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Roles of perinatal problems on adolescent antisocial behaviors among children born after 33 completed weeks: a prospective investigation

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Abstract

Background—There is uncertainty about the extent to which mildly sub-optimal perinatal characteristics among individuals born near-term (>33 weeks of gestation) are associated with various subsequent childhood problems, including antisocial behavior. There is even more uncertainty about whether the pathway to antisocial behavior differs by gender.

Methods—A sample of 1689 infants, born near-term, was followed from birth for over 30 years. Using structural equation modeling (SEM), the study evaluated hypothesized mechanisms linking perinatal problems to antisocial behavior, mediated through the following variables in early and later childhood: neurological abnormalities at age 1; hearing, speech, and language problems at age 3; cognitive function at age 4; and academic performance at age 7. Childhood problems were assessed by trained research clinicians, blind to perinatal status. An ‘antisocial behavior’ variable was created, based on retrospective self-report of six antisocial incidences assessed in adulthood.

Results—Path coefficients showed that birthweight, head circumference, and Apgar scores were indirectly associated with antisocial behavior in the presence of one or more of the following: neurological abnormalities, abnormality in language, speech, and hearing, cognitive function, or academic performance. We found gender differences only in the associations between hearing and IQ and between language perception and IQ. Poor academic performance was associated with antisocial behavior in both boys and girls.

Conclusion—Our hypothesis, that perinatal problems may progress to antisocial behavior when mediated by various markers of early childhood problems, was confirmed. Adverse perinatal events need to be considered in identifying infants who are at risk for academic problems and antisocial behavior, even when the infant is born relatively close to term (i.e., >33 weeks). Poor academic performance, which is indirectly influenced by a variety of neurological and cognitive problems during the perinatal period, infancy, and early childhood appear to increase antisocial behavioral problems in both girls and boys.

Keywords

Perinatal problems; birthweight; head circumference; Apgar scores; childhood problems; antisocial behavior; longitudinal study; hearing; language; intelligence; epidemiology

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A large body of research has demonstrated that perinatal problems such as low birthweight (LBW) and preterm birth are associated with a wide range of developmental problems throughout childhood – including language and speech problems (Kulkarni, Kalantre, Upadhye, Karande, & Ahuja, 2001; Aram, Hack, Hawkins, Weissman, & Borawski-Clark, 1991), poor cognitive functioning (Hack et al., 1994; Aylward, 2002; Litt, Taylor, Klein, & Hack, 2005), and learning difficulties (Klebanov, Brooks-Gunn, & McCormick, 1994; Kulkarni et al., 2001; Roberts, Bellinger, & McCormick, 2006). Recently, emerging evidence suggests that the risk for developmental problems may not be limited to those who had extremely low (<1000g) or very low (<1500g) birthweight, or those born very early (less than 24 weeks). It has also been suggested that the risk for developmental problems in the context of adverse perinatal conditions needs to be examined using continuous (e.g., birthweight in grams) rather than dichotomized (e.g., LBW vs. normal birthweight) measures, and behavior scales normed on population-based cohorts (Kirkegaard, Obel, Hedegaard, & Henriksen, 2006; Richards, Bellinger, & McCormick, 2001; Sorensen et al., 1997; Breslau, Chilcoat, DelDotto, Andreski, & Brown, 1996; Matte, Bresnahan, Begg, & Susser, 2001).

To date, however, no prior study has examined the various long-term implications of perinatal problems in early childhood – including hearing, speech, and language problems, cognitive functioning, and academic performance – on subsequent behavioral problems such as antisocial delinquency. This is especially true for those with milder perinatal problems, whose struggles may only become visible to others as academic demands increase. For this group of children, the trajectory leading to antisocial problems may be highly modifiable. It is also important to examine the nature of behavioral problems and consider possible differences in their meaning in boys and girls. For example, it could be that milder symptoms of noncompliant behavior are equally present in boys and girls, but this same level of early behavior problems might only be a precursor of more serious conduct problems in boys (Robins, 1991; Hinshaw, 1992).

