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Risk factors for conduct disorder and oppositional/defiant disorder: evidence from a New Zealand birth cohort

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Abstract

Objective: To examine the social, family background and individual antecedents of conduct disorder (CD) and oppositional defiant disorder (ODD), the extent to which CD and ODD symptoms were predicted by common environmental risk factors, and the extent to which the antecedents of CD and ODD accounted for the comorbidity between the two disorders.

Method: Data were gathered from 926 members of a New Zealand longitudinal birth cohort. The outcome measures were DSM-IV symptom count measures of CD and ODD at age 14-16 years. Predictors measured during the period 0-14 years included: maternal smoking during pregnancy; exposure to socioeconomic adversity; parental maladaptive behavior; childhood exposure to abuse and interparental violence; gender; cognitive ability; and affiliation with deviant peers in early adolescence. Associations between the predictors and outcome measures were modelled using structural equation modelling.

Results: The analyses showed that each of the predictors was significantly ($p < .05$) associated with CD and ODD, with the exception of gender and ODD. After model fitting, the profile of risk factors that predicted CD and ODD were largely similar. The analyses revealed that approximately 40% of the comorbidity between disorders could be accounted for by common factors.

Conclusions: The data showed that CD and ODD had largely similar social and environmental antecedents. One implication of this finding is that treatment and prevention approaches that are developed for use with a particular behavior disorder may in fact reduce the incidence of both disorders.

Over the past three decades there has been increasing research and debate concerning behavior disorders in childhood and adolescence, including conduct disorder (CD) and oppositional/defiant disorder (ODD) [1-3]. One aspect of this issue that has been studied extensively is the etiology of behavior disorders, and in particular the environmental factors that put young people at increased risk of later CD and ODD. A wide range of studies have examined the social, family background, and individual factors linked to the development of disruptive behavior disorders, and have found evidence for a number of factors that may increase the risk of these disorders [1, 4-7]. These factors include:

Adverse childhood family environment

A number of studies have identified a range of adverse childhood family environmental circumstances that are related to increased risk of behavior disorder later in life. One factor is maternal smoking during pregnancy; evidence suggests that children whose mothers smoked while pregnant are at increased risk of CD and ODD in childhood and adolescence [8-11]. A second factor is exposure to socioeconomic adversity; data show that living in impoverished or adverse socioeconomic conditions during childhood is associated with increased risks of later CD and ODD [12-15]. A third factor is parental maladaptive behavior; evidence suggests that exposure to parental maladaptive behavior, including parental alcoholism, illicit drug use, and criminality, is related to increasing risk of CD and ODD in children and adolescents [16-18]. A fourth factor related to adverse childhood family environment is family instability; a number of studies have shown that family instability in childhood is related to increased risks of disruptive behavior disorder in children [13, 19, 20].

Exposure to child abuse and interparental violence

A further factor that has been linked to increased risks of behavior disorder is exposure to abuse. A number of studies have shown that exposure to sexual abuse and physical abuse in childhood, and exposure to interparental violence, are associated with increased risks of both CD and ODD symptoms in both the short- and long-term [19, 21-23].

Individual characteristics

In addition to the factors discussed above, a number of individual factors have also been linked to increased risks of CD and ODD. These factors include gender, cognitive ability, and deviant peer affiliation. A wide range of studies have shown that behavior disorders are more likely to occur in males than in females [2, 24, 25], and are more likely to occur amongst those with lower levels of cognitive ability [2, 14, 24]. Also, affiliation with

deviant peers in childhood and adolescence has been shown to be linked to increased risks of behavior disorders [2, 24-26]

Background to the present study

One issue arising from the accumulated research on the etiology of behavior disorders is the extent to which the risk profile for each of the two disorders overlaps; that is, is each disorder predicted by the same risk factors, or is it the case that a particular combination of risk factors is more likely to lead to a particular disorder? A number of studies have found evidence that a common set of factors predict each of the two behavior disorders [1-3, 27]. On the other hand, recent evidence has suggested that each individual behavior disorder may have its own distinctive risk profile [28]. Furthermore, recent evidence suggests that the consequences of behavior disorders may in fact be disorder-specific [29].

