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Alcohol misuse and violent behavior: 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 violent offending and intimate partner violence (IPV) to age 30 in a New Zealand birth cohort.
Methods: Outcomes included: measures of violent offending, violence victimization, and physical IPV perpetration and victimization. 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 rates of violence outcomes that ranged from 4.10 to 11.85 times higher than those with no symptoms, but these associations did not differ by gender. Adjustment of the associations for both unobserved fixed effects and time-dynamic covariate factors reduced the magnitude of the associations for violent offending, violence victimization and IPV perpetration, with those with five or more AAD symptoms having rates of violence outcomes that were 1.91–3.58 times higher than those with no symptoms. However, control for both fixed effects and time-dynamic covariate factors reduced the associations between AAD symptoms and physical IPV victimization to statistical non-significance (IRR = 0.73, 95% CI: 0.51–1.06).
Conclusions: The results suggest a causal association between alcohol misuse and violent offending/victimization and IPV perpetration, with estimates suggesting that alcohol use disorder accounted for approximately 4.6–9.3% of the reported violent offending/victimization and IPV perpetration in the cohort.

1. Introduction

Recently there has been growing concern about the effects of alcohol misuse and the extent to which it may be responsible for antisocial behavior including violent crime and intimate partner violence (IPV). Research has shown that increasing misuse of alcohol is associated with increased rates of violent offending (Bureau of Justice Statistics, 2009; Fergusson and Horwood, 2000; Miller et al., 2006; Parkhill et al., 2009; Scott et al., 1999) and violence victimization (McClelland and Teplin, 2001; Mericle and Havassy, 2008), and IPV perpetration and victimization (Follingstad et al., 1999; Foran and O'Leary, 2008; Jewkes, 2002; White and Widom, 2003; Wolitzky-Taylor et al., 2008). One issue arising from this research is the extent to which the links between alcohol misuse and violence may be causal (Gmel and Rehm, 2003; Rehm et al., 2003, 2007). While a number of studies have asserted that there is a causal association between alcohol misuse and increased risk of violence (Gustafson, 1993; Kallmen and Gustafson, 1998; Vengeliene et al., 2008), it could be argued that the associations between alcohol misuse and antisocial behaviors may partly reflect the effects of third or confounding factors (Greenland and Morgenstern, 2001; Ward, 2009). This issue has been examined in a number of studies (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), all of which have found associations between alcohol misuse and violence persist after control for confounding. Nonetheless, the possibility remains that the associations between alcohol misuse and antisocial behavior may be explained by the effects of non-observed confounding factors.

One solution to this issue is to employ methods that account for the effects of non-observed confounding factors, such as fixed effects regression models, which can be employed in longitudinal studies (Cameron and Trivedi, 1998; Greene, 1990). Fixed effects models make it possible to take into account non-observed genetic and environmental factors that have a *fixed* effect on the associations between alcohol misuse and violence outcomes. In the context of research into alcohol and violence, factors that may potentially be subsumed by the fixed effects term are all individual, family, social, and related factors that are fixed at the point of

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adolescence and which have a fixed effect on later alcohol misuse and violence perpetration and victimization. However, the model does not address the issue of confounders that may vary over time and to control for such confounding, the fixed effects model needs to be augmented by observed time-dynamic confounding factors.

A second issue is the extent to which links between alcohol misuse and violence may differ according to gender. A number of recent studies have reported increases in the use and misuse of alcohol by women (Grucza et al., 2008; Keyes et al., 2008; McPherson et al., 2004), but there remain gender differences in patterns of alcohol consumption and symptoms of alcohol use disorder (McPherson et al., 2004), with males having an earlier onset of alcohol use, greater levels of consumption, and a greater risk of AAD than females across age ranges. It is therefore important to compare the effects of alcohol misuse on antisocial behavior perpetration and victimization by males and females to determine the extent to which any effects differ according to gender.

Against this background, the present study reports the results of a longitudinal study of the relationships between alcohol misuse and violence perpetration and victimization in a birth cohort of young people studied to age 30. The aims of this analysis were to: (a) control the associations between measures of alcohol misuse and measures of violence perpetration and victimization for both non-observed fixed and time-dynamic confounding factors; and (b) examine whether gender differences in rates of alcohol misuse may be related to violence perpetration and victimization outcomes.

