associated with psychopathy (Rutter, 2012). All of these are pertinent in relation to science, but none is yet ready for consideration with respect to the subclassification of Conduct/Dissocial Disorder. Accordingly, they will not be discussed further here. Instead, we focus on whether the DSM-5 specification of childhood-onset and adolescent-onset subtypes can be validated, and whether, within the childhood-onset group, it is possible to identify those likely to persist into adult life.

Moffitt (2006) has usefully brought together the evidence comparing LCP and AL antisocial behavior, and Moffitt et al. (2008) outlined the research needs in relation to DSM-5. Three main points from the latter review require emphasis. First, there was the need to have prospective data on age of onset in view of the demonstrated unreliability of retrospective recall (see Henry, Moffitt, Caspi, Langley, & Silva, 1994). Second, there was the desirability of using family history data. Third, there was the need to find a means of differentiating a childhood-limited (CL) type from a childhood-onset type that persisted into adult life (as LCP).

Barker and Maughan (2009) used the Avon dataset (with about half the sample lost through attrition) to examine whether parental or early postnatal (up to age 4 years) features predicted persistence of conduct problems up to age 13 years. An under-controlled temperament and negative parenting were both significant predictors of persistence of conduct problems.

A recent review by Fairchild, Goozen, Calder, and Goodyer (2013) argues that the whole developmental taxonomic theory required revision because the difference between AL and LCP appeared to be quantitative rather than qualitative. However, as long ago as 2002, Moffitt, Caspi, Harrington, and Milne (2002) concluded much the same – pointing to the evidence from the follow-up to age 26 years of the Dunedin cohort that the AL group showed elevated impulsive traits and a worse adult outcome than envisaged in the original Moffitt (1993) paper – albeit less abnormal than the childhood-onset group.

Methods

Research design

The purpose of our study was to test the validity of the differentiation between childhood-onset and adolescent-onset antisocial behavior and to determine whether, within the

childhood-onset group, it was possible to differentiate between those whose antisocial behavior did and did not persist into adult life.

Methodology—Our analyses are entirely based on the Virginia Twin Study of Adolescent Behavioral Development (Eaves et al., 1997; Hewitt et al., 1997; Simonoff et al., 1997), and its follow-up into early adult life (Silberg, Rutter, Tracy, Maes, & Eaves, 2007). Ethical approval for the VTSABD and YAFU studies were provided by the institutional review board of Virginia Commonwealth University, and appropriate informed consent was obtained from all persons participating in this research. The dataset has several important advantages – particularly yearly waves of data collection and the annual availability of ADHD symptom findings from the outset at the age of 8 years. In the interest of harmonization with the DSM-5, we decided to stick closely to the DSM approach. That is, an adolescent-onset required no symptoms of CD prior to adolescence and, conversely, childhood-onset meant any CD symptoms in that age period. In other words, the two are mutually exclusive and jointly exhaustive. We decided to follow the ‘OR’ rule, so that if a CD feature was reported by any informant, it was counted as present. It will be appreciated that is necessarily somewhat arbitrary but it seemed the most workable approach.

In any longitudinal study dealing with prospective data there is the problem of differentiating, at any given age, between individuals who do not have CD symptoms and those who will have them but have not yet developed them. In order to deal with that issue we decided to set a cutoff of age 14 years or more for adolescence, after the time when puberty has begun in most children and after the patterns of genetic influences has mainly stabilized (Silberg, Rutter, & Eaves, 2001). Many previous studies of the age of onset issue have relied on the use of a reported specific date of onset but that neglects the advantages of prospective annual data collection. Accordingly we defined the childhood-onset variety of CD as CD reported on one or more of the annual assessments in the childhood period (at or before age 11) beginning with data collection at age 8 onwards. All assessments made use of thorough assessments on the Child and Adolescent Psychiatric Assessment (CAPA; Angold et al., 1995) interview and not just questionnaire scores. The adolescent-onset variety was similarly defined on the basis of CD features on any of the annual assessments from ages 14 to 17 and no CD features in childhood.

