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The relationship between childhood conduct disorder and adult antisocial behavior is partially

mediated by early-onset alcohol abuse.

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ABSTRACT

Early-onset alcohol abuse (EOAA) was previously found to both mediate and moderate the effect of childhood conduct disorder (CD) on adult antisocial behavior (ASB) in an American community sample of young adults (Howard, Finn, Gallagher & Jose (2011). This study tested whether this result would generalize to a British forensic sample comprising 100 male forensic patients with confirmed personality disorder. Results confirmed that those in whom EOAA co-occurred with CD showed the highest level of personality pathology, particularly Cluster B traits and antisocial/borderline co-morbidity. Those with co-occurring CD with EOAA, compared with those showing only CD, showed more violence in their criminal history and greater recreational drug use. Regression analysis showed that both EOAA and CD predicted adult ASB when covariates were controlled. Further analysis showed that EOAA significantly mediated but did not moderate the effect of CD on ASB. The failure to demonstrate an exacerbating effect of EOAA on the relationship between CD and ASB likely reflects the high prevalence of CD in this forensic sample. Some implications of these findings are discussed.

INTRODUCTION

While much research attests to the continuity of disordered conduct from childhood into young adulthood and beyond, (e.g. Robins, 1966; 1978; Moffitt, Caspi, Harrington & Milne, 2002), the relationship between childhood conduct disorder (CD) and adult antisocial personality remains problematic for at least three reasons. First, CD appears to predispose to the development of a wide range of adult disorders (Kjelsberg, 2006) and to the entire spectrum of personality disorders rather than just antisocial personality disorder (APD) (Bernstein, Cohen, Skodol, Bezirganian & Brook, 1996; Blackburn, 2007). Consistent with this, Howard, Huband & Duggan (2011) recently reported an association between PD co-morbidity and CD severity. In comparison with PD patients who met only the adult criteria for APD, patients in whom adult antisociality co-occurred with borderline personality disorder (BPD) showed more PD co-morbidity and greater severity of CD. Second, although the rate of progression from childhood CD to adult antisocial personality disorder has been estimated as "around 50%" (Kendall et al., 2009, p.293), variable rates of progression have been reported ranging from 30% (Robins, 1978; Burke, Waldman, & Lahey, 2010) to as high as 61% in adolescents with concurrent substance abuse problems (Myers, Stewart & Brown, 1998). Third, it remains unclear to what extent the presence of childhood CD makes a clinically meaningful difference to adult antisocial personality. Several past studies (e.g. Black & Braun, 1998) suggested that individuals meeting the adult criteria for antisocial personality disorder (APD) suffer essentially the same disorder as those meeting full APD criteria (including CD). However, this was questioned by results of a recent study suggesting the existence of a clinically meaningful distinction between antisocial adults with and without antecedent childhood CD (Walters & Knight, 2010). These authors pertinently remarked: "What we now need is research on the transition from conduct disorder to antisocial personality in order to clarify the nature of this relationship" (p. 267). While factors in addition to childhood CD are clearly at play in determining a shift in the developmental trajectory towards antisocial personality disorder, precisely what these factors are remains unclear.

One highly relevant factor is early-onset alcohol abuse, which confers a significant risk for life-course persistent antisocial behavior and for both violence and heavy alcohol use in late adolescence and early adulthood (Farrington, Ttofi & Coid, 2009; Wells, Horwood & Fergusson, 2004; Buchmann, Schmid, Blomeyer....Laucht, 2009). The co-occurrence of CD and substance abuse in adolescents, particularly among those who subsequently show persistent violence (Soderstrom, Sjodin, Carlstedt & Forsman, 2004), presents a major problem for attempts to unravel their causal role in the etiology of adult antisocial, and in particular violent, behavior.