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.

2.3. Violent offending, violence victimization, and physical intimate partner violence perpetration and victimization, ages 17–30

2.3.1. Violent offending (ages 17–18; 20–21; 24–25; 29–30). At ages 18, 21, 25, and 30, respondents were questioned about their criminal behaviors since the previous assessment using the Self-Report Delinquency Inventory (SRDI: Elliott and Huizinga, 1989) supplemented by additional custom-written survey items. This information was used to derive count measures of the number of self-reported violent offenses committed in each year from age 17 to age 30. Violent offenses included assault, fighting, use of a weapon, or threats of violence against a person. For the purposes of the present analyses, measures of offending were truncated to a maximum of 50 offenses to reduce the influence of extreme outliers.

2.3.2. Violence victimization (ages 17–18; 20–21; 24–25; 29–30). Also at ages 18, 21, 25, and 30 respondents were questioned about a range of stressful life events that may have occurred in each year following the previous assessment using items from the Feeling Bad Scale (Lewis et al., 1984) and custom-written survey items. Two of the items (physical assault victimization; sexual assault victimization) were used to construct a measure of violence victimization during the 12 month period prior to each assessment. Participants who reported having been either physically or sexually assaulted during the year prior to each assessment (ages 17–18; 20–21; 24–25; 29–30) were classified as having been a victim of violence during that period.

2.3.3. Physical intimate partner violence (IPV) perpetration and victimization (ages 20–21; 24–25; 29–30). At ages 21, 25 and 30 sample members in partnerships of over one month duration in the last year were asked about the occurrence of physical IPV using a 22-item scale that used items from the Revised Conflict Tactics Scale (CTS2, Straus et al., 1996). The selected items included the domains of minor physical assault, severe physical assault, and sexual coercion as described by Straus et al. (1996). The scale was presented twice at each assessment; once to assess physical IPV perpetration, and then to assess the victimization by physical IPV. To devise measures of IPV perpetration and victimization each item was scored in dichotomous (absent/present) form and a diversity scale score created from the sum of these items during each assessment period (20–21 years, 24–25 years; 29–30 years). For the purposes of the present investigation, those individuals who did not report an intimate partnership at any assessment period received an IPV perpetration and victimization scale score of 0 for that period.

2.4. Time dynamic covariate factors

The following time-dynamic covariate factors were chosen for the analyses.

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; victimization; 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. These measures excluded the questions regarding physical and sexual assault as they had been used previously to construct the violence victimization outcome described above.

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 twelve 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: (A) 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 behavior 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 behaviors (none, some, most).

2.5. Statistical analyses

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

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

where $f(Y_{it})$ was either the log rate or log odds of each violence outcome reported by the ith subject in a given interval t (for violent offending and violence victimization, t = 17–18 years; 20–21 years; 24–25 years; and 29–30 years; for IPV perpetration and victimization, t = 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

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matrix. From the fitted models, estimates of the incidence rate ratio (IRR) for count measures or odds ratio (OR) for dichotomous measures, and 95% confidence intervals (CI) of AAD symptoms for violence outcomes were calculated. In addition, these models were extended to include a further two terms representing: (a) gender; (b) a gender × AAD symptoms interaction, in order to account for the possibility that rates of AAD symptoms varied by gender; and (c) 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) unobserved 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:

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

In this model α_i are the 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.

Finally, estimates of the population attributable risk (PAR) for each violence outcome were calculated using methods described by Coughlin et al. (1994), after adjustment for both unobserved fixed effects and time-dynamic covariate factors.

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 violence 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 ($p < .01$) 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 violence perpetration and symptoms of alcohol abuse/dependence (AAD), ages 17–30

Table 1a shows the association between per annum rates of self-reported violent offending and symptoms of AAD at ages 17–18; 20–21; 24–25; and 29–30. The table also shows population-averaged incidence rate ratios (IRR) and 95% confidence intervals (CI) for each level of the AAD symptoms measures, for males, females and the total sample. This comparison shows that those who reported 5 or more symptoms of AAD had rates of violent offending that were 11.85 (95% CI: 9.94–14.17) times greater than those who reported no symptoms ($p < .0001$) The analysis was further extended to examine age × AAD symptoms and gender × AAD symptoms interactions (see Section 2). These analyses showed that there was no significant gender × AAD interaction ($LR\chi^2(1) = 0.04$, $p > .80$) reflecting the fact that the associations between AAD symptoms and violent offending were similar for males and females. However, there was a significant age × AAD interaction ($LR\chi^2(1) = 14.97$, $p < .0001$) reflecting the fact that the association between AAD symptoms and violent offending at age 29–30 was weaker than at other ages.