A related issue is the extent to which common etiological factors may be responsible for the comorbidity between CD and ODD [1]. The links between CD and ODD have been examined in a number of studies. For example, Tuvblad et al, using twin data, found evidence of phenotypic correlations between CD and ODD ranging from .25-.36 [30]. Similar findings were reported by Hewitt, who found a phenotypic correlation between the two disorders of .36 [31]. Burt et al [32], also using twin data, computed estimates of the intraclass correlation between CD and ODD, finding that these ranged from .26 to .35 for parent reports. Also, Dick and colleagues [33] computed polychoric correlations between CD and ODD symptoms, finding correlations ranging from .25 to .42. In addition, a study by Burns et al [34] reported correlations between latent measures of CD and ODD ranging from .62 to .67, and Hewitt et al [31] reported a correlation between latent CD and ODD of .75. These data suggest that there are strong links between CD and ODD, and it could be argued that these links may be largely explained by the influence of etiological factors common to both disorders.

This paper builds on a previous analysis [29] of the outcomes of CD and ODD by examining the predictors of CD and ODD in adolescence (age 14-16 years) in a New Zealand birth cohort. The analysis utilizes a structural equation modeling approach in which latent measures of CD, ODD are simultaneously regressed on a series of predictor variables to examine the similarities and differences in the predictors of each behavior disorder. The general approach is illustrated in Figure 1. In this figure CD1, CD2 and ODD1, ODD2 are assumed to be a set of observed indicators of an underlying latent construct representing the severity of CD and ODD respectively. The two latent constructs are in turn regressed on a series of correlated predictors X1 … Xn. The disturbances of the two latent variables are also permitted to be correlated, reflecting sources of comorbidity between the two constructs that are not explained by the

predictor set. For the purposes of the present analysis the observed indicators CD1, CD2 and ODD1, ODD2 are constructed using split half symptom score measures of CD, ODD symptoms respectively over the period from age 14-16. These indicators are assumed to be linked to the underlying latent constructs via a so-called "true score" model [35] in which the variance in the observed indicators can be partitioned into true score variance reflecting the individual's underlying level of CD or ODD and random error due to unreliability in the observed indicators. Using this approach it thus becomes possible to: a) estimate the associations between a range of predictors and latent indices of CD, ODD that are corrected for measurement unreliability; b) examine the extent to which the associations between the predictors and the latent variables are similar for CD and ODD; and c) examine the extent to which the predictor set may account for the comorbidity between CD and ODD.

INSERT FIGURE 1 HERE

Methods

Data were gathered during the course of the Christchurch Health and Development Study, a longitudinal study of an unselected birth cohort of 1,265 children born in the Christchurch (New Zealand) urban region during a 4-month period in mid-1977. This cohort has been studied at birth, 4 months, 1 year, annual intervals to age 16 years, and at ages 18, 21, 25 and 30. A more detailed description of the study and an overview of study findings have been provided by Fergusson and Horwood [36]. The present analyses were based on the 926 participants for whom information was available regarding behavior disorders during the period 14-16 years and childhood predictors (73.2% of the original sample). The CHDS has received ethical approval from the Canterbury Ethics Committee.

Disruptive Childhood Behaviors 14-16 Years

Information concerning disruptive childhood behavior was obtained at two assessments taking place when the sample members were aged 15 and 16 years. At each age, participants were interviewed on a comprehensive mental health interview that examined aspects of mental health and adjustment over the previous 12 months. A parallel interview was also conducted with the child's mother at each assessment stage. The two interviews were conducted at different sites (mothers were interviewed at home and children at school) and by different interviewers. All information obtained was subject to the signed consent of study participants.

As part of the assessments at each age information was obtained on DSM-III-R [37] symptom criteria for disruptive childhood behaviors, including conduct disorder (CD) and oppositional defiant disorder (ODD) [38]. For child self-report, the assessment of ODD was based on the relevant sections of the Diagnostic Interview Schedule for Children (DISC) [39], whereas CD was assessed using the Self-Report Early Delinquency (SRED) scale [40]. For parental reports ODD was assessed using items from the Revised Behavior Problems Checklist (RBPC) [41], and CD was assessed using a parent version of the SRED. The combined symptom data thus comprised information on DSM-III-R symptom criteria for two separate 12 month periods (ages 14-15 and 15-16 years) from two sources (parent, self-report).

In a previous paper [29] we developed the link from DSM-III-R to DSM-IV symptoms; we assessed 14 of 15 symptom criteria for CD, and 8 of 8 symptom criteria for ODD. These symptom measures were used to create symptom count indicators of CD, ODD symptoms over the two year period from age 14-16 years for use in structural equation modelling. Specifically, the observed symptom measures for CD and ODD were randomly split into two series and the symptoms in each series summed to create the split half symptom counts for each disorder. The measures were based on a count of the number of symptoms of disorder reported by either the mother or child over the two year period. In calculating these measures a symptom was recorded as present if it was reported by either source (mother or child) in either of the interview periods (14-15, 15-16 years). No exclusion criteria were applied to the data for ODD (i.e. it was possible for individuals to have symptoms of both CD and ODD).