In this study, we utilize data from a randomly selected, population-based sample of children born after 33 gestational weeks to simultaneously examine the influence on subsequent antisocial behavior of: a) three perinatal indicators: birthweight, head circumference, and 5-minute Apgar scores; b) a number of neurological abnormalities detected in infancy; c) hearing, speech and language problems at age 3; d) cognitive functioning (IQ) at age 4; e) academic performance at age 7; and f) adolescent anti-social behavior. We hypothesized that perinatal problems would be associated with a higher prevalence of neurological abnormalities in infancy. We also hypothesized that perinatal problems would be indirectly (mediated via neurological abnormalities) associated with impairments in language, hearing and speech, which in turn would be associated with lower IQ. We further hypothesized that this lower IQ would be directly and indirectly (as a result of problems in the preceding stages) associated with poorer academic performance, and this would then be associated with antisocial behavior. These associations were examined simultaneously within a full-information estimation model.

Methods

Procedures

Data source and participants—Data were derived from the Johns Hopkins Collaborative Perinatal Study site, part of the National Collaborative Perinatal Project (NCP). Prospective data were collected from a random sample of pregnant women who received prenatal care and delivered their babies in the hospital between 1960 and 1964. Data were collected through prospective observation from pregnancy through the first 8 years of life, in an attempt to elucidate pathways during perinatal and early childhood periods that adversely influence subsequent child development (Hardy, 2003). The participation rates at the Johns Hopkins University site were quite consistent over time, with 90% of those recruited participating through the first years of the study and 88% still participating at ages 7 and 8.

A subsequent follow-up study conducted between 1992 and 1994 (PIs: J. Hardy & S. Shapiro) extended the assessment periods from between 7–8 to 27–33 years of age. Of the 2,694 offspring eligible for the follow-up study, 2,220 (82%) were located and 1,758 completed interviews. Overall, the participation rates were 71.4% (of the original sample) for known outcome and 65.3% for outcome confirmed by full interview. Those who were located but did not complete full interviews had mothers with characteristics generally similar to those who were interviewed. The study design, interview procedure, potential biases, and attrition of the sample are described in detail elsewhere (Hardy et al., 1997).

Of the 1,758 offspring who completed the adult interviews, 24 had missing information on gestational age at birth. An additional 48 (2.8%) were born before 33 weeks of gestation, and were excluded from the analysis, leaving 1,686 offspring in this study. Analyses of demographic differences between the 1,686 included and the 1,001 excluded revealed no differences in race (82.6% blacks vs. 87.0%), gender (54.2% female vs. 53.6%), poverty level at birth (46.1% below poverty line vs. 49.2%) and at age 8 (30.6% below poverty line vs. 36.2%), maternal education level (27.3% completed high-school vs. 28.2%), and maternal age at birth (26.5 vs. 25.0).

This study was ruled exempt by the institutional review board at Mount Sinai School of Medicine because it involved secondary data analysis of de-identified data.

Measures

Perinatal problems—Three perinatal outcomes, namely birthweight, 5-minute Apgar scores, and head circumference, were recorded by a nurse observer at the time of delivery. Birthweight was recorded in grams, Apgar scores on a scale ranging from 1 to 10 at 5 minutes postpartum, and head circumference in centimeters. To eliminate the possible confounding effect of preterm birth, we selected participants born after 33 completed weeks (Kirkegaard et al., 2006). Gestational age was based on mother's self-report of her last menstruation period or sonogram estimated gestational age.

Neurological abnormalities at age 1—The neurological examination evaluated the child's central nervous system and a variety of potential developmental abnormalities. The examination was conducted by a trained pediatrician or pediatric neurologist when the child was 50 to 56 weeks old. Overall, 116 items were used to characterize the child's neurological status. These indicators were classified to form groups described as normal, suspect, or abnormal. Abnormalities included 'abnormalities of skull size and shape, spinal anomalies, hemangiomas on the face and head, positional deformities of the feet and unusual facies.' The number of abnormalities reported serves as the outcome measure of neurological abnormalities.

Speech, language, and hearing problems at age 3—The examination, administered by a speech pathologist or audiologist, consisted of five areas: receptive and expressive language; hearing; speech mechanism; and production. These five areas were rated as normal, suspect or abnormal, and assigned a value of 0, 1, and 2 respectively.

Cognitive functioning (IQ) at age 4—The Stanford–Binet IQ (Thorndike, 1972), a widely used instrument which measures general cognitive ability, was administered by a child psychologist when the child was within 3 months of age 4. IQ scores were standardized.

Academic performance at age 7—The Wide Range Achievement Test measured academic abilities (i.e., reading, arithmetic, and spelling) (Jastak & Jastak, 1965). In view of the narrow age range of the sample at the time of testing, we used the raw scores for this analysis. Mean (sd) scores for the three areas were: Reading: 22.3 (.10), Arithmetic: 19.0 (.

