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Alcohol misuse and criminal offending: Findings from a 30-year longitudinal study

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ABSTRACT

Background: This study examined the associations between measures of alcohol abuse/dependence (AAD) and several classifications of offending behaviour to age 30 in a New Zealand birth cohort.
Methods: Outcomes included: assault; use of a weapon; theft/burglary/vehicle conversion; property damage/vandalism/arson; and fraud/embezzlement/misappropriation of funds. The study also used measures of AAD symptoms; and time-dynamic covariate factors including life stress, other substance use, mental health status, peer and partner substance use and offending, and unemployment. Data were analysed using conditional fixed effects regression modelling augmented by time-dynamic covariate factors to control for confounding.
Results: Those with five or more AAD symptoms had unadjusted odds of offending that ranged from 6.23 to 21.25 times higher than those with no symptoms, with little evidence to suggest these associations varied with age. Adjustment for both unobserved fixed effects and time-dynamic covariate factors reduced the magnitude of the associations between AAD and offending, with those with five or more AAD symptoms having odds of offending that were 0.88–4.10 times higher than those with no symptoms. After adjustment, only the associations between AAD and: a) assault (OR = 4.10; 95% CI = 1.91–8.62; $p < 0.0001$); and b) property damage/vandalism/arson (OR = 3.87; 95% CI = 1.30–11.39; $p < 0.0001$); remained statistically significant.
Conclusions: The results suggest a causal association between alcohol misuse and "impulsive" crimes such as assault and property damage/vandalism/arson, with estimates suggesting that AAD accounted for approximately 9.6–9.9% of these types of reported offending in the cohort.

1. Introduction

There has been substantial research into the linkages between the misuse of alcohol and risks of criminal offending (for reviews see: Baumberg, 2006; Bushman and Cooper, 1990; Giesbrecht, 2007; Martin, 2001; Parker, 2004; Rehm et al., 2009; World Health Organization, 2007). In general, this research suggests that the misuse of alcohol has been associated with increased risks of a wide range of offences in many countries (Boden et al., 2012; Bureau of Justice Statistics, 2009; Ensor and Godfrey, 1993; Farke and Anderson, 2007; Fergusson and Horwood, 2000; Gmel and Rehm, 2003; Huckle et al., 2006; Miller et al., 2006; Palk et al., 2007; Scott et al., 1999; Shepherd, 1994; Wood, 2005). While it is now widely accepted that alcohol is a risk factor for crime, there are three general issues that require further investigation and examination.

The first issue concerns the extent to which associations between alcohol misuse and crime reflects a cause and effect association in which the misuse of alcohol lead to an increased risks of crime. Specifically, it may be suggested that the apparent linkages between alcohol misuse and crime reflect the personal, social and other characteristics of those who drink heavily rather than the effects of alcohol misuse on the individual's propensity to offend (Greenland and Morgenstern, 2001; Ward, 2009; Ward and Johnson, 2008). This issue has been addressed in a growing number of studies which have controlled the associations between alcohol misuse and crime using a variety of statistical approaches to adjust for both observed and non-observed sources of confounding (Boden et al., 2012; Fergusson and Horwood, 2000; Fergusson et al., 1996; Flowers et al., 2008; Hingson et al., 2000; Hingson and Zha, 2009; McClelland and Teplin, 2001; Scott et al., 1999). In all cases these studies have reported that associations between alcohol misuse and crime persist following control for various sources of confounding. Of particular note are papers that have used fixed effects regression methods to adjust for non-observed sources of confounding (Boden et al., 2012; Fergusson and Horwood, 2000; Norström and Pape, 2010). These studies suggest that, subject to

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the availability of longitudinal data it proves possible to develop statistical models of the association between alcohol misuse which take into account fixed sources of confounding including common genetic and environmental influences that may confound the association between alcohol misuse and crime. In addition, in the use of these models it is important to account for possible sources of time-dynamic confounding that may also influence the relationship between alcohol and offending. Thus far, however, the weight of the evidence from fixed effects regression approaches suggests the possibility of causal linkages between the misuse of alcohol and odds of crime.

The second issue concerns the effects of age on the associations between alcohol misuse and crime. In particular, it is well-known that rates of both alcohol misuse and crime peak in adolescence and young adulthood (Agnew, 2003; Botvin and Griffin, 2007; Maggs and Schulenberg, 2005; McCarty et al., 2004; Monahan et al., 2009; Steinberg, 2009). It could therefore be suggested that the associations between alcohol and crime are largely or wholly confined to a developmental period spanning adolescence and young adulthood and that this association is reduced or non-existent for older drinkers. At present there is relatively little evidence that addresses this question directly (Lightowlers, 2011; Wei et al., 2004); a recent study by Lightowlers (2011) found that the strongest alcohol-related predictor of violent behaviour at any age was current binge-drinking. In order to understand the associations between alcohol and crime more fully it is necessary to examine possible age-related changes in this association.

