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The Path from Childhood Behavioural Disorders to Felony Offending: Investigating the Role of Adolescent Drinking, Peer Marginalization, and School Failure

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Abstract

Background—Although a pathway from childhood behavioural disorders to criminal offending is well-established, the aetiological processes remain poorly understood. Also, it is not clear if attention deficit hyperactivity disorder (ADHD) is predictive of crime in the absence of comorbid disruptive behaviour disorder (DBD).

Hypothesis—We examined two research questions: (1) Does ADHD have a unique effect on the risk of criminal offending, independently of DBD? (2) Is the effect of childhood behavioural disorders on criminal offending direct or mediated by adolescent processes related to school experience, substance misuse, and peers?

Method—Structural equation modelling, with latent variables, was applied to longitudinally collected data on 4,644 males from the 1986 Northern Finland Birth Cohort Study.

Results—Both ADHD and DBD separately predicted felony conviction risk. Most of these effects were mediated by adolescent alcohol use and low academic performance. The effect of DBD was stronger and included a direct pathway to criminal offending.

Conclusion—Findings were more consistent with the *life course mediation hypothesis* of pathways into crime, in that the effects of each disorder category were mediated by heavy drinking and educational failure. Preventing these adolescent risk outcomes may be an effective approach to closing pathways to criminal behaviour among behaviourally disordered children. However, as there was some evidence of a direct pathway from DBD, effective treatments targeting this disorder are also expected to reduce criminal offending.

INTRODUCTION

Three childhood diagnostic categories have commonly been associated with criminogenic potential: attention deficit hyperactive disorder (ADHD), conduct disorder (CD), and oppositional defiance disorder (ODD). ADHD is a neuropsychiatric condition characterised by developmentally inappropriate levels of inattention, impulsivity, and overactive behaviour (Nyman et al., 2007). CD is described as a persistent pattern of antisocial, aggressive or defiant conduct that extends beyond ordinary levels of childish mischief or adolescent rebelliousness (American Psychiatric Association, 2000). ODD occurs in younger children and is characterized by markedly defiant, disobedient, and disruptive behaviour, but does not include severe aggressive or antisocial behaviour as in CD (American Psychiatric Association, 2000). Disruptive behaviour disorder (DBD) is a more general diagnostic category that has been used to include CD and/or ODD (Karnik et al., 2006)

According to Barkley (1997) ADHD is essentially a clinical equivalent of *low self-control*, an individual trait frequently included in definitions of criminal propensity (Gottfredson & Hirschi, 1990; Rutter et al., 1998). It is therefore not surprising that the relationship between ADHD and criminal behaviour has received considerable attention in the literature (e.g., Farrington et al., 1990; Pratt et al., 2002). A study of prisoners in Germany found that 45% of the inmates meet diagnostic criteria for ADHD (Rösler et al., 2004). The corresponding rates in Finland and Sweden are even higher (Haapasalo & Hämäläinen, 1996; Lindgren et al., 2002). Studies based on self and parent reports have also found strong and consistent associations between ADHD and delinquent behaviour (Bussing et al., 2010; Sibley et al., 2011), but, because ADHD is also related to other behavioural disorders, it is not clear if ADHD alone is a significant risk factor for crime (Lahey & Waldman, 2003; Sibley et al., 2011).

Perhaps comorbid CD is the active ingredient in the “ADHD effect” on criminal offending (Fergusson et al., 2005). Literature is divided on this issue. A recent long-term follow-up of child-psychiatric in-patients found that, independently of conduct disorder, ADHD in childhood was not predictive of adult criminal offending (Mordre et al., 2011). On the other hand, a similar study focusing on individuals diagnosed with ADHD in the absence of CD

documents lifetime rates of criminal offending twice as high as in the non-ADHD comparison group (Mannuzza et al., 2008).