Some authors have asserted that almost all children and adolescents with CD will abuse alcohol and/or drugs (e.g. DeBrito & Hodgins, 2009). Evidence reviewed here suggests that EOAA and CD exert independent but interacting effects on adult antisociality, so that alcohol and other drug abuse becomes woven into the fabric of disordered conduct (Loeber, Burke, Lahey, Winters & Zera, 2000). First, earlier age of onset of alcohol abuse was found to predict both a range of adult externalizing behaviors, including violent recidivism and life-time aggression, as well as heavy alcohol consumption and violence in young adulthood, even when preceding externalizing symptoms (including CD) were controlled (Gustavson, Ståhlberg, Sjödin et al., 2007; Buchman et al., 2009; Wells et al., 2004). Second, in a study of 477 young adults at high risk for drug and alcohol use, it proved possible to identify a group of individuals who met DSM diagnostic criteria for CD but not for alcohol dependence (Finn, Rickert, Miller et al., 2009). While this CD-alone group were similar to a group having CD combined with alcohol dependence in terms of the age at which their alcohol abuse commenced, the latter (co-morbid) group showed a higher level of lifetime externalizing problems, including drug, alcohol and adult antisocial problems. However, the effect of alcohol dependence was confounded by CD, since severity of CD was greater in the group with both CD and alcohol dependence; moreover, some of the CD group had a history of alcohol abuse but not dependence. Nonetheless, these results were consistent with the concept of multifinality (Cicchetti & Rogosch, 1996), implying that alcohol abuse may have either moderated (i.e. exacerbated) or mediated the relationship between CD and adult antisociality. To verify this, a

further study was undertaken by Howard, Finn, Gallagher and Jose (in press) to examine the possible mediating and/or moderating role of EOAA using the Finn et al. (2009) sample (with alcohol abuse excluded from the CD-only group), using regression analysis to control for co-varying CD and EOAA. Results of the Howard et al. study showed that both EOAA and CD had significant and independent effects on adult antisociality, but that EOAA both significantly mediated and exacerbated the effect of CD.

The present study aimed to see whether the results obtained in a community sample by Howard et al. (in press) would generalize to a high-risk forensic sample comprising patients with PD detained under conditions of medium and high security. In line with previous results obtained by Finn et al. (2009) and Howard et al. (in press), it was predicted, first, that adult antisocial behavior (ASB) would be highest among those who, in addition to meeting DSM criteria for CD, showed significant early-onset alcohol abuse (EOAA). The critical comparison here was with those who showed CD alone. Regression analysis was conducted to test the prediction that both EOAA and CD would independently predict ASB as an outcome, after partialling out effects of covariates. Further analysis was then undertaken to test whether the effect of CD on ASB would be either mediated or moderated by EOAA. According to the hypothesis proposed by Howard (2006), early-onset alcohol abuse acts as a critical variable in mediating, rather than just moderating, the relationship between CD and adult ASB. The latter was the main outcome variable examined, but given the current interest in violence as it relates to personality disorder (e.g. McMurran & Howard, 2009), both severity and quantity of violence in the criminal history were also examined as dependent variables in the group-based analysis.

METHOD

The sample

Participants in this study were male offenders detained at different levels of security under the 1983 UK Mental Health Act category of Psychopathic Disorder. They were recruited from the personality

disorder services at two English high-secure forensic hospitals and one medium-secure hospital. All patients gave their informed consent to participate in the study, which was authorized by the local Research Ethics Committee. Criteria for inclusion were: (i) at least one definite DSM-IV personality disorder (PD) as assessed by the International Personality Disorder Examination (IPDE: (Loranger, Sartorius, Andreoli…Regier, 1999); (ii) a full-scale IQ of 70 or greater (Wecshler, 1997; (iii) no identifiable severe chronic major mental illness, i.e. Axis I diagnoses of psychosis or bipolar affective disorder on DSM-IV (American Psychiatric Association (APA), 1994); (iv) no history of head injury or neurological disorder such as epilepsy. Of those (N = 114) who met the study criteria, 2 refused consent to participate and 12 were excluded because of missing data, leaving a final sample of 100 PD patients, grouped as follows:

Group 1 (no-EOAA/no-CD, N = 17): those with neither a history of DSM IV alcohol abuse or dependence nor a history of CD. These patients lacked both a history of adolescent alcohol abuse (i.e. they consumed less than 42 units of alcohol per week in any continuous six- month period between ages 10 and 20) and a DSM IV diagnosis of CD.