Predictors of disruptive childhood behaviors

To examine the developmental antecedents of CD and ODD, a series of measures was chosen from the data base of the study for inclusion in the analysis. These measures were selected on the basis of the above literature identifying factors which previously have been found to be associated with increased risks of CD and ODD. The factors chosen for inclusion in the analyses were as follows:

Exposure to family socioeconomic disadvantage during childhood. For the purposes of the present study, a composite measure of exposure to adverse socioeconomic circumstances in childhood was created, based on measures of: maternal age; maternal and paternal education levels; family socioeconomic status at birth [42]; an average of interviewer-rated family living standards ages 0-10; and an average of decile ratings of family income from ages 0-10. Data reduction was achieved by fitting a single factor model using principal components analysis. Factor score coefficients obtained from the fitted model were then used to create a linear composite representing the extent of family socioeconomic disadvantage (Kaiser-Meyer-Olkin

measure of sampling adequacy = .803). This measure was scored such that higher scores implied greater disadvantage.

Exposure to parental maladaptive behavior. A composite measure of exposure to parental maladaptive behavior was created, based on three dichotomous measures of reflecting any parental history of alcohol problems (measured when cohort members were aged 15 years), criminal offending (measured at age 15) and illicit drug use (measured at age 11). These three measures were summed to create an overall count measure of the extent to which cohort members were exposed to parental maladaptive behavior in childhood.

Family instability (changes of parental figure) to age 14 years. As part of the study comprehensive data on changes of parents were collected at annual intervals. These data were used to construct a measure of family instability based on a count of the number of changes of parents experienced by the child during the interval from birth to the age of 14 years. Changes of parents included changes due to parental separation/divorce, reconciliation, remarriage, cohabitation, parental death, fostering and other changes of custodial parents.

Exposure to abuse and interparental violence in childhood. To create a graded measure of the individual's relative burden of abuse exposure [43], a count measure of types of abuse exposure (sexual abuse; physical abuse; interparental violence) prior to age 16 was created. The measure was calculated by assigning a value of 1 if an individual was exposed to the most severe level of childhood sexual abuse (attempted or completed intercourse), childhood physical punishment (harsh or abusive physical punishment), or was in the highest decile for witnessing interparental violence. For descriptions of each of the individual measures see: [44]. These values were then summed across the three forms of abuse to calculate the count measure.

Maternal smoking during pregnancy. At birth, mothers were questioned about their cigarette smoking during each trimester of pregnancy. Women who reported smoking at least one cigarette per day during any trimester were classified as smoking during pregnancy.

Child cognitive ability. Cognitive ability was assessed at ages 8 and 9 using the Revised Wechsler Intelligence Scale for Children WISC-R: [45]. Total IQ scores were computed on the basis of results on four verbal and four performance subscales. The split half reliabilities of these scores were .93 at age 8 and .95

at age 9. For the purposes of the present analysis the observed WISC-R total IQ scores at ages 8 and 9 were combined by averaging over the two administrations.

*Deviant Peer Affiliations (14 years).*To measure the extent to which the young person affiliated with delinquent or substance using peers at age 14 years, a self report index of peer affiliations was constructed, based on reports of the extent to which the young person's best friend and other friends: used tobacco, alcohol or cannabis, truanted or broke the law. These items were summed to produce a scale measure of the extent to which the young person reported affiliating with delinquent or substance using peers [46].

Gender. Gender was recorded at birth.

Statistical analyses

The analysis was based on the path diagram in Figure 1. The specification of the model and methods of estimation are described below.