The literature also remains unclear about the *mechanisms* responsible for the relationship between childhood onset behavioural disorders and criminal offending (Loeber & Burke, 2011). The putative processes can be grouped under two major perspectives. First, the longitudinal association may be understood as a simple manifestation of *behavioural continuity*. Theories that treat individual differences in antisocial propensity as the principal source of persistent offending are consistent with this perspective (e.g., Gottfredson & Hirschi, 1990; Moffitt, 1993; Lahey & Waldman, 2003). On the other hand, social-developmental theories (e.g., Catalano & Hawkins, 1996; Sampson & Laub, 1993) argue that the path from early childhood risk factors to adult criminality should be understood as a *life course process* with possibilities for both continuity and change. As Sampson and Laub (1993) have observed, propensity does not equal destiny: most antisocial children do not become criminals, and an antisocial childhood is not a necessary condition for adult involvement in criminal activity (p. 12-15). Thus, while the continuity hypothesis assumes a direct link between behavioural disorders and criminal offending, the life course perspective expects social-contextual factors to mediate this association.

Prior research provides sufficient evidence for us to consider three life course processes as childhood behavioural disorders have been shown to predict adolescent involvement in *drug and alcohol use* (Tuithof et al., 2012), *educational problems* (Fergusson et al., 1993; Rodriguez et al., 2007), and *peer marginalization* (Mrug et al., 2012). Each of these adolescent outcomes has been implicated as a robust precursor of criminal activity (Haynie, 2002; Felson et al., 2008; Maguin & Loeber, 1996). Despite the plausibility of these processes, however, empirical evidence of the role of social-environmental factors remains limited (Loeber & Burke, 2011). Some studies have found support for the hypothesis that problems related to the school domain mediate the influence of early childhood inattention, hyperactivity, and conduct problems on adult rates of violence and criminal offending (Herrenkohl et al., 2001; Savolainen et al., 2012), but another study underscores the significance of adolescent substance abuse in the causal chain linking childhood ADHD to the risk of arrest, conviction, and incarceration (Mannuzza et al., 2008).

Our aim for this study was to examine evidence for *behavioural continuity* hypothesis, which explains any association between childhood behaviour disorders and criminality as a direct expression of stable propensities, such as specific disorder, and for the *life course mediation* hypothesis which argues that early deficits in behavioural regulation set in motion a social-developmental process that materialises over the life course. Our research questions were: (1) Does ADHD have a unique effect on the risk of criminal offending, independently of DBD? (2) Do adolescent risk domains of substance use, peer associations, and academic failure mediate the effects of childhood behavioural disorders on criminal offending?

METHODS

Participants

We used data from the 1986 North Finland Birth Cohort Study (NFBC; see Hurtig et al. (2007) for details about data collection). Information from this longitudinal study of parents and children were complemented with data extracted from government population registries, including health and criminal records (Gissler & Haukka, 2004). Availability of interlinked government registries for research purposes is a unique strength of conducting population research in the Nordic countries. This resource is well-understood in health sciences but remains relatively underused in criminology (Lyngstad & Skardhamar, 2011). Given the low incidence of serious offending among females by age 21, the analysis is limited to male members of the birth cohort ($n = 4,644$).

Measures

Childhood behavioural disorders—The data on childhood behavioural disorders are based on diagnostic records from the Finnish Health Care Registry, which covers specialised medical treatment of any kind, including psychiatric care (Gissler & Haukka, 2004). We identified all individuals with ADHD and/or DBD at any time in their childhood or adolescence, as diagnosed by a child psychiatrist. The reference group consists of those with none of these diagnoses.

Felony offending—The Central Register for Criminal Records covers all court-imposed convictions and fines issued through summary penal proceedings (excluding fixed amount penal fees for minor traffic offences). We had access to data through 2007 allowing us to track officially sanctioned offending from age 15 through age 21 in this cohort. Focusing on the more serious end of the offending continuum, criminal behaviour was measured as a dichotomy indicating the presence of at least one felony-level conviction by age 21.