Adult outcome was defined in terms of antisocial behaviors as identified in a single telephone interview made at some point between age 18 and 25. Adult outcome was defined in terms of at least two antisocial behaviors and not in terms of antisocial behavior as dealt with in either ICD-10 or DSM IV (APA, 2013). In the DSM-5, the diagnosis of conduct disorder requires at least three out of a list of fifteen antisocial behaviors. As the telephone interview included only seven antisocial behaviors, clearly we needed a lower cut-off to achieve rough comparability. On the other hand, we wanted a cut-off that was likely to reflect a clinically meaningful pattern (but not one so high that it produced an unduly low prevalence rate). With multifactorial syndromes that are basically dimensional, there is an inevitable arbitrariness of the precise cut-off chosen, but a cut-off of two seemed most appropriate.

To assess the possible role of ADHD in differentiating between childhood and adolescent onsets, we needed to decide on its measurement. The first question was whether or not to include inattention as well as hyperactivity/impulsivity items. Because the trajectories of the two are rather different (Fergusson, Horwood, & Lynskey, 1993; Larsson, Larsson, & Lichtenstein, 2004), we restricted ADHD features to hyperactivity/impulsivity items and that these had to be present during the age period up to age 10 (because of the wish to treat ADHD symptoms as a neurodevelopmental phenomenon). Because of concern over possible rater effects when looking at phenotypic covariation (e.g. between CD and ADHD), teacher ratings from the Rutter B teacher scale (Rutter, Tizard, & Whitmore, 1970) supplemented with hyperactivity items from the Conners scale (Conners, 1997) were used.

The validity of the child versus adolescent distinction was tested by using these criteria. First, we examined differences between the two groups on the basis of the presence or absence of antisocial behavior in early adult life. Note that if we had data extending the adult period beyond age 25 years there would be greater persistence, but the relevant finding here is that the same age was used for the child versus adolescent contrast. Second, we compared the groups on the basis of ADHD in childhood, as defined above. Third, we compared the groups on the basis of alcohol abuse or Antisocial Personality Disorder in either parent terming this 'family dysfunction'. All these comparisons were based on individuals, rather than twin pairs, but we undertook analyses to check whether any bias was created by the clustering within twin pairs.

Secondly, taking advantage of the use of a twin sample, we examined the differences between the childhood-onset and adolescent-onset groups with respect to genetic influences. There is good evidence of genetic influences on the liability to Conduct Disorder (Moffitt, 2005; Rhee & Waldman, 2002) but a lack of evidence on whether these vary by age of onset (Moffitt et al., 2008). The issue is not just whether the age of onset groups vary in their level of heritability, but also whether they involve the same genetic influences. This combination of phenotypic and quantitative genetic analyses constitutes one novel aspect of our research.

Finally, (the most novel feature) we sought to determine whether the features that differentiated early- and late-onset (i.e. family history of antisocial behavior/alcohol abuse, and hyperactivity) also differentiated the early-onset antisocial behavior that persisted into adult life from that which did not. Because we wished to check the specificity of the family history effect, we compared it with a family history of depression.

In order to test the validity of the child versus adolescent distinction, we used three criteria. First, on the basis of the Moffitt (1993, 2003) focus, we compared the adult outcome of the two groups – in each case testing the significance of the difference from the general population base rate (i.e. those without antisocial behavior in childhood or adolescence). Second, we compared the groups on the basis of ADHD symptoms in childhood. Third, in view of the importance of family history as an index of both genetic and environmental risk, we compared the groups on family dysfunction as indexed by alcohol problems or Antisocial Personality Disorder in either parent (Odgers et al., 2007). To check the specificity of these problems we also compared the groups on rates of maternal depression.

Statistical approach

Phenotypic analyses—To test the significance of association between the two forms of CD with early ADHD, young adult antisocial behavior (ASB), family dysfunction, and parental depression, the Fisher exact statistic was used (SAS, 2000). The influence of these same risk factors on the persistence of antisocial behavior into adult life was also evaluated using this approach.