Group 2 (CD-alone, N = 35): those with a DSM diagnosis of CD (3 or more of the 15 CD items scoring positive) but with no lifetime history of alcohol abuse or dependence (DSM) and no history of adolescent alcohol abuse (they consumed fewer than 42 units of alcohol per week over any continuous six-month period between ages 10 and 20).

Group 3 (CD+EOAA, N = 41): those with both a DSM diagnosis of CD (3 or more of the 15 CD items scored positive), a lifetime DSM-IV diagnosis of alcohol dependence/abuse, and a history of adolescent (before age 20) alcohol abuse comprising continuous use (over a 6-month period) of at least 42 units of alcohol per week.

Seven participants showed a history of EOAA and a lifetime history of DSM IV alcohol abuse or dependence without co-occurring CD. These participants were included in the regression analyses (see below) but were excluded for the purpose of between-group comparisons on account of the small sample size.

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Assessment: procedure and instruments.

Consenting patients were recruited into the study by inspection of their case files to ensure they met the inclusion criteria in terms of IQ and clinical diagnosis, as above. Information concerning patients' clinical diagnoses (DSM IV Axes I & II), their history of offending (including index offence) and current psychotropic medication (e.g. antipsychotics and antidepressants) was recorded. Participants then underwent the following assessments:

1. Assessment of psychopathology.

The computerized version of the National Institute of Mental Health Diagnostic Interview Schedule (C-DIS: Robins, Helzer, Cottler & Goldring, 1989) was used to make diagnostic assessments of the following DSM-IV Axis I disorders (APA, 1994): childhood conduct disorder, attention deficit hyperactivity disorder, schizophrenia, bipolar affective disorder and alcohol abuse/dependence. C-DIS has been found to show adequate diagnostic reliability and validity (e.g. Horton, Compton, & Cottler, 1998; Dascalu, Compton, Horton & Cottler, 2001).

DSM-IV PDs were assessed using the International Personality Disorder Examination (IDPE), interview version which has good inter-rate reliability (kappa of 0.70 and above: Zimmerman, 1994) and temporal stability (Loranger et al., 1994). This 99-item semi-structured interview is designed to assess the ten DSM-IV Axis II personality disorders and personality disorder not otherwise specified. Individual IPDE items are scored on a three- point scale (0=absent, 1=partially present, 2=definitely present) allowing dimensional scores to be derived for individual personality disorder categories as well as personality disorder clusters (cluster A, odd and eccentric; cluster B, dramatic; and cluster C, anxious avoidant).

2. Assessment of drug and alcohol use.

Detailed drug and alcohol use histories were obtained using a standardized drug and alcohol assessment developed for use with offenders with mental disorders (for details, see Lumsden et al., 2005). As well as obtaining collateral information from case notes, information was obtained by interviewing patients regarding their early alcohol use (e.g. When did you start to drink alcohol