Adolescent-level mediators—All the measures under this heading are based on self-report data from the adolescent survey administered at age 15; fifteen is the age of criminal responsibility in Finland.

Alcohol use was measured as a latent variable consisting of six indicators ($\alpha=0.83$): (1) age at first intoxication, (2) frequency of alcohol intoxication in the past 12 months, and the more specific frequencies of consuming (3) beer, (4) “alcopop” (e.g. wine coolers and malt beverages), (5) wine, and (6) spirits (e.g. vodka, gin, whiskey). The age at first intoxication was coded into seven categories ranging from never (coded ‘0’) to first intoxication by age 16 (coded ‘6’).

Academic performance is a latent variable based on self-reported performance in core academic subjects ($\alpha=0.76$): Finnish (mother tongue), humanities, mathematics, and science. The response categories are 1=very bad, 2=below average, 3=average, and 4=above average.

Peer marginalisation: Items from the Finnish translation of Achenbach's (2001) Youth Self-Report (YSR) questionnaire were used to measure peer rejection and marginalization. The latent variable is based on responses indicating agreement with six statements

($\alpha=0.707$): (1) I don't get along with others, (2) nobody likes me, (3) I get teased a lot, (4) I feel lonely, (5) I am not liked by other kids, and (6) I feel worthless or inferior.

Index of family adversity—We attended to childhood family characteristics as a potential confounding factor. In the absence of items suitable for one-dimensional scaling, family adversity was measured with a cumulative risk score of eight domains describing circumstances around the time of birth (pregnancy questionnaire) and early childhood (parental questionnaire at child age 7). Following Streiner (2003), we do not report internal consistency for an *index* of “causal indicators” as opposed to a *scale* consisting of “effect indicators”. The items from the pregnancy questionnaire are as follows: (1) parents were not living together (neither married nor cohabiting), (2) mother was unemployed or on disability, (3) father was unemployed or on disability, and (4) mother's years of education was less than 10. The items from the parent survey at age 7 are: (5) father dropped out of school (i.e., did not finish compulsory school), (6) mother dropped out of school, (7) father and (8) mother was unemployed or on disability.

Analyses

The hypothesised causal model (see Figure 1) was tested via structural equation modelling (SEM) conducted in Mplus 7 (Muthén & Muthén, 1998-2012). Although not depicted in the figure, the measure of family adversity was included as a covariate and allowed to covary with ADHD and DBD. As shown, covariances among the disturbances or residuals of the latent mediators (alcohol use, peer marginalisation, and academic performance) were also estimated. Parameter estimates were generated using the weighted least squares means-variance (WLSMV) estimator. WLSMV is appropriate for latent variable models with ordinal indicators (i.e. peer marginalization and academic performance) and dichotomous measured dependent variables (i.e. felony conviction).

As implemented in Mplus, this model incorporates pair-wise missing data procedures to maximise use of the available data from the full sample of 4,644 cases. Model fit was evaluated with the chi-square statistic, the Comparative Fit Index (CFI), and the Root Mean Square Error of Approximation (RMSEA). The chi-square value, which provides an indication of the degree of model misfit in SEM, is sensitive to sample size and often statistically significant in large samples; thus, recommended guidelines suggest that CFI values close to 0.95 or greater and RMSEA values close to 0.06 or less indicate acceptable model fit (Hu & Bentler, 1999). For the mediation tests, bias-corrected bootstrapped 95% confidence intervals were computed to determine the statistical significance of the indirect effects based on 1,000 bootstrap samples (MacKinnon et al., 2004).