Genetic analyses—Standard biometrical genetic model fitting methods were used (Neale & Cardon, 1992) for decomposing the observed variation in early and late CD and their covariation with early ADHD and ASB in terms of additive genetic (A), shared environmental (C) and nonshared or unique environmental (E) risks. Additive gene action (A) reflects the additive or average effect of individual alleles at genetic loci influencing a trait or behavior. Common environmental effects (C) describe influences that make family members more alike compared to random pairs of individuals. Nonshared or unique environmental risks (E) capture aspects of the environment that are unique to each individual, plus error, and are therefore uncorrelated between twins. An MZ correlation that is about twice the DZ correlation suggests the presence of an underlying genetic influence that is additive in nature. A DZ correlation that is greater than half the MZ correlation suggests common environmental influences. An MZ correlation that is less than one suggests the presence of specific or unique environmental influences. The comparison of the cross-twin/cross-variable association in the MZ and DZ twins provides the information for testing whether genetic and/or shared environmental influences account for the association between the two variables (e.g. CD and ADHD/ASB). Because early and late CD and their associated risk factors were coded as present or absent ('0' or '1') the data for model fitting was analyzed using the raw ordinal data option in the Mx statistical software package (Neale, Baker, Xie, & Maes, 2003).

Results

Phenotypic associations

Because the early and late CD groups were defined to be mutually exclusive, differences in the rates of young adult antisocial behavior, ADHD, family dysfunction, and parental depression were evaluated within rather than across the CD groups. The GENMOD regression procedure in SAS (2000) was used to confirm whether there were any biases created by the nonindependence of the twin data. The prevalence and significance of ADHD, young adult ASB, family dysfunction, and parental depression within childhood-onset CD and adolescent-onset CD groups are shown in Table 1.

Early childhood CD

Highly significant associations were observed between childhood-onset CD, and both hyperactivity and young adult antisocial behavior (ASB), $p < .001$. Of the 478 children with conduct disturbance, 40% were rated by teachers as having ADHD. Those with childhood-onset CD also reported antisocial behavior in young adulthood. 36% of those with young adult ASB had childhood-onset CD; 48% of those with childhood-onset CD continued to present with antisocial behaviors in young adulthood. Those with childhood-onset CD also

came from families with high levels of dysfunction, characterized by at least one parent with alcoholism or Antisocial Personality Disorder. Nearly 65% of those children from dysfunctional families had childhood-onset CD, $p < .0001$. Of those families with parental depression, only 22% had a child with childhood-onset CD, $p < .02$.

Adolescent CD

In marked contrast to the findings for childhood-onset CD, the associations between adolescent-onset CD and early ADHD, young adult ASB, and family dysfunction were not statistically significant. There was a marginally significant association of late CD with parental depression.

Modeling of twin data

The twin data were then used to examine whether the heritability of childhood-onset CD differed from that of adolescent-onset CD. The standard partitioning of the variance, making use of the difference between monozygotic and dizygotic pairs, showed strong genetic influences on both traits. Univariate genetic models were fitted to the early and late CD groups separately using Mx (Neale et al., 2003). Based upon likelihood ratio criteria for evaluating the fit of alternative univariate genetic models, the shared environmental parameter could be dropped from the full model for both the early and late CD groups without a significant deterioration in fit ($p = 1$; $p > .12$, respectively). In contrast, fixing the genetic effect to zero significantly affected the fit of both models ($p < .037$; $p < .048$). Based upon the best fitting models, 62% of the variance in childhood-onset CD was due to additive genetic factors (95% CI: .50-.73) and 65% (95% CI: .56-.71) explained the variance in adolescent-onset CD. The remaining variation was accounted for by individual specific environmental factors (not shown).

We next wanted to investigate the causes of association between childhood-onset CD with ADHD and young adult ASB. Figures 1 and 2 show the parameter estimates for childhood-onset CD and ADHD, and for childhood-onset CD and ASB. For ADHD, a common genetic factor accounted for the cooccurrence between childhood-onset CD and early ADHD with a specific genetic influence on ADHD. A common genetic factor and genetic specific effects on ASB best explained the association between childhood-onset CD and young adult ASB. As expected, there was no genetic link between early ADHD and young adult ASB in those with adolescent-onset CD. The results of modeling the associations between early and late CD underscored a marked genetic influence on both forms of CD. There was no significant phenotypic overlap between adolescent-onset CD and early ADHD or young adult ASB and hence no point in partitioning the genetic and environmental influences. In the younger children, the association between Conduct Disorder and ADHD and with ASB was largely genetic.