08), and Spelling: 30.8 (.20). Ranges were 0–56, 0–32, and 0–76 respectively. These three separate academic ability scores were used to extract a single latent variable of academic performance.

Antisocial behavior—Antisocial behavior data were collected retrospectively during face-to-face interviews by trained researchers blind to any of the childhood variables at the last interview when participants were adults. Measures of antisocial behavior include frequency of playing hooky from school (more than 10 days in one month, less than 10 days in one month, never), getting into trouble at school from fighting (more than once, once, and never), having threatened to hit or hit someone at school (more than once, once, and never), the time of the first incidence of misbehavior at school, the time of the first suspension from school, and the time of the first running away from home. The time of the first occurrence of the problem (misbehavior, suspension from schools, running away from home) was categorized so that the earlier occurrence received a higher score: 0 for absence of the behavior; 1 for ages 15–18 [high-school], 2 for ages 12–14 [junior high-school], 3 for ages 9–11 [4 to 6th grade – upper elementary school grades], and 4 for ages 5–8 [kinder-garten to 3rd grade – lower elementary school grades]. These six observed indicators tapped a single ‘latent’ variable of antisocial behavior based on explanatory factor analysis.

Statistical analysis

We tested our model using SEM, which allows simultaneous testing of all the associations among the different constructs studied and thus assessment of the direct and indirect associations of all predictors, while taking into account a variety of control variables (Linver, Brooks-Gunn, & Kohen, 2002). The Analysis of Moment Structure program (Arbuckle & Wothke, 1999) allows models to be estimated with missing data using the full information maximum-likelihood (FIML) method. It involves the computation of a case-wise likelihood function using all observed variables for a particular case, while including partially complete cases to estimate parameters for the missing data. Monte Carlo studies have shown that the FIML method involves less restrictive assumptions about patterns of missing information, yields unbiased parameter estimates, increases the efficiency of parameter estimates, and eliminates bias in estimation arising from listwise or pairwise deletion and mean substitution of cases (Arbuckle, 1996; Enders & Bandalos, 2001; McArdle & Hamagami, 1996).

Prior to the analysis, the data set was evaluated for normality by examining the univariate indices of skewness and kurtosis for that of 1.96 or more. Path coefficients (standardized beta weights) can be interpreted both in terms of their significance and magnitude. Residual variances were allowed to covary when theoretically meaningful. For example, the error terms of coincident speech, language, and hearing problems at age 3 were correlated. The overall fit of the hypothesized model was evaluated by various indices: a non-significant χ^2 , normed fit index (NFI) closer to 1.00, comparative fit index (CFI) greater than .95, and root mean square errors of approximation (RMSEA) less than .08 were used to indicate a good fit.

A structural equation model was created to test the five hypotheses in the total sample of 1686 and then in multi-group model by gender.

Results

Descriptive and model fit

Table 1 represents the mean, standard deviations and range of the measured variables in the model. Our use of SEM allowed for a complete and simultaneous test of all the associations between perinatal problems, neurological abnormalities, speech, language, and hearing problems at age 3, IQ at age 4, academic performance at age 7, and the six behavioral problems

that were used to estimate antisocial behavior. Cronbach's Alpha for those six observed variables for antisocial behavior showed good internal consistency (Alpha = .71). The correlation matrix of the variables used for testing the model is presented in Table 2. The correlations between the hypothesized relationships were significant but modest. As multiple direct and indirect relationships were tested, all theoretically meaningful variables were retained for model building, even if they showed modest correlation. Note that the use of SEM enables full estimation of the model and the absence of high correlations between variables not directly related to each other does not affect the strength of the overall model.

Overall, our hypothesized model, based on the full sample, had a good fit (NFI = .928, CFI = .943, and RMSEA = .046. 90% CI, .042–.049). We reached this conclusion despite the significant χ^2 test of model fit ($\chi^2(133) = 598.4, p < .0001$), as this test is known to be especially sensitive to large sample size and captures even small deviations from the causal model (Byrne, 2001). Indices for the multi-group model also demonstrated a good fit (NFI = .919, CFI = .948, and RMSEA = .030. 90% CI, .027–.033). However, as was the case in the full model, χ^2 was significant ($\chi^2(266) = 674.52, p < .0001$).