Model specification. The model in Figure 1 involves the specification of two linked systems of simultaneous linear equations representing: a) the measurement model linking the observed indicators of CD, ODD to the corresponding latent factors; b) the structural equation model linking the predictor variables X1 … Xn to the latent factors. The model specification is given below. This specification assumes that all variables have been scaled to a mean of zero.

a) Measurement model: The relationships between the observed indicators (CD1, CD2, ODD1, ODD2) and the latent factors (CD, ODD) were given by the following linear equations:

$$CD1 = \lambda1 \; CD + \varepsilon1$$

$$CD2 = \lambda2 \; CD + \varepsilon2$$

$$ODD1 = \lambda3 \; ODD + \varepsilon3$$

$$ODD2 = \lambda4 \; ODD + \varepsilon4$$

where the parameters $\lambda1 - \lambda4$ were factor loadings linking the underlying latent variables to their observed indicators, and the terms $\varepsilon1 - \varepsilon4$ represented random errors of measurement. These errors were assumed to be uncorrelated with each other and with the latent variables CD, ODD. The latent variables were scaled to have a variance of one to ensure that these variables were on a common metric for the purposes of comparison of the regression coefficients for the effects of predictors on latent CD and ODD.

b) Structural model: The linkages between the predictors and the latent constructs were specified by the equations:

$$CD = \Sigma \, B1j \, Xj + U1$$

$$ODD = \Sigma \, B2j \, Xj + U2$$

where Xj $(j = 1 \dots 8)$ were the set of observed predictors; $B1j$, $B2j$ were the regression parameters linking each predictor Xj to the latent outcomes CD, ODD respectively; and U1, U2 were disturbance terms. The predictors Xj were assumed to be uncorrelated with the disturbances U1, U2. However, the disturbances were permitted to be correlated, reflecting covariance between latent CD, ODD that was not explained by the predictor set.

Model estimation. With the above model specification the model was over identified and hence the model parameters were estimable. Model fitting was conducted using Mplus and methods of weighted least squares estimation that produced estimates of standard errors and model chi square that were robust to departures from multivariate normality [47]. Since the observed split half indicators for CD, ODD were markedly skewed these variables were modelled using the techniques described by Muthen [47] for ordered categorical data. For each predictor a chi square test of the equivalence of the regression parameters was obtained by comparing the fit of a constrained model in which the regression parameters for the given predictor were constrained to be equal across CD, ODD with the fit of the unconstrained model. Model goodness of fit was assessed on the basis of the model chi square, the Root Mean Square Error of approximation (RMSEA) and the comparative fit Index (CFI). In well fitting models the RMSEA should be less than .05 and the CFI close to 1 [47].

In the final fitted models reported below the factor loadings for the split half indicators of CD, ODD were constrained to be equal (ie 1 = 2, 3 = 4). This assumption was based on preliminary analyses that showed that constraining the factor loadings to be equal did not result in a significant reduction in model fit. In effect this assumption implies that the split half symptom scores were equally reliable as indicators of their corresponding latent constructs.

Results

Correlations between latent CD, ODD (ages 14-16 years) and predictors

Table 1 shows the estimated bivariate correlations between the latent CD and ODD constructs (see Methods) and each of the predictors, including: family socioeconomic disadvantage; changes of parents; parental adjustment problems; childhood abuse and interparental violence exposure; maternal smoking during pregnancy; gender; IQ; and deviant peer affiliations. The Table shows that:

1. With the exception of gender and ODD, each of the predictors was significantly $(p < .001)$ correlated with both the latent CD and ODD factors. These correlations suggest that higher levels of CD and ODD at

ages 14-16 were associated with greater socioeconomic adversity, greater family dysfunction, exposure to violence, maternal smoking during pregnancy, lower cognitive ability, and higher deviant peer affiliations. In addition, males had significantly higher levels of CD at ages 14-16 than females.

2. The correlations between CD and predictors were typically stronger than the correlations between ODD and predictors. For CD, the correlations with predictors ranged in absolute value from .43 to .17, with a median value of .31. For ODD, the correlations with predictors ranged in absolute value from .36 to .02, with a median value of .27.

INSERT TABLE 1 HERE

Structural modelling of the associations between predictors and latent CD, ODD at ages 14-16 years

As noted in Methods, the analysis was then extended to fit a full structural model in which the latent CD and ODD measures were regressed on all predictors simultaneously. This model showed an adequate fit to the observed data (model X^2 = 20.4, df=13, p=.09; RMSEA = .025; CFI = .998). The results of the fitted model are summarised in Table 2, which shows the standardised regression coefficients for the predictors of latent CD and ODD, the R^2 estimate for each regression, and tests of parameter equality for the effect of each predictor across outcomes, from a fully standardised solution. The Table shows:

1. All eight predictors made significant (p < .05) net contributions to the prediction of latent CD. For ODD, six of the eight predictors were statistically significant (p < .05), the two exceptions being gender and parental maladaptive behaviour. In general, the findings suggest that CD and ODD had very similar risk factor profiles.