Finally, there is a need for research which takes into account heterogeneity in criminal offending. Many studies in this area have examined the linkages between overall rates of criminal offending without discriminating between different types of offences, or have focussed solely on violent offending. However it may be hypothesised that the contribution of alcohol to crime will vary by the type of offence with those offences involving short-term impulsive actions (e.g., assault; vandalism) being more influenced by alcohol than crimes that require some degree of planning and premeditation (Kallmen and Gustafson, 1998; Parker and Auerhahn, 1998). An earlier study (Welte and Miller, 1987) failed to find strong differences in alcohol use amongst individuals who had been incarcerated for either violent crimes or property crimes, but it could be argued that a simple division between violent and property offending may obscure the possible role of disinhibition in various forms of offending.

Against this background, this paper reports the results of a 30-year longitudinal study of the linkages between the misuse of alcohol and odds of crime in a New Zealand birth cohort studied from birth to the age of 30. The aims of this study were to:

1) Estimate the associations between the misuse of alcohol and odds of offending of different classes, including: assault; use of a weapon; theft/burglary/vehicle conversion; property damage/vandalism/arson; and fraud/embezzlement/misappropriation of funds.
2) To adjust associations between alcohol misuse and crime for both observed and non-observed sources of confounding using methods of fixed effects regression.
3) To examine age-related variation in the associations between alcohol misuse and crime.

More generally the aims of this research were to use rich data from a longitudinal study to clarify and elaborate the well-documented associations between alcohol misuse and crime.

2. Methods

2.1. Participants

The data were gathered during the course of the Christchurch Health and Development Study (CHDS). In this study a birth cohort of 1265 children (635 males, 630 females) born in the Christchurch (New Zealand) urban region in mid-1977 has been studied at birth, 4 months, 1 year and annually to age 16 years, and again at ages 18, 21, 25 and 30 years (Fergusson and Horwood, 2001; Fergusson et al., 1989). All study information was collected on the basis of signed consent from study participants and all information is fully confidential. All aspects of the study have been approved by the Canterbury (NZ) Ethics Committee.

2.2. Alcohol abuse/dependence (AAD) symptoms, ages 17–30

In this investigation, alcohol misuse has been operationalized as symptoms of alcohol abuse/dependence (AAD). At ages 18, 21, 25 and 30 years, study participants were interviewed concerning alcohol use using components of the Composite International Diagnostic Interview (CIDI; World Health Organization, 1993) to assess DSM-IV (American Psychiatric Association, 1994) symptom criteria for AAD. At each point of observation a scale score was constructed based on the number of symptom criteria for AAD that the individual met during the preceding 12-month period, with this score ranging from 0 for those meeting no criteria to a maximum of 11 for those meeting all criteria. Participants were categorized using a four-point scale as to the number of symptoms reported at each age. These categories were: 0, 1–2 symptoms, 3–4 symptoms, or 5+ symptoms.

For the purposes of conducting sensitivity analyses, DSM-IV (American Psychiatric Association, 1994) symptom criteria for alcohol dependence were also used to construct a scale score based on the number of symptom criteria for alcohol dependence that each individual met during the 12-month period preceding each assessment, with scores ranging from 0 for those meeting no criteria to a maximum of 7 for those meeting all criteria. Participants were categorized using a four-point scale as to the number of symptoms reported at each age. These categories were: 0, 1–2 symptoms, 3–4 symptoms, or 5+ symptoms.

2.3. Offending outcomes, ages 17–18, 20–21, 24–25, and 25–30

At ages 18, 21, 25, and 30, respondents were questioned about their criminal behaviours since the previous assessment using the Self-Report Delinquency Inventory (SRDI; Elliott and Huizinga, 1989) supplemented by additional custom-written survey items. For the purposes of the present investigation, a number of classifications of types of offending were created using a subset of these questions. This information was used to derive both categorical (yes/no) and count measures of the number of self-reported offenses, across several categories, committed in the year prior to each assessment from age 18 to age 30. The classifications of offences and the items used to create these classifications are described below. For all classifications, participants indicating at least one of these offenses were classified as having committed an offense of that type at that assessment period. In addition, responses to these items were summed to create a count measure of the number of instances of an offense at each assessment period. The classifications included as follows:

2.3.1. Assault. Assault was assessed via responses to three items concerning physical assault and fighting, including: assaulting a person with whom the respondent lived; assaulting a person with the idea of hurting them; and being involved in a gang (group) fight.

2.3.2. Use of a weapon. Use of a weapon was assessed using four items concerning: use of a weapon in a violent assault; using a weapon against someone with whom the respondent lived; aggravated robbery (using a weapon to rob a person or business); or carrying a hidden weapon.

2.3.3. Theft/burglary/vehicle conversion. This category was assessed by several items concerning: petty theft; grand theft (cash or goods over $500); theft from an automobile; breaking and entering; handling stolen goods; shoplifting; burglary; and taking and driving away an automobile without permission.

2.3.4. Property damage/vandalism/arson. These forms of offending were assessed via two items concerning: destroying or damaging property that did not belong to the respondent (vandalism); and setting fire to a house, building, car or other property.

2.3.5. Fraud/embezzlement/misappropriation of funds. Fraud and related offences were assessed by several items pertaining to: obtaining goods via deception; stealing money from employers or others whom had entrusted the respondent with money; failure to pay bills; and benefit fraud.