regularly, say once or more a month? How old were you when you first got drunk?). Information was obtained about how much patients drank in units of alcohol per week across their lifetime. The threshold for early alcohol abuse was defined as consumption of 42 or more units of alcohol per week for at least 6 months continuously before the age 20. We used the number of months in which the individual consumed 42 or more units of alcohol for 6 months continuously before the age 20 to derive a continuous measure of early-onset alcohol abuse (mean=21.19, SD=24.8). This measure was supplemented by two additional measures of EOAA: age when first tasted alcohol (mean =11.4 years, SD=4.4) and age when first got drunk (mean=14.6 years, SD= 4.5). A weighted measure of early-onset alcohol abuse (EOAA) was derived using the sum of the following weighted scores: (i) number of months in which the individual consumed 42 or more units of alcohol per week before age 20: 0 = 0 months; 1 = 1-19 months; 2 = 20-59 months; 3 = more than 60 months; (ii) age when first tasted alcohol; and (iii) age when first got drunk. The scoring for (ii) and (iii) was as follows: 0 = age > 20 years; 1 = age 16-20 years; 2 = age 11-15 years; 3 = age 6-10 years; 4 = age 5 years or younger. Detailed information was obtained about lifetime use of the following classes of drugs: opiates, stimulants, ecstasy, cannabis, hallucinogens, sedatives/tranquillizers, anabolic steroids and solvents.

3. Assessment of violence.

Assessment of violence focused on both quantity and severity of violence. Quantity of violence was based on the number of violent offences in the patient's criminal history, obtained from case files and self-reports. Severity of violence was measured using a severity of violence rating scale (SVRS) adapted from that originally developed by Gunn & Robertson (1976) and validated in hospitalized forensic patients by Wong, Lumsden, Fenton & Fenwick (1993). This comprised 2 sub-scales for each of the admission (index) offence and the previous criminal history[1]. Both were rated on a 5-point scale describing a range of severity from 0 (= no violence) to 4 (= severe violence, e.g. victim died or life and health were seriously endangered).

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[1] An additional scale, severity of violence perpetrated in the institution, was also used in this study but is not considered in the current report.

Analytic strategy.

The analysis proceeded in four stages. In the first stage of the analysis between-group comparisons were carried out using one way ANOVA for continuous variables and the chi-square statistic for categorical variables (see Tables 1 and 2). Tukey test was used in the poc hoc analysis.

The second analysis stage involved multiple linear regression to examine relationships between ASB (total dimensional score summed across all 7 adult APD criteria), which was treated as the dependent variable, and the predictor variables: namely CD (the total dimensional score for conduct problems as measured by the IPDE); and EOAA (weighted score as detailed above), after partialling out the effects of covariates as described below.

The third analysis stage explored the extent to which EOAA moderated the effect of CD on ASB. Moderation was examined using Modprobe, an aid used to probe interactions in ordinary least squares (OLS) and logistic regression (Hayes & Matthes, 2009). It estimates model coefficients and standard errors in a model including a focal predictor (CD), a moderating predictor (EOAA), the product of the two (i.e. the interaction), and any additional covariates to estimate the dependent variable (ASB).

In the final stage of analysis, a multiple mediation procedure developed by Preacher & Hayes (2008) was used to test the prediction that EOAA will mediate, at least in part, the relationship between CD and ASB This initially estimated the direct, indirect and total effects of CD (independent variable) on ASB (dependent variable) through EOAA (proposed mediator). Sobel test (Sobel, 1982) values for the total and specific indirect effects of CD on ASB were estimated. The analysis was then repeated after controlling for covariates.

The data were analyzed using the PASW version 18. Moderation and mediation analyses were tested using corresponding macros obtained from www.afhayes.com.

Testing of potential covariates

The literature on risk factors for antisocial behavior has developed rapidly in the last few decades indicating that a host of risk factors, other than CD and EOAA, may also be associated with adult antisocial behaviour (for a comprehensive review see Farrington, 2010). Factors that have been empirically linked with antisocial behaviour in individuals with mental disorder which were measured in this study include low IQ (Farrington, 1995), age (for a comprehensive account of the link between age and antisocial behavior see Moffitt, 1993), substance misuse (Coid, Kahtan, Gault & Jarman, 1999) and past history of violence in terms of both the quantity and severity (Wong et al, 1993).