2. Consistent with Table 1, the fitted regression coefficients for latent CD were generally stronger than those for ODD. However, tests of parameter equality showed evidence of significant effect size differences for only three predictors. Male gender (p < .001) and socioeconomic disadvantage (p = .014) were more strongly predictive of CD than ODD; whereas child IQ was more strongly related to ODD than CD (p = .045).

3. The R^2 estimate for latent CD was substantially larger than that of latent ODD model (.41 v .27), suggesting that the predictors accounted for a somewhat larger amount of the variance in latent CD than in latent ODD.

INSERT TABLE 2 HERE

Decomposition of correlation between latent CD, ODD (14-16 years)

From the fitted regression model it was possible to estimate the component (i.e. fraction) of the correlation between latent CD and ODD that was explained by the correlated effects of the common predictor set. The estimated bivariate correlation between CD and ODD was .81, reflecting the strong comorbidity between these disorders. The component of correlation explained by the predictor set was .32, leaving .49 unexplained.

Discussion

In this paper we have used data from a longitudinal birth cohort to examine three related issues: a) the extent to which a range of social, family background, and individual predictors were associated with increased risk of later CD and ODD; b) the extent to which the predictors of CD and ODD were similar; and c) the extent to which similarity of predictors could account for the observed comorbidity between CD and ODD. These issues are addressed below.

The predictors of CD and ODD

An important feature of the present study was the availability of extensive longitudinal data that made it possible to examine the childhood social and environmental antecedents of adolescent CD and ODD. This analysis identified a series of domains of development that were associated with increased risks of later CD and ODD.

First, adolescents who showed higher levels of CD and ODD in adolescence were more likely to have been raised in home environments that were subject to multiple social, economic, and related adversity including maternal smoking, socioeconomic disadvantage, family instability, and parental adjustment problems. Second, adolescents with increased levels of CD and ODD were more likely to have had greater exposure to child abuse and family violence. Third, adolescents with higher levels of CD and ODD were more likely to have had stronger affiliations with delinquent and substance-using peers and to be of lower cognitive ability. Furthermore, those adolescents with higher levels of CD were more likely to have been male. This evidence is consistent with a very large body of evidence linking childhood family environment, abuse exposure, and a range of individual factors to increased risk of later CD and ODD [1, 2, 4-7].

Similarity between predictors of CD and ODD

A further feature of the present study was that, through use of structural equation modelling techniques, it proved possible to examine the extent to which the social and environmental predictors of CD and ODD were similar after accounting for the net effects of the complete set of predictors in the model. The data suggested that the CD and ODD had generally similar patterns of predictors, with some exceptions. One exception was that CD was more likely to occur amongst males, whereas gender did not predict ODD.   Also, socioeconomic disadvantage was more strongly predictive of CD than ODD; whereas child IQ was more strongly related to ODD than CD. These data are in general agreement with a number of studies that have suggested that the etiology of CD and ODD is influenced by common factors that increase the risk of both CD and ODD [1-3, 27]. However, the results of the present study are not congruent with recent data suggesting that CD and ODD may have different social and environmental risk profiles [28].   Reasons for these discrepant findings may be that the present study used data from a birth cohort, whereas the study by Petty et al employed data from a case control sample of boys with ADHD, and that the study by Petty et al focused on estimation of familial risks, rather than examining a range of social and environmental factors increasing the risks of both disorders. The present data were also not congruent with those from a recent study by Stringaris and Goodman [48], who found evidence that ODD may be comprised of three sub-dimensions, and that these dimensions may have differing etiological features, as well as etiological features different to those of CD. There may be a number of methodological reasons for these discrepant findings; for example, the Stringaris and Goodman study employed data from a large cross-sectional mental health survey, whereas the present study used data from a longitudinal birth cohort. Also, the Stringaris and Goodman study used data from a range of ages (5-16 years), whereas the cohort members in the present study were the same age. In addition, as noted above, the present study examined the links between observed social and environmental factors related to CD and ODD, whereas the Stringaris and Goodman study focused on the associations between subtypes of behavior disorders. More generally, it could also be argued that the findings of similar antecedents in the present study may be due in part to the high correlation between the latent factors (.81), which is generally stronger than those reported in other studies employing latent CD and ODD [31, 34].

In addition, through the use of structural equation modelling techniques, the present study was able to examine the extent to which the estimated correlation between latent CD and ODD was due to the effects of the set of common predictors. The analyses revealed that around 40% of the correlation between CD and ODD could be accounted for by the net effects of the common risk factors, suggesting a developmental process in which these social and environmental factors act jointly to increase the risk of both CD and ODD.