2.4. Time-dynamic covariate factors (ages 18–30)

In order to examine the extent to which the associations between alcohol misuse and offending could be explained by factors to which individuals were exposed

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at a time contemporaneous to both alcohol misuse and offending, the following measures were abstracted from the study database.

2.4.1. History of previous anxiety and depression. These were assessed via items from the CIDI (World Health Organization, 1993), matched against DSM criteria (American Psychiatric Association, 1987, 1994) to derive dichotomous measures of anxiety disorder (of any sub-type) and major depressive disorder for each year. Participants were classified as having had a history of previous anxiety or depression, from that assessment point forward, if they met DSM criteria for anxiety disorder or major depression at any assessment.

2.4.2. Stressful life events. Assessed by responses to items from the Feeling Bad Scale (Lewis et al., 1984) and custom-written survey items. The life events items spanned such areas as relationship problems and difficulties; serious illness, accident, or death in the family; pregnancy and parenthood; educational or employment difficulties; victimisation; and financial difficulties. Using this information a total life events score was constructed for each year by summing the number of life events reported by the participant in that year.

2.4.3. Cannabis and other illicit drug use. Cannabis use and other illicit drug use were measured by: (a) a six-point categorical measure of cannabis use frequency (never to daily) in the twelve months prior to each assessment; and (b) a dichotomous (yes/no) measure of whether the individual had used any illicit drugs other than cannabis at any point during the 12 months prior to each assessment period.

2.4.4. Unemployment. Assessed for the 12 months prior to each assessment by asking participants about their experience of unemployment in each period and classified into four levels reflecting the duration of unemployment in the year; never unemployed; <3 months, 3–5 months; 6+ months.

2.4.5. Peer and partner substance use and offending. Measured for the twelve month period prior to each assessment on the basis of four single items assessing: the extent to which the participants' romantic partners and friends: (a) used illicit drugs or had problems resulting from alcohol or illicit drugs, and (b) engaged in criminal offending, had problems with aggressive behaviour or were in trouble with the law. The questions assessing peer substance use and offending queried the relative percentage of the individual's friends who engaged in each of these behaviours (none, some, most).

2.4.6. Conduct/anti-social personality disorder. Measured at age 18 using the Self Report Delinquency Inventory (SRDI: Elliott and Huizinga, 1989). In order to use a more age-appropriate instrument, from age 21 onwards conduct/anti-social personality disorder was assessed using custom-written survey items reflecting the DSM-IV criteria for anti-social personality disorder. Sample members who met diagnostic criteria for conduct disorder or anti-social personality disorder during an assessment period were classified as having the disorder during that period.

2.5. Statistical analyses

2.5.1. Associations between AAD and offending. In the first stage of the analyses, the pooled association between AAD symptoms and each offending outcome was estimated via Generalized Estimating Equation methods (Liang and Zeger, 1986; Zeger and Liang, 1986) to fit a population-averaged logistic regression model in which the risk of each outcome each time period was modelled as a function of AAD symptoms during each time period. These models were of the form:

$$f(Y_{it}) = B_0 + B_1 X_{it} \quad (1)$$

where $f(Y_{it})$ was the log odds of each offending outcome reported by the ith subject in a given interval t (t = 17–18 years; 20–21 years; 24–25 years; and 29–30 years), and X_{it} represented AAD symptoms during the interval t. In this model observations from the same individual over time were permitted to be correlated with an unstructured correlation matrix. From the fitted models, estimates of the odds ratio (OR) and 95% confidence intervals (CI) of AAD symptoms for violence outcomes were calculated. In addition, these models were extended to include terms representing: (a) time; and (b) a time period × AAD symptoms interaction, in order to account for the possibility that rates of AAD symptoms varied over time.

2.5.2. Fixed effects model for covariate adjustment. To adjust the associations between AAD symptoms and violence outcomes for both: (a) non-observed fixed effects; and (b) observed time-dynamic confounding factors, conditional fixed effects regression models were fitted to the joint data for each of the four outcomes over the measurement periods. These models were of the form:

$$f(Y_{it}) = \alpha_i + B_1 X_{it} + \sum B_k Z_{ikt} \quad (2)$$

In this model α_i are individual specific terms that are assumed to reflect the effects of all fixed sources of variation in the outcome Y_{it}. The fixed effects α_i are assumed to be constant over time and to be correlated with other predictors in the model. The models were also augmented by the terms Z_{ikt}, representing the set of observed time-dynamic covariates.

Estimates of the population attributable risk (PAR) for each offending outcome that remained statistically significant after adjustment were calculated using methods described by Bruzzi et al. (1985), after adjustment for both non-observed fixed effects and time-dynamic covariate factors.

In addition, the analyses above were repeated using a count measure of the number of offences in place of the dichotomous measure at each age, with negative binomial regression models used in place of logistic regression. In all cases the count measures of offending outcomes were truncated to a maximum of 50 offences per individual in order to avoid the undue influence of extreme outliers.

Also as noted previously, the above analyses were repeated using a categorical measure of the number of symptoms of alcohol dependence in place of the categorical measure of the number of symptoms of AAD.