The next set of analyses sought to elucidate factors that contributed to the continuity of antisocial behavior into adulthood. Of those with persistent antisocial behavior, 16% had at least one parent with Antisocial Personality Disorder and/or alcohol abuse (i.e. family dysfunction) and more than 50% had early hyperactivity. The results from regressing the persistence of antisocial behavior were consistent in showing a significant effect of ADHD

($p < .01$), and of family dysfunction, ($p < .02$). Both remained statistically significant when both variables were included in the model ($p < .01$ and $p < .03$, respectively). Parental depression had no contributory role on the persistence of antisocial behavior into young adulthood. These results are presented in Table 2.

Discussion

Whereas the phenotypic analyses were reasonably clear-cut in showing that the onset of CD in childhood was significantly associated with early hyperactivity, neither young adult ASB, early hyperactivity, or family dysfunction (indexed by a family history of alcoholism or Antisocial Personality Disorder) were significantly associated with adolescent-onset CD. Parental depression showed only a weak, but statistically significant, association with both childhood-onset CD and adolescent-onset CD. It was, therefore, clear that the differentiating features according to age of onset were specific to hyperactivity and alcoholism/Antisocial Personality Disorder in parents. In short, our findings provide validation of the differentiation within Conduct Disorder of the variety beginning in childhood and the variety beginning in adolescence.

Perhaps surprisingly, the genetic findings did not differentiate between the early and later onsets of antisocial behavior. Both involved a substantial genetic influence. The findings rather run against the original Moffitt notion that the etiology of early- and late-onset CD was quite different, with the late-onset group associated with peer group influences in a way that did not apply to early-onset CD. Our findings suggest that both involve a shared genetic liability and hence that, as suggested by Roisman et al. (2010) adolescent-onset antisocial behavior is not as normative as originally proposed by Moffitt (1993).

We went on, however, to investigate the causes of association between childhood-onset CD with hyperactivity and with antisocial behavior in young adulthood. Figures 1 and 2 show the standardized components of variance first for childhood-onset CD and ADHD and for childhood-onset CD and antisocial behavior in adult life. For ADHD a common genetic factor accounted for the cooccurrence between childhood-onset CD and early ADHD and a genetic effect specific to ADHD. The genetic correlation between the two was estimated at .7. A shared genetic factor explained the association between childhood-onset CD and young adult antisocial behavior with a genetic correlation of .44 and a genetic effect specific to young adult ASB. As expected from the earlier findings, there was no etiological link between adolescent-onset CD and either early ADHD or young adult antisocial behavior.

Finally, we turned attention to which variables were associated with the persistence of antisocial behavior into adult life among those with an onset in childhood. We found that both early ADHD and family dysfunction accounted for the persistence of antisocial behavior into young adulthood ($p < .0001$). Nearly 53% of those with persistent antisocial behavior had early hyperactivity; 16% had at least one parent with Antisocial Personality Disorder and/or alcohol abuse. Moreover, both variables continued to have a significant effect on the persistence after taking into account the other variable. The Dunedin study (Milne et al., 2009) findings showed that family history was associated with persistence irrespective of the age of onset. What is new and important in our findings is that, within the

childhood-onset group, it predicted persistence. This provides, for the first time, a means of sub-classifying Conduct/Dissocial disorder.

There are two findings with clear policy and practice implications. First, conduct disorder that is first manifest by age ten years is much more likely to persist into early adult life than conduct disorder that is not manifest until adolescence. Accordingly, early onset conduct disorder constitutes an important target for intervention – possibly by a parenting program (Scott, Briskman, & O'Connor, 2014). Second, within the early onset group, persistence into adult life is most likely when there is associated hyperactivity/impulsivity in the child and/or alcohol abuse or antisocial personality disorder in either parent. These features further narrow down the group with the highest risk of persistence of antisocial behavior into adult life and hence the greatest need for intervention.