Magnitude of associations and testing the hypothesized pathway

Figure 1 presents the standardized path coefficients for the model. The results show that the perinatal condition led significantly to neurological abnormalities at year 1 (−.08 for Apgar scores: −.08 for head circumference, and −.12 for birthweight). Negative coefficients explain that greater perinatal indicator scores (i.e., heavier birthweight, higher Apgar scores, and larger head circumference) were negatively associated with neurological abnormalities. Neurological abnormalities were positively associated with problems in receptive language (.12, $p < .0001$), expressive language (.18, $p < .0001$), hearing (.13, $p < .0001$), speech mechanism (.08, $p = .003$), and speech production (.17, $p < .0001$). With the exception of abnormality in speech mechanism, the other abnormalities in language, hearing and speech had links to lower IQ (coefficients ranging from −.14 to −.28, all $p < .0001$). There was also a direct path from neurological abnormalities to IQ (−.12, $p < .0001$). Higher IQ then was associated with higher academic performance (.48, $p < .0001$). Finally, higher academic performance was associated with lower level of antisocial behavior (−.13, $p < .0001$).

Magnitude of associations and testing of hypothesized pathways in the multi-group model

The structural coefficients for the model among girls and boys are presented in Figure 2A and Figure 2B respectively. The overall patterns of associations were very similar in girls and boys. The path from perinatal conditions significantly led to neurological abnormalities at year 1. Neurological abnormalities were significantly associated with abnormality in receptive language, expressive language, hearing, speech mechanism, and speech production. Again, except for abnormality in speech mechanism, the other abnormality in language, hearing and speech had significant links to lower IQ. Receptive language had an especially strong association with IQ in girls and hearing had a strong association with IQ in boys. There was a direct path from neurological abnormalities to lower IQ. IQ was, in turn, positively associated with higher academic performance. Finally, academic performance was negatively associated with antisocial behavior.

We further tested whether the structural coefficients for the model differ by gender. Using the twogroup unconstrained model as the comparative model, a fully constrained path model with every pathway and error variance held constant across groups was tested. This model was found to be significantly different from the unconstrained baseline model ($\Delta\chi^2(36) = 72.58, p = .0003$), suggesting there was difference in the magnitude of associations in at least one path between the two groups. Therefore, we further investigated the source of this difference by fitting models holding each individual structural and latent variable path constant (table

available upon request). Although the latent variable for antisocial behavior was not significantly different between boys and girls ($\Delta\chi^2(1) = 1.42, p = .92$), that of academic performance was found to be differently interpreted by them ($\Delta\chi^2(2) = 17.99, p = .0001$). Specifically, the impact of arithmetic was greater in boys and that of reading was greater in girls. Moreover, the magnitude of association between hearing problems and lower IQ was greater in boys than girls ($-.23$ vs. $-.07, \Delta\chi^2(1) = 7.37, p = .007$) and that between language perception and IQ was smaller in boys than in girls ($-.22$ vs. $-.33, \Delta\chi^2(1) = 6.35, p = .01$). None of the other models showed a significant difference between boys and girls.

Discussion

Exploiting the extended time line of the Johns Hopkins NCPP follow-up, systematic and rigorous prospective evaluation of developmental function in early childhood, as well as advanced statistical methodology, this study investigated the relationship between perinatal problems, neurological abnormalities detected within the first year of life, abnormality in language, hearing, and speech at age 3, IQ at age 4, academic performance at age 7, and antisocial behavior in adolescence – in the hope that we would be able to identify mediating conditions for later antisocial behavior among children with near-term birth (>33 weeks). This is potentially important for two reasons. First, if we were able to identify such mediators of outcome, these could theoretically be targeted for remediation, with expected improvement in outcome. Second, we studied a group that excluded children with more serious pre-term (born before 33 weeks) risk, to determine whether the hypothesized associations are present even in near-term births. This latter group is often neglected in the literature. Our findings demonstrate that even among near-term births, suboptimal perinatal indices were related to the presence of neurological abnormalities at age 1; neurological abnormalities then were positively associated with hearing, speech, and language problems at age 3; these specific cognitive deficits were negatively associated with IQ at age 4; IQ was positively related to academic performance at age 7; and academic performance was negatively associated with antisocial behavior in adolescence.