2.6. Sample sizes

The present analyses were based on samples ranging from 987 to 1025, representing 78–81% of the original cohort of 1265 participants, for whom data were available concerning AAD and offending outcomes at ages 18, 21, 25, and 30. To examine the effects of sample losses on the representativeness of the sample, the obtained samples with complete data at each age, were compared with the remaining sample members on a series of socio-demographic measures collected at birth. This analysis suggested that there were statistically significant ($p < 0.01$) tendencies for the obtained samples to under-represent individuals from socially disadvantaged backgrounds characterized by low parental education, low socio-economic status and single parenthood. To address this issue, the data weighting methods described by Carlin et al. (1999) were used to examine the possible implications of selection effects arising from the pattern of missing data. These analyses produced essentially the same pattern of results to those reported here, suggesting that the conclusions of this study were unlikely to have been influenced by selection bias.

3. Results

3.1. Associations between symptoms of AAD and offending categories, ages 18–30

Table 1 shows the associations between AAD and a series of five self-reported offending categories at ages 17–18, 20–21, 24–24, and 29–30. For each category of offending, the percentage reporting at least one instance of that crime for each level of AAD symptoms (0, 1–2, 3–4, 5+) during the 12 months prior to each assessment period is shown. The Table also shows estimates of the population-averaged odds ratio (OR) and 95% confidence interval (CI) for the pooled association between AAD symptoms and each category of offending, derived from generalized estimating equation (GEE) logistic regression models (see Section 2). In addition, the Table reports on tests of age × AAD symptoms interactions derived from the GEE models. The table shows:

1. There were statistically significant associations ($p < 0.0001$) between AAD symptoms and each category of self-reported offending, with those reporting higher levels of AAD symptoms having significantly increased odds of reporting offending. Those with five or more symptoms of AAD had odds of offending that ranged from 6.23 times to 21.25 times higher than those reporting no symptoms.
2. Examination of the overall odds of offending categories show that odds of offending decreased as age increased. The main effect for age was statistically significant ($p < 0.0001$) for all five of the offending categorizations.
3. For one outcome (theft/burglary/vehicle conversion) there was evidence of a statistically significant ($p < 0.05$) age × AAD symptoms interaction, and there was one marginally significant ($p < 0.10$) age × AAD symptoms interaction (use of a weapon). In both cases, the age × AAD symptoms interaction term was used in all subsequent analyses. In all other cases, there was no evidence of statistically significant age × AAD symptoms interactions, suggesting that for those outcomes the strength of association between AAD symptoms and outcomes did not vary by age.

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Table 1
Associations between symptoms of alcohol abuse/dependence and types of offending, ages 18–30.

Outcome/age (% reporting)	Number of symptoms of AAD				Overall %	p
	0	1–2	3–4	5+		
Assault						
Age 17–18	10.5	21.0	50.9	70.0	17.0	
Age 20–21	3.6	12.4	20.6	28.9	7.0	
Age 24–25	3.1	10.8	20.5	18.2	5.1	
Age 29–30	3.5	5.2	11.1	50.0	4.3	
Population-averaged OR (95% CI)	1	2.28	5.20	11.85		<0.0001
	–	(2.02–2.56)	(4.08–6.55)	(8.24–16.78)		
Test of age x AAD symptoms interaction	LR$\chi^2(1) = 2.36$, $p > 0.10$					
Use of a weapon						
Age 17–18	2.6	8.7	16.4	30.0	5.5	
Age 20–21	1.5	3.7	11.1	13.2	2.9	
Age 24–25	0.7	3.1	2.6	4.5	1.2	
Age 29–30	0.9	3.1	0.0	0.0	1.1	
Population-averaged OR (95% CI)	1	1.84	3.38	6.23		<0.0001
	–	(1.41–2.40)	(1.99–5.76)	(2.80–13.82)		
Test of age × AAD symptoms interaction	LR$\chi^2(1) = 3.79$, $p < 0.10$					
Theft, burglary, vehicle conversion						
Age 17–18	7.8	21.7	34.5	68.0	14.0	
Age 20–21	3.2	14.9	20.6	31.6	7.2	
Age 24–25	2.1	8.5	7.7	13.6	3.4	
Age 29–30	1.8	5.2	0.0	25.0	2.2	
Population-averaged OR (95% CI)	1	2.04	4.16	8.49		<0.0001
	–	(1.72–2.43)	(2.96–5.90)	(5.09–14.35)		
Test of age × AAD symptoms interaction	LR$\chi^2(1) = 5.00$, $p < 0.05$					
Property damage, vandalism, arson						
Age 17–18	2.8	10.9	30.9	42.0	7.3	
Age 20–21	1.1	8.1	14.3	28.9	4.1	
Age 24–25	1.4	4.6	7.7	13.6	2.3	
Age 29–30	0.4	2.1	0.0	12.5	0.6	
Population-averaged OR (95% CI)	1	2.77	7.67	21.25		<0.0001
	–	(2.36–3.25)	(5.57–10.56)	(13.14–34.33)		
Test of age × AAD symptoms interaction	LR$\chi^2(1) = 1.43$, $p > 0.20$					
Fraud, embezzlement, misappropriation of funds						
18	2.3	8.0	18.2	42.0	5.9	
21	2.8	13.7	15.9	28.9	6.3	
25	3.0	6.9	5.1	22.7	4.0	
30	0.6	2.1	0.0	12.5	0.8	
Population-averaged OR (95% CI)	1	2.02	4.08	8.24		<0.0001
	–	(1.80–2.27)	(3.24–5.15)	(5.83–11.70)		
Test of age × AAD symptoms interaction	LR$\chi^2(1) = 0.52$, $p > 0.40$					
N observed						
18	782	138	55	50		
21	749	161	63	38		
25	812	130	39	22		
30	855	97	27	8		