Strengths and limitations

Our study has several important strengths. First, all the behavioral measures in childhood derived from detailed systematic diagnostic interviews. Second, the differentiation between child and adolescent onsets relied on prospective annual assessments rather than retrospective recall of age of onset. Third, to avoid the problem of differentiating conduct symptoms that were absent from those that were absent but had yet to occur, we set a lower age cut-off of 14 years for adolescent-onset antisocial behavior. Fourth, we were able to provide a systematic testing of the validity of age of onset effects through a combination of phenotypic and genetic analyses. Finally we were able to determine whether the variables that differentiated between child and adolescent onsets also predicted persistence into adult life – finding that they did.

The main limitation concerned the fact that the data on adult follow-up occurred at ages that varied across the age range of 18-25 years (making for some lack of comparability on adult antisocial behavior across individuals) and reliance of a telephone interview rather than a face to face interview. The need to restrict adolescence to age 14 years or older also meant that our late-onset group was smaller than might have been expected. Moreover, the effect of gender on the patterns observed in the early onset and late onset group were not considered. A further limitation is the lack of measurement of callous/unemotional traits in childhood (because they were not considered at the time the childhood data were collected).

Conclusions

Three main conclusions may be drawn. First, our findings provide strong confirmation of the validity (and utility) of the Moffitt and DSM-5 differentiation between childhood and adolescent onsets of Conduct Disorder. Second, the features associated with child-onset also predict persistence into adult life within that group. Both hyperactivity and family history of Antisocial Personality Disorder/alcoholism were independently predictive of persistence of antisocial behavior. Third, genetic influences did not differ between child-onset and adolescent-onset antisocial behavior. It should be appreciated, however, that no firm conclusion on the role of genetic influences on adult antisocial behavior are possible in view of the early age at which the follow-up took place.

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Key points

- Univariate genetic analyses showed no age of onset difference in the strength of genetic influences. Nevertheless, the multivariate genetic analyses showed large differences in the specific genetic patterns.
- Conduct Disorder that is first manifest by age 11 years is much more likely to persist into early adult life than CD that does not manifest until age 14 years.
- Whereas Conduct Disorder beginning in childhood is associated with ADHD, persistence into only adult life, and a family history of Antisocial Personality Disorder or alcoholism, this did not apply to Conduct Disorder not manifest until adolescence.
- Within those with childhood-onset Conduct Disorder, persistence into adult life was associated with both ADHD and family dysfunction.
- It is necessary to recognize, however, that there is a substantial group with childhood-limited Conduct Disorder

Figure 1.
Shared genetic influences on childhood-onset CD and hyperactivity (standardized variance components with upper and lower 95% confidence intervals)

Figure 2. Shared genetic influences on childhood-onset CD and young adult ASB (standardized variance components with upper and lower 95% confidence intervals)

Table 1

Rates of childhood hyperactivity, young adult antisocial behavior, and family dysfunction in childhood-onset and adolescent-onset CD

	Childhood CD			Adolescent CD		
	Absent	Present	<i>p</i> <	Absent	Present	<i>p</i> >
Childhood Hyperactivity						
Absent	487	290	.001	395	113	.39
Present	175	188		190	46	
Young Adult ASB						
Absent	560	217	.001	324	210	.19
Present	345	199		85	71	
Family Dysfunction						
Absent	673	442	.001	562	160	.13
Present	44	80		73	13	
Parental Depression						
Absent	535	114	.02	247	24	.05
Present	394	110		150	20	

Table 2

Logistic regression parameter estimates for persistence of antisocial behavior into young adulthood

		<u>95% Confidence Intervals</u>		
	Odds Ratio	Lower	Upper	<i>p</i> <
Univariate Models				
Childhood Hyperactivity	1.1878	1.1096	1.2710	.01
Family Dysfunction	1.0812	1.0177	1.1488	.02
Multivariate Model				
Childhood Hyperactivity	1.1748	1.0860	1.2710	.01
Family Dysfunction	1.0707	1.0053	1.1403	.03