The results of this study provide a basis for understanding the pathways through which perinatal risk factors can influence long-term behavioral outcomes among the near-term births. Further, the focus on mediating factors provides a rationale for developing targeted interventions for children at risk at different developmental stages. This study eliminated possible confounds from cases with severe neurological abnormalities by excluding children born earlier than 33 weeks. This allowed us to consider the potential affect of mild perinatal disadvantage on the trajectory of developmental outcomes. The majority of studies of perinatal risk still focus on children born very early or with very LBW (Hack et al., 1994; Gray, Indurkha, & McCormick, 2004; Litt et al., 2005; Saigal, Pinelli, Hoult, Kim, & Boyle, 2003; Saigal et al., 2006; Rickards et al., 2001). While this strategy is likely to yield the higher percentage of children at risk for severe developmental problems, it also will tend to obscure the more moderate but real risks that are potentially present in children with milder perinatal problems. Our results indicate that, even among children with mild perinatal impairments, developmental deviations are evident in several cognitive domains in early childhood. Our model shows that suboptimal perinatal characteristics lead to increased frequency of neurological problems. Except for one path from speech mechanism to IQ, all the path coefficients were significant at the $<.0001$ level. Most importantly, academic performance at age 7 was also a moderately strong and significant predictor of subsequent anti-social behavior.

Another important finding in this study is that the overall pathway from mild perinatal problems to adverse behavioral outcome was similar in boys and girls, with only a few exceptions. Hearing problems were more strongly associated with lower IQ in boys than girls, while language perception problems in girls were more strongly associated with lower IQ than in

boys. Although reading in girls and arithmetic in boys had greater meaning for academic performance, poor overall performance led to a higher likelihood of antisocial problems in both genders. This underscores the importance of examining learning in both girls and boys in early childhood, and suggests a potentially critical role for early remediation efforts in preventing more serious antisocial behavior. Enhanced awareness of the potential adverse behavioral consequences of early cognitive dysfunction may lead to provision of enriched learning opportunities and educational support, which in turn could modify the trajectory to adolescent antisocial behavioral problems. It is essential that policy-makers understand that risk for antisocial behavioral problems can be identified through screening of early cognitive indicators, in addition to the more frequently studied behavioral measures. This provides a clear rationale for funding programs that maximize early educational attainment, and highlights the likely long-term financial and human benefits such programs can offer to our society.

We need to note that although our hypothesized models were found to have a good model fit as measured by several fit indices such as NFI, CFI, and RMSEA, χ^2 was significant for both full and multigroup models. Ideally, χ^2 statistics should be nonsignificant for good fitting models. However, when the sample size is large, the χ^2 statistic is not considered a reliable index of model fit (Raykov & Marcoulides, 2006; Byrne, 2001), because it becomes significant with even small deviations from a perfect fit. This could lead to falsely rejecting well-fitting models that are tested on the basis of a large sample size, such as ours ($N = 1686$). As all other indices of the model fit showed a good fit, we concluded that our hypothesized models had acceptable fit, despite the significant χ^2 findings.

This study needs to be evaluated in light of its various methodological strengths and weaknesses. Notably, this was a randomly selected cohort, prospectively and systematically followed from birth as a part of the NCPP, and studied longitudinally over 30 years. Birthweight, Apgar scores, and head circumference were recorded by a nurse observer at the time of delivery, a method which is far superior to examining risk indicators culled from mothers' retrospective reports. Further, IQ at age 4 was assessed systematically by trained child psychologists, blind to the child's perinatal risk status. Thus there is little room for subjective bias contributing to measurement errors in these domains.

Despite these considerable strengths, there are also several limitations. First, although the sample was randomly selected and representative of the geographical region, there was an overrepresentation of poor and minority subjects, especially African-Americans; the sample was only 18% Caucasian. This potentially limits external validity to the larger population. Second, gestational age was determined by maternal self-report based on the mother's recollection of the date of her last menstrual period, and determined by sonogram only in the absence of this information. Third, the level of obstetric care in the 1960s was, of course, different than the current standard of care. Our sample was born in the pre-NICU era and the mortality rate for those born very early and with very LBW was higher than it is currently. Also, there has been considerable progress in the development of more extensive special or remedial education, and the realization of early intervention programming, since the late 1960s. It is therefore likely that many of the children with speech, language, and hearing problems in this study would have been offered more effective and targeted remedial assistance before they started their elementary education had they been born today. However, we know that, even today, there are substantive limitations to access to care. We are left to speculate whether children with mild perinatal problems and subsequent cognitive risk factors might have qualified for, received, and benefited from, such services were they available. It is hoped that more specific and accurate delineation of underlying risk mechanisms in subsequent birth cohort studies will enable clarification of the extent to which the risk for antisocial behavior problems can be modified through early cognitive remediation and related interventions. We also acknowledge that we lack information about the family history of antisocial behavior or harsh parenting

practices in our model. Those are factors are widely known as carrying risks for poor academic performance and antisocial problems in offspring. If information regarding those factors had been available, it could have improved our model greatly.