4. In addition, there were also trends for rates of AAD symptoms to decline with age ($p < 0.0001$).

3.2. Associations between AAD symptoms and offending outcomes, adjusted for (a) non-observed confounding factors; and (b) time-dynamic covariate factors

In order to examine the extent to which the associations between AAD symptoms and offending outcomes could be explained by the influence of: (a) sources of non-observed fixed confounding; and (b) time-dynamic covariate factors occurring contemporaneously with AAD symptoms and offending outcomes, the analyses depicted in Table 1 were extended over by fitting conditional fixed effects logistic regression models to the data, augmented by a series of observed time-dynamic covariate factors measured contemporaneously with both AAD symptoms and offending outcomes (see Section 2). The observed time-dynamic factors reflected each individual's exposure to: stressful life events, associating with deviant peers and partners, and substance use and mental health issues that may have influenced the observed bivariate associations between AAD symptoms and offending outcomes. The results of these analyses are shown in Table 2, which shows the adjusted ORs and 95% CIs for the associations between AAD symptoms and each offending outcome, after adjustment for: (a) non-observed sources of confounding; and (b) time-dynamic covariate factors. The table shows:

1. Adjustment for non-observed confounding and time-dynamic covariate factors reduced the magnitude of the associations between AAD symptoms and offending outcomes. After adjustment, the associations between AAD symptoms and: (a) use of a weapon; (b) theft/burglary/vehicle conversion; and (c) fraud/embezzlement/misappropriation of funds; were reduced to statistical non-significance (all p values > 0.05).
2. Although adjustment for fixed effects and time-dynamic covariate factors reduced the magnitude of the association between AAD symptoms and: (a) assault; and (b) property damage/vandalism/arson; the associations remained statistically significant ($p < 0.05$). After adjustment, those having five or more symptoms of AAD having odds of offending that ranged from 3.87 to 4.10 times higher than those with no symptoms of AAD.

In general, the results of the analyses suggest that the linkages between alcohol misuse and offending outcomes varied depending

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Table 2
ORs (and 95% CI) for the associations between alcohol abuse/dependence (AAD) symptoms and types of offending, after adjustment for non-observed fixed effects and time-dynamic covariate factors.

Measure	Number of symptoms of AAD				*p*
	0	1–2	3–4	5+	
Assault	1	1.60	2.56	4.10	<0.0001
	–	(1.24–2.05)	(1.53–4.20)	(1.91–8.62)	
Use of a weapon	1	0.96	0.92	0.88	>0.80
	–	(0.59–1.55)	(0.35–2.40)	(0.21–3.72)	
Theft, burglary, vehicle conversion	1	1.17	1.37	1.60	>0.30
	–	(0.83–1.64)	(0.69–2.69)	(0.57–4.41)	
Property damage, vandalism, arson	1	1.57	2.46	3.87	<0.05
	–	(1.09–2.25)	(1.19–5.06)	(1.30–11.39)	
Fraud, embezzlement, misappropriation of funds	1	1.32	1.74	2.3	<0.10
	–	(0.95–1.84)	(0.90–3.39)	(0.86–6.23)	

Note: Statistically significant ($p < 0.05$) time-dynamic covariate factors included: history of anxiety and depression; stressful life events; cannabis and other illicit drug use; unemployment; peer substance use and offending; partner substance use and offending.

upon the nature of the offence in question. For those offences that may be related to impulsive behaviour (assault, property damage/vandalism/arson), the findings suggest that alcohol misuse was related to increased risks of these outcomes, even after controlling for possible confounding. However, the analyses suggest that the observed linkages between alcohol misuse and other forms of offending that may appear to require more planning and coordination (use of a weapon; theft/burglary/vehicle conversion; fraud/embezzlement/misappropriation of funds) were largely explained by a range of sources of potential confounding.

3.3. Population attributable risk (PAR)

As noted in Section 2, estimates of the PAR for the role of AAD in the outcomes that remained statistically significant after adjustment (assault; property damage) were calculated using methods described by Bruzzi et al. (1985), after adjustment for fixed effects and time-dynamic confounding factors. These analyses showed that 9.9% of assault and 9.6% of property damage/vandalism/arson could be accounted for by the presence of AAD.