Therefore, the effects of the above factors on the dependent variable were partialled out by including them in the regression analysis. Additionally, multicollinearity diagnostics, such as tolerance test and variance inflation factor (VIF), were applied to assess whether the main predictors (i.e. CD and EOAA) had a strong linear relationship with the covariates. The covariates which failed to have significant effects on the parameters of the regression model were excluded from subsequent moderation and mediation analyses as described below.

RESULTS

1. Sample characteristics.

Patients' mean age at the time of assessment was 35.2 years (standard deviation = 9.2; range 21 to 64). All patients had a history of mostly violent offending starting from a young age: mean age of first offence was 15 years (standard deviation = 4.5), and of first violent offence, 18 years (standard deviation = 5.1). Patients had a history of chronic offending, with a mean number of 33 lifetime offences (range 1-154) and of 12.5 violent offences (range 1-135). Most (91%) had received a DSM-IV Cluster B PD diagnosis: antisocial (72%), borderline (47%), histrionic (7%) or narcissistic (13%) PD; fewer received Cluster A (45%) or Cluster C (42%) diagnoses. The mean number of PD diagnoses was 2.9 (SD = 1.5). Almost half (47%) were classified in terms of Tyrer and Johnson's

(1996) severity scale as "severe" (25%) or "very severe" (22%). Three-quarters of the sample (76%) had a history of childhood CD, and a quarter (25%) additionally had a diagnosis of childhood ADHD. A large proportion received co-morbid lifetime diagnoses of major depression and alcohol dependence (56% and 54% respectively).

2. Between-groups comparisons.

Psychometric characteristics of the 3 groups are shown in Table 1 below, and details of their criminal (including violence) and substance abuse (including EOAA) history in Table 2 below. From Table 1 it may be seen that the CD-alone and CD+EOAA groups did not differ from each other in terms of Cluster B traits, CD traits or adult antisociality, but both groups scored significantly higher than the No EOAA/No CD group on these measures, as well as on a dimensional measure of antisocial/borderline co-morbidity. From Table 2 it may be seen that the CD+EOAA group, compared with both other groups, engaged in significantly greater EOAA as well as use of cannabis (likelihood ratio = 22.223, p=0.001) , stimulants (likelihood ratio = 16.494, p=0.001) and opiates (x^2 = 15.012, p=0.002). The CD+EOAA group scored significantly higher than both other groups in terms of the greater quantity of violence shown in their criminal history; they also scored higher on the measure of ASB and showed more *severe* violence in their criminal history compared with the no EOAA/no CD group, but not when compared with the CD only group

TABLES 1 & 2 HERE

3. *Did early-onset alcohol abuse moderate the effect of CD on adult antisocial outcomes?*

Initially, the relationship between CD, EOAA and ASB was examined using correlational analysis. This revealed that EOAA correlated positively and significantly with CD (r^2= 0.376, p <0.001) and ASB scores (r^2= 0.485, p <0.001).The highest correlation was between CD and ASB scores (r^2= 0.501, p <0.001).

Multiple regression analysis revealed that both CD (β=0.371) and EOAA (β=0.346) significantly predicted ASB (p<0.001). The effects of CD (β=-0.278, p=0.02), and EOAA (β=-0.229, p=0.012) on ASB remained significant after partialling out the effects of covariates: namely IQ (β=-0.012, p=0.875), age (β=-0.171, p=0.045), violence quantity (β=-0.017, p=0.842), violence severity (β=0.261, p=0.004) and regular/daily use of cannabis (β=0.017, p=0.845), stimulants (β=0.125, p 0.205), opiates (β=-0.052, p=0.586) and hallucinogens (β=0.170, p=0.032). Tests for multicollinearity (e.g. Menard, 1995; Myers, 1990) showed acceptable tolerance and VIF values (0.86 and 1.2 respectively) indicating that multicollinearity between the predictor variables and covariates was unlikely. However, since violence quantity and regular/daily use of cannabis, stimulants and opiate did not show significant effects on the parameters of the regression model they were excluded from the final regression model and from subsequent analyses.