FINDINGS

Characteristics of the cohort

Table 1 presents descriptive statistics for the study variables. Seventy-eight (1.7%) of the males had been diagnosed with ADHD alone and 96 (2.1%) with DBD at some stage in the course of medical treatment. These prevalence rates are lower than typically reported which suggests that the symptoms of behavioural disorder need to be unequivocal before they merit

an official diagnosis under the Finnish healthcare system. The mean value (2.1) for the categorised age of first intoxication corresponds to ages 15-16, but this statistic includes those reporting having never been intoxicated (36%). The average values for the four indicators of academic performance show that respondents tended to rate themselves slightly above average. Finally, as expected, felony convictions are rare at this stage of the life course. A total of 434 (9.3%) of the males in the cohort had been convicted of a felony-level offence by age 21. Table 1 reveals a substantial amount of missing data for some of the measures. This was mostly due to attrition as only 67% of the adolescents alive at the time of the follow-up participated in the survey. Attrition analyses conducted elsewhere suggest that this does not bias the estimates (Miettunen et al. 20013).

The fit between the hypothesised model and the data was acceptable (χ^2 (df=154, $N=4644$) = 955.63, $p < 0.05$, CFI=0.95, RMSEA=0.033). Because the estimated model is structurally saturated no degrees of freedom come from the structural portion of the model. The fit of the model is, therefore, identical to that of a confirmatory factor analysis evaluating the measurement model. As shown in table 2, all factor loadings were statistically significant, with standardised values ranging from 0.46 to 0.88. Standardised path coefficients from the structural equation model are shown in Figure 1. The disorder groups had statistically significant positive associations with alcohol use and statistically significant negative associations with academic performance. ADHD but not DBD had a statistically significant positive association with peer marginalisation. Alcohol use and low academic performance were significantly related to the risk of felony conviction. Peer marginalisation was, however, unrelated to the outcome. The direct effect of ADHD on felony conviction was non-significant, whereas the direct effect of DBD was positive and statistically significant.

Although not shown, *The Index of Family Adversity* had a statistically significant positive association with felony offending ($b=0.10$, $p < 0.05$; $\beta=0.11$) and a statistically significant negative association with academic performance ($b=-0.14$, $p < 0.05$; $\beta=-0.22$), but was unrelated to both alcohol use and peer marginalisation in the structural equation model.

Standardised and unstandardised direct, indirect, and total effects are presented in table 3, along with bias-corrected bootstrapped 95% confidence intervals for each parameter estimate. The total indirect effect of each disorder group in relation to felony conviction was statistically significant, operating through both alcohol use and academic performance. As would be expected, none of the specific indirect effects through peer marginalisation was statistically significant. As an indication of effect size, standardised specific and total indirect effects were relatively small in magnitude. Taken together, the disorder groups and the mediators accounted for an estimated 28% of the variance in the risk of felony conviction.

DISCUSSION

Our first important finding was that attention deficit hyperactivity disorder (ADHD) predicts felony conviction risk independently of the hybrid condition of disruptive behaviour disorder (DBD).

Our mediation hypotheses were partly sustained in that this effect was mediated by alcohol use and low academic performance. These associations were, however, quite weak. It does not appear that ADHD by itself is an important correlate of serious offending. The risk of felony conviction was substantially higher for those with DBD. The effect of DBD included both direct and indirect pathways. As with ADHD, heavy drinking and educational failure mediated a substantial share of the DBD effect on felony conviction.

Our finding of substantial evidence for mediation effects supports the utility of the life course approach to mapping aetiological pathways from childhood behavioural disorder to post-adolescent offending. Our results are consistent with prior research which suggested that substance use and educational disengagement in adolescence operate as stepping stones towards adult criminality among behaviourally disordered children (Herrenkohl et al., 2001; Mannuzza et al., 2008; Savolainen et al., 2012). The fact that we did not find evidence of mediation through peer marginalisation does not necessarily mean that peer context is not influential. It is possible that a measure focusing on *delinquent* peer associations would capture the hypothesised processes more effectively (Gudjonsson et al., 2014).