3.4. Supplementary analyses

As described in Section 2, the analyses reported above were repeated using count measures of the number of offending in each category, in place of the dichotomous measure of the percentage reporting each crime category at each age period. Repeated measures negative binomial GEE models were used to model the associations between AAD symptoms and the count measures of offending categories, and to adjust the associations for both non-observed confounding and time-dynamic covariate factors. In all cases the results of these analyses were congruent with those reported above, suggesting that the use of count measures in place of percentage measures did not materially alter the conclusions drawn by the study.

Also as described in Section 2, the analyses reported above were repeated using a categorical measure of the number of symptoms of alcohol dependence in place of the categorical measure of AAD symptoms. In all cases the results of these analyses were congruent with those reported above.

4. Discussion

The present study is a partial replication and extension of the findings from the same CHDS cohort presented by Boden et al. (2012), using similar methods. The earlier study found evidence of persistent associations between AAD symptoms and violence involvement (violent offending/victimisation/intimate partner violence perpetration) after controlling for both non-observed sources of confounding and time-dynamic covariate factors. In the present paper, we examined the linkages between AAD symptoms and odds of perpetration of a range of different categories of crime (including both violent crime and property crime), over the period from ages 17–30. One key purpose of the present paper was to determine whether the linkages between alcohol misuse and crime were similar across different types of crime, or whether particular kinds of criminal offending were more sensitive to the effects of alcohol misuse. The key findings and their implications are summarised below.

First, consistent with previous studies examining the linkages between alcohol misuse and crime (Bureau of Justice Statistics, 2009; Farke and Anderson, 2007; Gmel and Rehm, 2003; Huckle et al., 2006; Palk et al., 2007; Scott et al., 1999), this study produced evidence of bivariate statistical associations between the extent of alcohol misuse and all types of crime at all ages. In general, increasing rates of AAD symptoms were associated with increased risks of: assault; use of a weapon; theft/burglary/vehicle conversion; property damage/vandalism/arson; and fraud/embezzlement/misappropriation of funds. Compared to those with no symptoms of AAD, those with five or more symptoms had odds of crime that were 6–21 times higher. Further, these associations remained relatively stable over the period from 17 to 30 years, and in only one case was there evidence of a statistically significant age × AAD symptoms interaction. On the basis of this there is little doubt that there were strong associations between the misuse of alcohol and all types of crime with these associations being evident up to the age of 30. However, the study findings also showed that both odds of offending and rates of AAD symptoms declined with age. The net effects of these age-related changes was that while the relationships between alcohol misuse and crime were evident at all ages, the number of alcohol-related crimes declined with age.

Further analyses of the associations between offending and AAD showed that much of this apparent association was spurious and explained by common confounding factors that were associated with both alcohol misuse and crime. Specifically control for common fixed effects and time-dynamic sources of confounding reduced the ORs for those with five or more AAD symptoms to between to 0.88 to 4.10. After control for all factors, the misuse of alcohol was associated with only two outcomes: assault (AOR = 4.10; 95% CI: 1.91–8.62; PAR = 9.9%); and property damage/vandalism/arson (AOR = 3.87; 95% CI: 1.30–11.39; PAR = 9.6%). These findings are consistent with the conjecture made in the Introduction that the effects of alcohol misuse are most likely to be associated with crimes involving impulsive behaviours, and least likely to be involved in crimes requiring planning and forethought.

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It is also interesting to note that the present study suggests that much of the association between AAD and crime is spurious and arises from common factors that are associated with both AAD and crime (Greenland and Morgenstern, 2001; Ward, 2009; Ward and Johnson, 2008). These factors may include both common genes that influence the propensity to alcohol misuse and crime (Holmes et al., 2001; Osby et al., 2010; Tuvblad et al., 2006; Whitfield et al., 2004) and common environmental factors (e.g. peer affiliations) that are associated with both outcomes (Fergusson et al., 1996; Widom and White, 1997).

While these findings provide some confirmation of previous research into alcohol misuse and crime, there are limitations of the research that need to be recognised. First, the ways in which alcohol misuse has been measured in this research means that the association being studied is the relationship between the individual's propensity to alcohol abuse and their propensity to crime. What this research does not examine is the role of alcohol misuse in specific instances of crimes committed by those cohort members whose drinking was generally non-problematic but who may have committed a crime whilst under the influence of alcohol. The ways in which the AAD symptoms were gathered meant that individuals who were generally non-problematic drinkers but who may have engaged in instances of criminal activity as a result of heavy drinking would have been coded as having low levels of AAD symptomatology. For these reasons the present study is likely to under-estimate the effects of alcohol misuse on crime.

Second, the fact that symptoms of alcohol misuse and criminal offending were measured at the same time periods raises the possibility of a feedback loop in which alcohol misuse leads to offending, and offending increases alcohol misuse (Menard et al., 2001). Further research is necessary to examine possible bi-directionality in the linkages between alcohol and criminal offending.

Nonetheless within these limitations the study findings suggest that the increasing misuse of alcohol was associated with increasing risk of crimes involving impulsive acts including assault, property damage, vandalism and arson. These increased risks were evident up to the age of 30, suggesting that the linkages between alcohol misuse and crime were robust to changes in levels of alcohol consumption and involvement in criminal behaviour over the lifespan.