Goodman (1990) proposed that the association between obstetric and neonatal complications (ONCs) and autism could be explained simply by pre-existing autistic liability, and that ONCs were just innocuous markers. It is, therefore, important to remember that an association between perinatal risk and neurological abnormalities in our study does not prove any causal relationship. It is possible, for example, that perinatal risk in our study is a marker of genetic susceptibility, which happens to influence the developmental process, and in turn leads to subsequent antisocial behavioral problems. Although we need to be cautious in interpreting our findings, our study was not designed to investigate the causal link. Rather, it focuses on the time from the perinatal period to later in childhood and adolescence in order to elucidate developmental pathways, and thereby determine critical intervention points. More precise delineation of the interrelationships across multiple perinatal and childhood risk factors, such as poverty, family environment, and peer-relationship problems, will likely yield additional information regarding risk mechanisms, and facilitate the development of more targeted preventive intervention strategies in children born near term with suboptimal birthweight, head circumference, and Apgar scores.

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Figure 1.

Hypothesized model of the relations among perinatal problems, various early childhood impairment and delinquency

Figure 2.
Hypothesized model of the relations among perinatal problems, various early childhood impairment and delinquency among girls (A) and among boys (B)

Table 1
Descriptive statistics for study variables ($n = 1686$)

Variable	M	SD	Min.	Max.
Perinatal indicators (at birth)				
Birth weight (g)	3041	539	1106	4933
5 min Apgar scores	8.84	1.12	1	10
Head circumference	33.6	1.7	25.0	39.0
Neurological abnormalities (age 1)	.56	.98	0	7
Speech, language, and hearing ability (age 3)				
Receptive language	.62	.75	0	2
Abnormal, N (%)	436 (18.3)			
Suspect, N (%)	718 (30.1)			
Expressive language	.36	.60	0	2
Abnormal, N (%)	169 (7.2)			
Suspect, N (%)	580 (24.6)			
Hearing	.24	.48	0	2
Abnormal, N (%)	61 (2.7)			
Suspect, N (%)	440 (19.4)			
Speech mechanisms	.25	.51	0	2
Abnormal, N (%)	84 (3.8)			
Suspect, N (%)	414 (18.6)			
Speech production	.39	.59	0	2
Abnormal, N (%)	133 (5.7)			
Suspect, N (%)	688 (29.5)			
Cognitive functioning (age 4) ^a	93.6	14.2	46	142
Learning abilities (age 7) ^b				
Spelling	22.58	5.2	0	43
Reading	31.50	9.9	0	71
Arithmetic	19.28	4.0	0	31
Antisocial problems				
Run-away from home				
Never, % (N)	82.9 (1398)			
Once, % (N)	11.1 (187)			
More than once, % (N)	5.9 (100)			
Play hooky				
Never, % (N)	31.5 (529)			
Once, % (N)	55.6 (934)			
More than once, % (N)	12.9 (216)			
In trouble at school for fighting				
Never, % (N)	58.8 (990)			
Once, % (N)	17.1 (288)			
More than once, % (N)	24.1 (405)			
(Threaten to) hit a friend				
Never, % (N)	57.7 (972)			
Once, % (N)	10.6 (179)			
More than once, % (N)	31.6 (533)			
Suspension ^c				
Never, % (N)	76.5 (1253)			
At high school, % (N)	10.3 (169)			
At junior high school, % (N)	9.4 (154)			
At elementary school (upper grades), % (N)	2.5 (41)			
At elementary school (lower grades), % (N)	1.2 (20)			
Misbehaving at school ^c				
Never, % (N)	82.7 (1168)			
At high school, % (N)	3.4 (48)			
At junior high school, % (N)	6.5 (92)			
At elementary school (upper grades), % (N)	4.1 (58)			
At elementary school (lower grades), % (N)	3.3 (47)			

^aBased on the Stanford–Binet Intelligence Scale.