It could be argued that a developmental process common to both CD and ODD is the cause of the high degree of comorbidity between the two disorders [2, 3].

The results of the present study have implications for the understanding of the nature of disruptive behavior disorders, and for classification schemes such as the imminent DSM-V revision [49]. In an earlier study of the present cohort, the findings suggested that the consequences of CD and ODD in adulthood differed somewhat according to the disorder, lending support to the DSM classification scheme that differentiates between CD and ODD [29]. The results of the present study suggest that, although the consequences of CD and ODD may be distinct, the social and environmental etiologies of the disorders are largely similar. The question then arises as to how to reconcile the two sets of findings. One possibility that has been suggested is that, in many cases, it may be that CD and ODD follow somewhat different courses due to their comorbidity with other disorders, such as internalizing disorders [50]. However, further research is needed to clarify the pathways that lead from common risk factors to CD and ODD, and then on to disorder-specific consequences [1].

The results of the present study also have a number of implications for the treatment and prevention approaches to behavior disorders. One key implication is that, because the social and environmental factors that increase the risk of CD and ODD are largely similar, treatment and prevention approaches that target these factors will likely have the benefit of reducing overall levels of both CD and ODD.

The present study has a number of limitations that should be borne in mind. Perhaps the most important of these was that the assessments of DSM criteria for disruptive behavior disorders were obtained in adolescence. This limitation made it difficult to distinguish between life-course persistent and adolescent limited disorders [51], although it was clear that the majority of those meeting criteria for behavior disorder in adolescence also displayed behavior problems in middle childhood.

A second limitation is that the study was unable to examine the issue of genetic factors with regard to the developmental antecedents of CD and ODD. A number of studies have addressed the issue of the extent to which disruptive behavior disorders are heritable [52]; however, twin data may be one way to provide an estimate of the extent to which the risk of CD and ODD may be linked to genetic sources [53, 54].

An additional limitation in the present study relates to the use of a general SES factor in examining the antecedents of CD and ODD. It could be argued that alternative measurement models, employing multiple measures of SES, could result in observing differences between CD and ODD on more specific SES measures.

A further limitation is that the findings apply to a specific cohort studied over a particular historical period in a specific social context. The extent to which the present findings can be generalised to other

cohort and social contexts requires further investigation. Finally, as with all observational studies, the influence of unmeasured predictors remains a potential threat to study validity.

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Table 1. Estimated correlations between latent conduct disorder and oppositional defiant disorder (14-16 years) and predictors (n = 926)

Predictor	CD	ODD
Family socioeconomic disadvantage	.40	.31
Changes of parents (0-14 years)	.31	.28
Parental maladaptive behavior (0-15 years)	.31	.25
Childhood exposure to abuse/violence (0-16 years)	.32	.26
Maternal smoking during pregnancy	.30	.22
Male gender	.17	.02*
Child IQ (8-9 years)	-.30	-.31
Deviant peer affiliations (14 years)	.43	.36

Note: All correlations statistically significant (p < .001) except those asterisked *

Table 2. Standardized regression coefficients, standard errors and tests of parameter equality for predictors of latent conduct disorder and oppositional defiant disorder (14-16 years) (n = 926)

Predictor	CD		ODD		Parameter test of equality	
	β (SE)	p	β (SE)	p	X^2 (1df)	p
Family socioeconomic disadvantage	.17 (.04)	<.001	.08 (.04)	.041	6.07	.014
Changes of parents (0-14 years)	.06 (.03)	.041	.08 (.04)	.032	0.37	.54
Parental maladaptive behavior (0-15 years)	.09 (.04)	.010	.07 (.04)	.065	0.57	.45
Childhood exposure to abuse/violence (0-16 years)	.14 (.03)	<.001	.11 (.03)	<.001	1.81	.18
Maternal smoking during pregnancy	.14 (.04)	<.001	.09 (.04)	.008	2.30	.13
Male gender	.20 (.04)	<.001	.05 (.03)	.177	20.9	<.001
Child IQ (8-9 years)	-.12 (.03)	<.001	-.19 (.04)	<.001	4.02	.045
Deviant peer affiliations (14 years)	.32 (.03)	<.001	.27 (.03)	<.001	2.97	.09
R^2	.41	<.001	.27	<.001		

Figure 1 Model depicting associations between series of predictors (X_1 to X_n) and latent measures of conduct disorder and oppositional defiant disorder.