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Contributors

JMB performed literature searches, analysed the data and wrote the draft article. DMF designed the study, reviewed the analyses and wrote the draft article. LJH collected the data, designed the study and wrote the draft article. All authors have contributed to and approved of the final manuscript.

Conflict of interest

The authors declare no conflicts of interest.

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References

Agnew, R., 2003. An integrated theory of the adolescent peak in offending. Youth Soc. 34, 263–299.
American Psychiatric Association, 1987. Diagnostic and Statistical Manual of Mental Disorders, 3rd ed. - rev. American Psychiatric Association, Washington, DC.
American Psychiatric Association, 1994. Diagnostic and Statistical Manual of Mental Disorders, 4th ed. American Psychiatric Association, Washington DC.
Baumberg, B., 2006. The global economic burden of alcohol: a review and some suggestions. Drug Alcohol Rev. 25, 537–551.
Boden, J.M., Fergusson, D.M., Horwood, L.J., 2012. Alcohol misuse and violent behavior: findings from a 30-year longitudinal study. Drug Alcohol Depend. 122, 135–141.
Botvin, G.J., Griffin, K.W., 2007. School-based programmes to prevent alcohol, tobacco and other drug use. Int. Rev. Psychiatr. 19, 607–615.
Bruzzi, P., Green, S.B., Byar, D.P., Brinton, L.A., Schairer, C., 1985. Estimating the population attributable risk for multiple risk factors using case–control data. Am. J. Epidemiol. 122, 904–914.
Bureau of Justice Statistics, 2009. Crime characteristics. http://www.ojp.gov/bjs/cvict_c.htm#alcohol (accessed 16.11.09).
Bushman, B.J., Cooper, H.M., 1990. Effects of alcohol on human aggression: an integrative research review. Psychol. Bull. 107, 341–354.
Carlin, J.B., Wolfe, R., Coffey, C., Patton, G.C., 1999. Tutorial in biostatistics, analysis of binary outcomes in longitudinal studies using weighted estimating equations and discrete-time survival methods: prevalence and incidence of smoking in an adolescent cohort. Stat. Med. 18, 2655–2679.
Elliott, D.S., Huizinga, D., 1989. Improving self-reported measures of delinquency. In: Klein, M.W. (Ed.), Cross-National Research in Self-Reported Crime and Delinquency. Kluwer, Boston, MA, pp. 155–186.
Ensor, T., Godfrey, C., 1993. Modelling the interactions between alcohol, crime and the criminal justice system. Addiction 88, 477–487.
Farke, W., Anderson, P., 2007. Binge drinking in Europe. Adicciones 19, 333–339.
Fergusson, D.M., Horwood, L.J., 2000. Alcohol abuse and crime: a fixed effects regression analysis. Addiction 95, 1525–1536.
Fergusson, D.M., Horwood, L.J., 2001. The Christchurch Health and Development Study: review of findings on child and adolescent mental health. Aust. N. Z. Psychiatry 35, 287–296.
Fergusson, D.M., Horwood, L.J., Shannon, F.T., Lawton, J.M., 1989. The Christchurch Child Development Study: a review of epidemiological findings. Paediatr. Perinat. Epidemiol. 3, 278–301.
Fergusson, D.M., Lynskey, M.T., Horwood, L.J., 1996. Alcohol misuse and juvenile offending in adolescence. Addiction 91, 483–494.
Flowers, N.T., Naimi, T.S., Brewer, R.D., Elder, R.W., Shults, R.A., Jiles, R., 2008. Patterns of alcohol consumption and alcohol-impaired driving in the United States. Alcohol. Clin. Exp. Res. 32, 639–644.
Giesbrecht, N., 2007. Reducing alcohol-related damage in populations: rethinking the roles of education and persuasion interventions. Addiction 102, 1345–1349.
Gmel, G., Rehm, J., 2003. Harmful alcohol use. Alcohol Res. Health 27, 52–62.
Greenland, S., Morgenstern, H., 2001. Confounding in health research. Annu. Rev. Public Health 22, 189–212.
Hingson, R.W., Heeren, T., Jamanka, A., Howland, J., 2000. Age of drinking onset and unintentional injury involvement after drinking. JAMA 284, 1527–1533.
Hingson, R.W., Zha, W., 2009. Age of drinking onset, alcohol use disorders, frequent heavy drinking, and unintentionally injuring oneself and others after drinking. Pediatrics 123, 1477–1484.
Holmes, S.E., Slaughter, J.R., Kashani, J., 2001. Risk factors in childhood that lead to the development of conduct disorder and antisocial personality disorder. Child Psychiatry Hum. Dev. 31, 183–193.
Huckle, T., Pledger, M., Casswell, S., 2006. Trends in alcohol-related harms and offences in a liberalized alcohol environment. Addiction 101, 232–240.
Kallmen, H., Gustafson, R., 1998. Alcohol and disinhibition. Eur. Addict. Res. 4, 150–162.
Lewis, C.E., Seigel, J.M., Lewis, M.A., 1984. Feeling bad: exploring sources of distress among pre-adolescent children. Am. J. Public Health 74, 117–122.
Liang, K.-Y., Zeger, S.L., 1986. Longitudinal data analysis using generalized linear models. Biometrika 73, 13–22.
Lightowlers, C., 2011. Exploring the temporal association between young people's alcohol consumption patterns and violent behavior. Contemp. Drug Prob. 38, 191–212.
Maggs, J.L., Schulenberg, J.E., 2005. Initiation and course of alcohol consumption among adolescents and young adults. In: Galanter, M., Lowman, C., Boyd, G.M., Faden, V.B., Witt, E., Lagressa, D. (Eds.), Recent Developments in Alcoholism. Springer, New York, pp. 29–47.
Martin, S.E., 2001. The links between alcohol, crime and the criminal justice system: explanations, evidence and interventions. Am. J. Addict. 10, 136–158.