There are additional limitations with some of the measures. Our measure of ADHD does not distinguish those with attention deficits *without* hyperactivity (ADD) from those with it, so it remains possible that problems with hyperactivity alone are responsible for the ADHD effects observed in this research (Babinski et al., 1999). The measures of ADHD and DBD used in this research are based on data obtained from the national health care registry, so must have come to the attention of the health care system. It is unlikely that all the children who meet the clinical threshold of behavioural disorders do so. A study based on a subset of the 1986 cohort estimated the adolescent prevalence of ADHD to be 8.5% in this population (Smalley et al., 2007), suggesting that the measures used in our research cover the tip of the iceberg. We suspect the medical records reflect the more serious end of the diagnostic spectrum, but we cannot be certain. The source of diagnosis has a further implication too. Because they were identified as behaviourally disordered from health care records, the affected children were likely to have received treatment for their disorders. Given that effective treatments for ADHD exist, access to treatment may explain reduced symptoms of externalising behaviour in the ADHD-only category. Although our findings may not, therefore, be generalisable to behaviourally disordered children who have not attended mental health services, the observed patterns of mediation are similar to those reported in other studies based on parent and teacher reports of hyperactivity, conduct problems and inattention (Herrenkohl et al. 2001; Savolainen et al. 2012).

Despite these limitations, our research provides clear indication that heavy drinking and academic failure in adolescence facilitate criminal activity among behaviourally disordered children. Efforts to reduce criminal behaviour in this population may benefit from focusing on preventing substance misuse and educational disengagement in early adolescence. Future research is needed to evaluate the effectiveness of suitable prevention programmes and of the influence of additional environmental processes – such as involvement in antisocial peer groups.

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REFERENCES

- Achenbach, TM. Manual for the ASEBA school-age forms and profiles. University of Vermont, Research Center for Children, Youth & Families; Burlington, VT: 2001.
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSMIV-TR. American Psychiatric Association; Washington, DC: 2000.
- Babinski LM, Hartsough CS, Lambert NM. Childhood conduct problems, hyperactivity-impulsivity, and inattention as predictors of adult criminal activity. *Journal of Child Psychology and Psychiatry*. 1999; 40:347–355. [PubMed: 10190336]
- Barkley, RA. ADHD and the nature of self-control. Guilford Press; New York, NY: 1997.
- Bussing R, Mason DM, Bell L, Porter P, Garvan C. Adolescent outcomes of childhood attention-deficit/hyperactivity disorder in a diverse community sample. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2010; 49(6):595–605. [PubMed: 20494269]
- Catalano, RF.; Hawkins, JD. The social development model: A theory of antisocial behaviour.. In: Hawkins, JD., editor. *Delinquency and crime: Current theories*. Cambridge University Press; New York, NY: 1996. p. 149-197.
- Farrington, DP.; Loeber, R.; Van Kammen, WB. Long-term criminal outcomes of hyperactivity-impulsivity-attention deficit and conduct problems in childhood.. In: Robins, LE.; Rutter, M., editors. *Straight and Devious Pathways from Childhood to Adulthood*. Cambridge University Press; Cambridge, UK: 1990. p. 62-81.
- Felson RB, Savolainen J, Aaltonen M, Moustgaard H. Is the association between alcohol use and delinquency causal or spurious? *Criminology*. 2008; 46:301–324.
- Fergusson DM, Horwood LJ, Lynskey MT. The effects of conduct disorder and attention deficit in middle childhood on offending and scholastic ability at age 13. *Journal of Child Psychology and Psychiatry*. 1993; 34:899–916. [PubMed: 8408374]
- Fergusson DM, John Horwood L, Ridder EM. Show me the child at seven: The consequences of conduct problems in childhood for psychosocial functioning in adulthood. *Journal of Child Psychology and Psychiatry*. 2005; 46:837–849. [PubMed: 16033632]
- Gissler M, Haukka J. Finnish health and social welfare registers in epidemiological research. *Norsk Epidemiology*. 2004; 14(1):113–120.
- Gottfredson, MR.; Hirschi, T. *A general theory of crime*. Stanford University Press; Stanford, CA: 1990.
- Gudjonsson GH, Sigurdsson JF, Sigfusdottir ID, Young S. A national epidemiological study of offending and its relationship with ADHD symptoms and associated risk factors. *Journal of Attention Disorders*. 2014; 18(1):3–13. [PubMed: 22522573]
- Haapasalo J, Hämäläinen T. Childhood family problems and current psychiatric problems among young violent and property offenders. *Journal of the American Academy of Child and Adolescent Psychiatry*. 1996; 35:1394–401. [PubMed: 8885594]
- Haynie DL. Friendship networks and adolescent delinquency: The relative nature of peer delinquency. *Journal of Quantitative Criminology*. 2002; 18(2):99–134.
- Herrenkohl TI, Guo J, Kosterman R, Hawkins JD, Catalano RF, Smith BH. Early adolescent predictors of youth violence as mediators of childhood risks. *Journal of Early Adolescence*. 2001; 21:447–469.
- Hu L, Bentler PM. Covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*. 1999; 6(1):1–55.