Results of the moderation analysis using Modprobe (Hayes & Matthes, 2009) indicated that the model accounted for a significant proportion of the variance in the relationship between CD and ASB (r^2=0.3577, F=17.8177, p<0.0001). However, the interaction term for CD and EOAA was not significant, indicating that EOAA did not moderate the effect of CD on ASB.

4. *Did early-onset alcohol abuse mediate the effect of CD on adult antisocial behavior?*

Results of multiple mediation analysis (Preacher & Hayes, 2008) indicated that EOAA significantly mediated the effect of CD on ASB (Sobel's Z= 2.8278, p=0.0047). The indirect effect of CD on adult antisociality through the proposed mediator remained significant even after partialling out the effect of covariates including age, violence severity and hallucinogen use (see figure 1).

FIGURE 1 HERE

DISCUSSION

The current results from a high-risk British forensic sample comprising patients with confirmed personality disorder partially replicated previous findings obtained from an American community sample (Finn et al., 2009; Howard et al, in press). Compared with individuals showing only a history of CD, those showing CD combined with EOAA showed more violence in their criminal

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history and significantly more recreational drug use. Of the 3 groups, the CD+EOAA group scored highest on all measures of personality pathology, particularly Cluster B disorders and antisocial/borderline co-morbidity. Despite this, the group with combined CD and EOAA did not show a significantly greater degree of CD in their history compared with the CD alone group. Nonetheless, CD and EOAA were significantly and positively correlated, confirming findings that that those with a history of CD are more likely to engage in early-onset abuse of alcohol (e.g. Gustavson et al., 2007; Buchmann et al., 2010) and suggesting a reciprocal relationship between CD and adolescent substance use (Loeber et al., 2000).

Further regression analysis demonstrated that both CD and EOAA independently predicted the antisocial outcome. Moreover, the effect of CD on adult ASB was significantly mediated by EOAA, even when covariates (including age, violence severity and hallucinogen use) were partialled out. This suggests that the resulting adult antisocial behavior could be partially predicted by individuals who initially displayed CD in childhood and adolescence and subsequently engaged in alcohol abuse before the age of 20. Nonetheless, CD and substance use likely act reciprocally with each other, so that by late adolescence alcohol abuse becomes woven into the fabric of disordered conduct (Loeber, Burke, Lahey et al. (2000). The finding that EOAA mediates the effect of CD on adult ASB replicates the finding obtained in an American community sample (Howard et al., in press) and is consistent with previous findings: first, that younger age of onset of substance (including alcohol) abuse predicted violent recidivism, CD, and life-time aggression (Gustavson et al., 2007); and second, that early alcohol abuse is a significant risk factor for life-course persistent antisocial behavior (Farrington et al., 2009). Taken together, these findings are consistent with the hypothesis that early-onset alcohol abuse acts as a critical variable in mediating the relationship between disinhibitory childhood psychopathology and adult antisociality (Howard, 2006). This hypothesis further suggested poor emotional self-regulation, consequent upon impaired frontal brain function brought about by adolescent alcohol abuse (De Bellis, Narasimhan,Thatcher, Kashavan, Soloff & Clark, 2005), as the mechanism underlying the link between CD early-onset alcohol abuse

and adult antisociality. This part of the hypothesis was not addressed in the current study and will require verification in future studies.

One notable difference between the current findings and those obtained in an American community sample by Howard et al. (in press) is that EOAA was previously found to significantly moderate (i.e. exacerbate) the effect of CD on ASB. In that study, the effect of EOAA was greatest in those who scored highest on CD, and was minimal in those who scored lowest on CD. In contrast, we could find no evidence in this forensic sample that EOAA significantly moderated the effect of CD on ASB. This discrepancy is likely attributable to differences in the composition of the two samples. In contrast with the previously studied community sample comprising males and females, the current forensic sample were highly deviant offenders, all males, most with a history of serious and often violent offending. All had confirmed personality disorders, often severe and with a high level of PD (particularly antisocial/borderline) co-morbidity as well as co-morbidity with DSM Axis I disorders (particularly depressive disorders). Importantly, over three-quarters of the sample showed a history of CD. This high prevalence of CD in the sample meant that the range of CD scores was restricted, with very few showing absent or low levels of CD symptoms. This restricted range of CD scores would have limited the possibility of finding an interaction between EOAA and CD.