^bBased on the Wide Range Achievement Test.

^cThe level of severity was rated depending on the time of occurrence of the event. Never occurred was coded as 0, occurred at high school (ages 14–18) as 1, at junior high school (ages 11–13) as 2, at elementary school – upper grades (ages 8–10) as 3, and at elementary school – lower grades as 4.

Table 2

study variables

	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
**	-.19**	-.09**	-.04**	-.03	-.03	-.08**	.12**	.09**	.10**	.15**	-.01	.06	.04	.03	-.04	.01	.11	.15	.03
**	-.09**	-.02**	-.06**	-.002	-.03	-.05*	.03	.05	.05**	.06**	-.002	-.02	-.02	.03	.03	-.003	-.007	.03	-.02
**	-.17**	-.09**	-.02	-.03	-.02	-.05	.09**	.07**	.08**	.13**	-.01*	.09**	.05**	.05*	-.01	.05*	.12**	.13**	.03
—	—	-.09**	.16**	.11**	.06**	.14**	-.21**	-.20**	.16**	-.22**	.04	-.05*	.02	.04	.01	.06*	.02	.05*	.08**
—	—	—	.38**	.27**	.23**	.33**	-.43**	-.19**	.19**	-.25**	.01	-.05*	.01	-.06	.01	.008	-.12**	.06*	-.07**
	—	—	—	.28**	.23**	.31**	-.39**	-.22**	-.21*	-.23**	-.002	-.05*	.03	-.06	.01	.007	-.02	.03	-.07**
	—	—	—	—	.26**	.31**	-.30*	-.17**	-.18**	-.18**	.002	.03	-.00	-.04†	.03	-.01	-.05*	-.002	**
	—	—	—	—	—	.25**	.16*	-.15**	-.11**	-.15**	.01	.04*	.01	.02	.04	.01	-.01	.04†	-.09**
	—	—	—	—	—	—	-.35**	-.24**	-.22**	-.20**	.02	-.03	.05*	-.02	.007	.001	-.04	.08**	-.11**
	—	—	—	—	—	—	—	.42**	.39**	.47**	-.01	.04**	-.08**	.07**	.06*	-.04	.15**	.01	.19**
	—	—	—	—	—	—	—	—	.85**	.70**	.01	-.03**	-.14**	.03	-.11**	-.04	.03	.11**	.24**
	—	—	—	—	—	—	—	—	—	.71	.01	-.02**	-.14**	-.02*	-.11**	-.05†	.02	-.14**	.25**
	—	—	—	—	—	—	—	—	—	—	.005	.05**	-.09**	-.07**	-.11**	-.05*	.00	-.09**	.21**
	—	—	—	—	—	—	—	—	—	—	—	.16**	.16**	.11	.09	.10	-.16	-.08*	-.07*
	—	—	—	—	—	—	—	—	—	—	—	—	.23**	.19**	.11*	.12**	-.07**	-.02**	*
	—	—	—	—	—	—	—	—	—	—	—	—	—	.33**	.23**	.25**	-.03	.04	-.10**
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	.14**	.13**	-.02	-.01	.02
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	.49**	-.10	-.01	-.14**
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	-.09**	-.01	-.07**
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	.64**	-.01
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	-.17**
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

Appendix Table 3

Goodness of fit statistics for tests of invariance between the two groups (boys and girls)

	χ^2	df	$\Delta\chi^2$ (Δ df), <i>p</i> -value
Unconstrained baseline model	674.52	266	—
Fully constrained model	747.08	302	72.58 (36), .0003
Indicators of antisocial behaviour constrained	675.94	271	1.42(5), .92
Indicators of academic performance constrained	692.51	268	17.99(2), .0001
Academic performance to antisocial behaviour constrained	674.60	267	.06(1), .81
IQ to academic performance	674.54	267	.02(1), .89
Language perception to IQ	680.87	267	6.35(1), .01
Language expression to IQ	675.41	267	.89(1), .35
Hearing to IQ	681.89	267	7.37(1), .007
Speech mechanism to IQ	676.04	267	1.52(1), .22
Speech production to IQ	677.59	267	3.07(1), .08
Apgar to neurological abnormalities	675.00	267	.48(1), .49
BW to neurological abnormalities	674.70	267	.18(1), .67
Head circumference to neurological abnormalities	675.07	267	.55(1), .46