- Hurtig T, Ebeling H, Taanila A, Miettunen J, Smalley SL, McGough JJ, Loo SK, Järvelin M, Moilanen IK. ADHD symptoms and subtypes: Relationship between childhood and adolescent symptoms. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2007; 46:1605–13. [PubMed: 18030082]
- Karnik NS, McMullin MA, Steiner H. Disruptive behaviours: Conduct and oppositional disorders in adolescents. *Adolescent Medical Clinics*. 2006; 17(1):97–114.
- Lahey, BB.; Waldman, ID. A developmental propensity model of the origins of conduct problems during childhood and adolescence.. In: Lahey, BB.; Moffitt, TE.; Caspi, A., editors. *Causes of conduct disorder and juvenile delinquency*. The Guildford Press; New York, NY: 2003. p. 76-117.
- Lindgren M, Jensen J, Dalteg A, Meurling AW, Ingvar DH, Levander S. Dyslexia and AD/HD among Swedish prison inmates. *Journal of Scandinavian Studies in Criminology and Crime Prevention*. 2002; 3:84–95.
- Loeber R, Burke JD. Developmental pathways in juvenile externalizing and internalizing problems. *Journal of Research on Adolescence*. 2011; 21(1):34–46. [PubMed: 22468115]
- Lyngstad TH, Skardhamar T. Nordic register data and their untapped potential for criminological knowledge. *Crime and Justice*. 2011; 40(1):613–645.
- MacKinnon DP, Lockwood CM, Williams J. Confidence limits for the indirect effect: Distribution of the product and resampling methods. *Multivariate Behavioural Research*. 2004; 39:99–128.
- Maguin, E.; Loeber, R. Academic performance and delinquency.. In: Tonry, M., editor. *Crime and Justice: A review of research*. Vol. 20. University of Chicago Press; Chicago, IL: 1996. p. 145-264.
- Mannuzza S, Klein RG, Moulton JL III. Lifetime criminality among boys with attention deficit hyperactivity disorder: A prospective follow-up study into adulthood using official arrest records. *Psychiatry Research*. 2008; 160:237–246. [PubMed: 18707766]
- Miettunen, Jouko; Murray, GK.; Jones, PB.; Mäki, P.; Ebeling, H.; Taanila, A.; Joukamaa, M.; Savolainen, J.; Törmänen, S.; Järvelin, MR.; Veijola, J.; Moilanen, I. Longitudinal associations between childhood and adulthood externalizing and internalizing psychopathology and adolescent substance use. *Psychological Medicine*. 2013 available online as *First View* article.
- Moffitt TE. Adolescence-limited and life-course-persistent antisocial behaviour: A developmental taxonomy. *Psychological Review*. 1993; 100:674–701. [PubMed: 8255953]
- Mordre M, Groholt B, Kjelsberg E, Sandstad B, Myhre AM. The impact of ADHD and conduct disorder in childhood on adult delinquency: A 30 year follow-up study using official crime records. *BMC Psychiatry*. 2011; 11:57. [PubMed: 21481227]
- Mrug S, Molina BSG, Hoza B, Gerdes AC, Hinshaw SP, Hechtman L, Arnold LE. Peer rejection and friendships in children with attention-deficit/hyperactivity disorder: Contributions to long-term outcomes. *Journal of Abnormal Child Psychology*. 2012; 40:1013–1026. [PubMed: 22331455]
- Muthén, LK.; Muthén, BO. *Mplus User's Guide*. Seventh Edition. Muthén & Muthén; Los Angeles, CA: 1998-2012.
- Nyman E, Ogdie MN, Loukola A, Varilo T, Taanila A, Hurtig T, Moilanen IK, Loo SK, McGough JJ, Järvelin MR, Smalley SL, Nelson SF, Peltonen L. ADHD candidate gene study in a population-based birth cohort: Association with *DBH* and *DRD2*. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2007; 46:1614–1621. [PubMed: 18030083]
- Pratt TC, Cullen FT, Blevins KR, Daigle L, Unnever JD. The relationship of attention deficit hyperactivity disorder to crime and delinquency: A meta-analysis. *International Journal of Police Science and Management*. 2002; 4:344–60.
- Rodriguez A, Järvelin MR, Obel C, Taanila A, Miettunen J, Moilanen I, Henriksen TB, Pietiläinen K, Ebeling H, Kotimaa AJ, Linnet KM, Olsen J. Do inattention and hyperactivity symptoms equal scholastic impairment? Evidence from three European cohorts. *BMC Public Health*. 2007; 7:327. [PubMed: 17999767]
- Rösler M, Retz W, Retz-Junginger P, Henges G, Schneider M, Supprian T, Schwitzgebel P, Pinhard K, Dovi-Akue N, Wender P, Thome J. Prevalence of attention deficit-/hyperactivity disorder (ADHD) and comorbid disorders in young male prison inmates. *European Archives of Psychiatry and Clinical Neuroscience*. 2004; 254:365–71. [PubMed: 15538605]
- Rutter, M.; Giller, H.; Hagell, A. *Antisocial behaviour by young people*. Cambridge University Press; Cambridge, UK: 1998.