Results of this study have two important implications. First, in order to prevent CD from translating into adult antisocial outcomes, conduct disordered children should be particularly targeted for interventions aimed at preventing them from using alcohol to excess. Second, since early-onset alcohol abuse is both common among antisocial populations (Gustavson et al., 2007; Bakken, Landheim & Vaglum, 2004), and is associated with structural brain changes (DeBellis, Van Vorhees, Hooper et al., et al, 2008), findings of brain abnormalities in antisocial samples should be interpreted cautiously, and only after due consideration has been given to the possibility that they may have arisen as a result of adolescent alcohol and other drug abuse. Indeed, we would

argue that such substance abuse and its neurological consequences are an important part of the etiology of adult antisocial behavior.

Several caveats should be born in mind when interpreting the results of this study. First, this was a relatively small-sized sample, limiting the generalizability of our findings and pointing to the need for replication in a larger forensic sample. The small sample size could have limited the ability to detect a moderation effect of EOAA in the CD-adult antisociality relationship. Second, the study was cross-sectional and assessment of symptoms was retrospective, relying on interviewees being truthful in their responses and accurate in their recollections. This applies particularly to assessment of patients' alcohol abuse history and their CD symptoms. Self-report can result in both false-positive and false-negative errors, particularly for recalled childhood behaviors (Rueter, Chao & Conger, 2000). Third, it should be noted that the group with both CD and early-onset alcohol abuse showed strong evidence of abusing all classes of psychotropic drug, not just alcohol. While this is not surprising, it raises the strong possibility that alcohol acts in synergy with other psychotropic drugs in mediating the link between CD and adult antisocial behavior. Longitudinal studies, rather than the cross-sectional design used here, will be required to verify the relative contribution made by adolescent alcohol *versus* other drug use to adult ASB. Prospective studies will also be required to confirm that EOAA is not simply a reflection of severe CD. Despite the present data indicating that the CD+EOAA group did not differ in CD severity from the CD-alone group, nonetheless inspection of Table 1 indicates the CD+EOAA group showed the highest level of CD severity among the three groups.

In conclusion, the current results confirm previous findings by Howard et al. (in press) in suggesting that, by partially mediating the effects of childhood CD, early-onset alcohol abuse plays a critical role in the etiology of adult antisocial behavior.

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	No-EOAA/no-CD (N = 17)	CD-alone (N = 35)	CD+EOAA (N = 41)
Mean age at assessment (SD)	39.1 (8.2)	36.6 (9.4)	32.3 $(8)_b$*
Mean IQ (SD)	86.1 (13.9)	93 (14.2)	89.6 (11.5)
Cluster A dimensional score$^\beta$	10.2 (7.9)	11.2 (7.3)	10 (6)
Cluster B dimensional score $^\beta$	31.1 (14.6)	39.7 (14)	46.1_{b**} (14.4)
Cluster C dimensional score $^\beta$	8.9 (5.6)	8.9 (7)	10.1 (6.7)
Adult antisocial score $^\beta$	8.1 (3)	9.9 (2.7)	10.7 $(2.9)_{b**}$
Mean conduct disorder dimensional score $^\beta$ (SD)	6.2 (5.6)	11.8 (5.8)	15.4 $(6.5)_b$**
No. meeting full ASPD criteria, n (%)	7 (9.7)	28 (38.9)	33 (45.8)**
Mean APD+BPD dimensional scores $^\beta$ (SD)	22.5 (10.3)	33.0 $(10.3)_c$	37.0_b (11.3)**

Table 1. Psychometric characteristics of the 3 patient groups.
Note: β: Scores derived from International Personality Disorder Examination (IPDE);
b: CD+EOAA differed significantly (*p < .05, **p < .01) from No EOAA/No CD group;
c: CD-alone group differed significantly from No EOAA/No CD group.