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McCarty, C.A., Ebel, B.E., Garrison, M.M., DiGiuseppe, D.L., Christakis, D.A., Rivara, F.P., 2004. Continuity of binge and harmful drinking from late adolescence to early adulthood. Pediatrics 114, 714–719.
McClelland, G.M., Teplin, L.A., 2001. Alcohol introxication and violent crime: implications for public health policy. Am. J. Addict. 10 (Suppl.), 70–85.
Menard, S., Mihalic, S., Huizinga, D., 2001. Drugs and crime revisited. Justice Q. 18, 269–299.
Miller, T.R., Levy, D.T., Spicer, R.S., Taylor, D.M., 2006. Societal costs of underage drinking. J. Stud. Alcohol 67, 519–528.
Monahan, K.C., Steinberg, L., Cauffman, E., Mulvey, E.P., 2009. Trajectories of antisocial behavior and psychosocial maturity from adolescence to young adulthood. Dev. Psychol. 45, 1654–1668.
Norström, T., Pape, H., 2010. Alcohol, suppressed anger and violence. Addiction 105, 1580–1586.
Osby, U., Liljenberg, J., Kockum, I., Gunnar, A., Terenius, L., 2010. Genes and alcohol. Eur. Psychiatry 25, 281–283.
Palk, G., Davey, J., Freeman, J., 2007. Prevalence and characteristics of alcohol-related incidents requiring police attendance. J. Stud. Alcohol Drugs 68, 575–581.
Parker, R.N., 2004. Alcohol and violence: connections, evidence and possibilities for prevention. J. Psychoactive Drugs Suppl. 2, 157–163.
Parker, R.N., Auerhahn, K., 1998. Alcohol, drugs, and violence. Annu. Rev. Sociol. 24, 291–311.
Rehm, J., Mathers, C., Popova, S., Thavorncharoensap, M., Teerawattananon, Y., Patra, J., 2009. Global burden of disease and injury and economic cost attributable to alcohol use and alcohol-use disorders. Lancet 373, 2223–2233.
Scott, K.D., Schafer, J., Greenfield, T.K., 1999. The role of alcohol in physical assault perpetration and victimization. J. Stud. Alcohol 60, 528–536.
Shepherd, J., 1994. Violent crime: the role of alcohol and new approaches to the prevention of injury. Alcohol Alcohol. 29, 5–10.
Steinberg, L., 2009. Adolescent development and juvenile justice. Annu. Rev. Clin. Psychol. 5, 459–485.
Tuvblad, C., Grann, M., Lichtenstein, P., 2006. Heritability for adolescent antisocial behavior differs with socioeconomic status: gene–environment interaction. J. Child Psychol. Psychiatry 47, 734–743.
Ward, A., 2009. The role of causal criteria in causal inferences: Bradford Hill's "aspects of association". Epidemiol. Perspect. Innovat. 6, 2.
Ward, A., Johnson, P., 2008. Addressing confounding errors when using non-experimental, observational data to make causal claims. Synthese 163, 419–432.
Wei, E.H., Loeber, R., White, H.R., 2004. Teasing apart the developmental associations between alcohol and marijuana use and violence. J. Contemp. Crim. Justice 20, 166–183.
Welte, J.W., Miller, B.A., 1987. Alcohol use by violent and property offenders. Drug Alcohol Depend. 19, 313–324.
Whitfield, J.B., Zhu, G., Madden, P.A., Neale, M.C., Heath, A.C., Martin, N.G., 2004. The genetics of alcohol intake and of alcohol dependence. Alcohol. Clin. Exp. Res. 28, 1153–1160.
Widom, C.S., White, H.R., 1997. Problem behaviours in abused and neglected children grown up: prevalence and co-occurrence of substance abuse, crime and violence. Crim. Behav. Mental Health 7, 287–310.
Wood, J., 2005. Alcohol and crime in New Zealand. In: Working Together Conference. New Zealand Ministry of Justice, Wellington.
World Health Organization, 1993. Composite International Diagnostic Interview (CIDI). World Health Organization, Geneva, Switzerland.
World Health Organization, 2007. WHO expert committee on problems related to alcohol consumption. In: Second Report. World Health Organ., Tech. Rep. Ser., 1–53, 55–57, back cover.
Zeger, S.L., Liang, K.-Y., 1986. Longitudinal data analysis for discrete and continuous outcomes. Biometrics 42, 121–130.

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