- Sampson, R.J.; Laub, J.H. Crime in the making. Pathways and turning points through life. Harvard University Press; Cambridge, MA: 1993.
- Savolainen J, Hughes LA, Mason WA, Hurtig TM, Ebeling H, Moilanen IK, Taanila AM. Antisocial propensity, adolescent school outcomes, and the risk of criminal conviction. *Journal of Research on Adolescence*. 2012; 22(1):54–64.
- Sibley MH, Pelham WE, Molina BSG, Gnagy EM, Waschbusch DA, Biswas A, MacLean MG, Babinski DE, Karch KM. The delinquency outcomes of boys with ADHD with and without comorbidity. *Journal of Abnormal Child Psychology*. 2011; 39:21–32. [PubMed: 20697799]
- Smalley SL, McGough JJ, Moilanen IK, Loo SK, Taanila A, Ebeling H, Hurtig T, Kaakinen M, Humphrey LA, McCracken JT, Varilo T, Yang MH, Nelson SF, Peltonen L, Järvelin M-RR. Prevalence and psychiatric comorbidity of Attention-Deficit/Hyperactivity Disorder in an adolescent Finnish population. *J Am Acad Child Adolesc Psychiatry*. 2007; 46(12):1575–1583. [PubMed: 18030079]
- Streiner DL. Being inconsistent about consistency: When coefficient alpha does and doesn't matter. *Journal of Personality Assessment*. 2003; 80(3):217–222. [PubMed: 12763696]
- Tuithof M, Have MT, Brink W, van den, Vollebergh W, Graaf RD. The role of conduct disorder in the association between ADHD and alcohol use (disorder). Results from the Netherlands mental health survey and incidence study-2. *Drug and Alcohol Dependence*. 2012; 123:115–121. [PubMed: 22118715]