	No EOAA/ no CD (N = 17)	CD-alone (N = 35)	CD+EOAA (N = 41)
Early-onset alcohol abuse Mean weighted score (SD)	3.9 (2)	3.9 (2)	6.6 (1.4) $_{a,b}$
regular/daily use of:	N (%)	N (%)	N (%)
Cannabis (%)	7 (11.1)	19 (30.2)	37 (58.7)*
Stimulants (%)	7 (16.7)	8 (18.6)	27 (64.3)*
Opiates (%)	2 (7.4)	5 (18.5)	20 (74.1)*
Hallucinogens (%)	1 (5.9)	5 (29.4)	11 (64.7)
No. violent offences (log trans.) in criminal history	0.7 (0.5)	0.8 (0.3)$_c$	1.0$_{a,b}$ (0.4)*
Severity of violence in criminal history	1.5 (1.2)	2.4 (1.1)	2.7$_b$ (0.8)*

Table 2. History of substance abuse and violent offending in the 3 groups.

Note: Note: a: CD+EOAA group differed significantly from CD-alone group; b: CD+EOAA group differed significantly ($*p < .05$, $**p < .01$) from No EOAA/No CD group; c: CD-alone group differed significantly from No EOAA/No CD group.
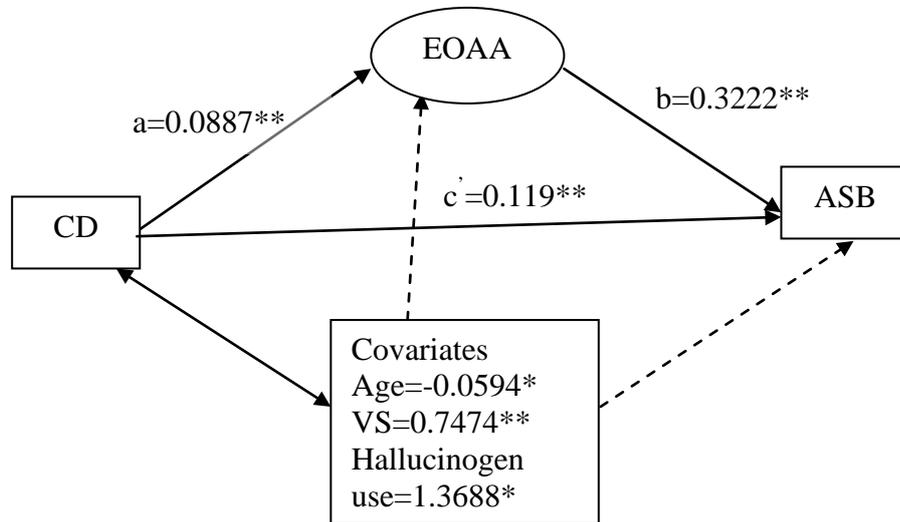
A:

B:

Figure 1: Multiple Mediation Model (after Preacher & Hayes, 2008): Figure 1A shows the total effect of CD (independent variable) on ASB (outcome variable) - path c. Figure 1B depicts the direct effect of CD on ASB (path c') and the indirect effects of CD on ASB via the mediator, namely EOAA (path a-b), controlling for age, violence severity (VS) and hallucinogen use (covariates). The numeric values represent unstandardized coefficients. All the paths are statistically significant confirming the prediction that EOAA partially mediates the effect of CD on adult antisociality.

Note: *$p < 0.05$; **$p < 0.01$