Figure 1.
Structural Equation Model (Standardised Path Estimates are Reported)

Table 1

Descriptive statistics of analytic variables

Variable	Mean / %	SD	Range	N
Family Adversity Index	1.20	1.17	0-8	4,073
Childhood Behavioural Disorder				
ADHD only	1.7%			78
DBD	2.1%			96
Neither Disorder	96.3%			4,470
Alcohol Use				
Age at first alcohol intoxication	2.06	1.78	0-6	2,781
Intoxication frequency	1.57	1.69	0-6	2,787
Beer consumption	2.25	2.10	0-9	2,787
“Alco pop” consumption	0.47	0.97	0-9	2,781
Wine consumption	0.76	1.21	0-9	2,792
Spirits consumption	1.18	1.52	0-9	2,798
Peer Marginalization				
I feel lonely	0.22	0.47	0-2	3,293
I don't get along with others	0.18	0.47	0-2	3,290
Nobody likes me	0.18	0.43	0-2	3,272
I feel worthless or inferior	0.13	0.37	0-2	3,277
I get teased a lot	0.06	0.26	0-2	3,281
I am not liked by other kids	0.13	0.35	0-2	3,277
Academic Performance				
Finnish	3.05	0.60	1-4	3,309
Humanities	3.14	0.64	1-4	3,306
Mathematics	3.13	0.77	1-4	3,297
Science	3.12	0.68	1-4	3,292
Felony Conviction (1=yes)	9.3%			434

Table 2

Factor loadings from the hypothesised structural equation model

Factor/Indicator	b	SE	β
Alcohol Use			
Age at first alcohol intoxication	1.00 ^r	-----	0.74
Intoxication frequency	1.05 [*]	0.03	0.81
Beer consumption	1.32 [*]	0.05	0.83
“Alco pop” consumption	0.34 [*]	0.01	0.46
Wine consumption	0.57 [*]	0.02	0.62
Spirits consumption	.92 [*]	0.03	0.80
Peer Marginalization			
I feel lonely	1.00 ^r	-----	0.71
I don't get along with others	0.70 [*]	0.04	0.49
Nobody likes me	1.24 [*]	0.04	0.88
I feel worthless or inferior	1.12 [*]	0.04	0.79
I get teased a lot	0.97 [*]	0.05	0.69
I am not liked by other kids	1.14 [*]	0.04	0.81
Academic Performance			
Finnish	1.00 ^r	-----	0.75
Humanities	0.95 [*]	0.03	0.71
Mathematics	0.95 [*]	0.02	0.72
Science	1.09 [*]	0.03	0.82

^r= reference indicator fixed at 1.0 for identification and scaling purposes^{*} p < 0.05.

Table 3
Unstandardised (standardised) total, direct and indirect effects [bias-corrected bootstrapped 95% confidence intervals]

Exogenous Predictor	Mediator				Direct Effect	Total Indirect Effect	Total Effect
	Alcohol Use	Peer Marginalization	Academic performance	Total performance			
ADHD	0.136 (.02) [0.033-0.239]	0.008 (0.00) [-0.042-0.066]	0.166 (0.02) [0.101-0.261]	0.309 (0.04) [0.180-0.473]	-0.201(-0.03) [-0.604-0.169]	0.108 (0.01) [-0.303-0.466]	
DBD	0.341 (0.05) [0.222-0.476]	0.002 (0.00) [-0.010-0.034]	0.252 (0.04) [0.167-0.358]	0.594 (0.08) [0.439-0.768]	0.482 (0.07) [0.201-0.776]	1.076 (0.15) [0.813-1.328]	

Note. Confidence intervals that do not include zero are statistically significant from zero.