Table 1b presents an analysis of the relationships between mean physical IPV perpetration diversity scores and rates of AAD symptoms assessed at ages 20–21; 24–25 and 29–30. The table shows that at all three ages there was clear evidence that increasing ADD symptoms were associated with increasing IPV diversity scores. This conclusion is supported by the population-averaged IRR which shows that those reporting five or more AAD symptoms had mean diversity scores which were 4.41 (95% CI: 3.38–5.83) times greater than those who reported no AAD symptoms ($p < .0001$). However, there was no evidence of significant gender × AAD interaction ($LR\chi^2(1) = 0.12$, $p > .70$) or age × AAD interaction ($LR\chi^2(1) = 0.72$, $p > .30$).

3.2. Associations between violence victimization and symptoms of alcohol abuse/dependence (AAD), ages 17–30

Table 2a shows the association between rates of self-reported violence victimization and symptoms of ADD at ages 17–18; 20–21; 24–25; and 29–30. The table shows the presence of clear associations between symptoms of AAD and rates of violence victimization at each assessment. The table also shows population-averaged odds ratios (OR) and 95% confidence intervals (CI) for each level of the AAD symptoms measures. This comparison shows that those who reported 5 or more symptoms of AAD had rates of violence victimization that were 6.75 (95% CI: 4.74–9.66) times greater than those who reported no symptoms ($p < .0001$) However, there was no evidence of significant gender × AAD interactions ($LR\chi^2(1) = 0.44$, $p > .50$) or age × AAD interaction ($LR\chi^2(1) = 1.02$, $p > .30$).

Table 2b presents an analysis of the relationships between mean physical IPV victimization diversity scores and rates of AAD symptoms assessed at ages 20–21; 24–25 and 29–30. The table shows that at all three ages there was clear evidence that increasing ADD symptoms were associated with increasing IPV victimization scores. This conclusion is supported by the population-averaged IRR which shows that those reporting five or more AAD symptoms had mean diversity scores that were 4.10 (95% CI: 3.31–5.09) times greater than those who reported no AAD symptoms ($p < .0001$). However, there was no evidence of significant gender × AAD interactions ($LR\chi^2(1) = 0.04$, $p > .80$) or age × AAD interaction ($LR\chi^2(1) = 1.40$, $p > .20$).

3.3. Associations between violence perpetration/victimization and AAD symptoms after adjustment for fixed effects and time-dynamic covariate factors

A potential limitation to the results presented in Tables 1a, 1b, 2a and 2b is the possibility that the associations between violence perpetration/victimization and AAD symptoms could be due to the effects of non-observed confounding that was related to both violence and AAD symptoms. In order to examine these issues, the associations between violence perpetration/victimization and AAD were adjusted for both non-observed fixed sources of confounding and time-dynamic covariate factors that may have influenced the association between violence and AAD symptoms. These 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. The results of this modelling are displayed in Table 3, which shows the adjusted IRRs and ORs and 95% CIs for the associations between each violence outcome and AAD symptoms, after controlling for both fixed effects and time-dynamic covariate factors. The table shows:

1. For the associations between violent offending, violence victimization, IPV perpetration, and AAD symptoms, controlling for non-observed fixed effects and time-dynamic covariate factors reduced the magnitude of the association between violence outcomes and AAD. However, in all cases the associations remained statistically significant ($p < .05$). When compared with those with no symptoms of AAD, those individuals having five or more symptoms had: (a) rates of violent offending that were 3.58 times (95% CI: 1.60–8.12) greater; (b) rates of physical IPV perpetration

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Table 1a
Associations between violent offending and concurrently measured alcohol abuse/dependence (AAD) symptoms, ages 17–30.

Age	Number of symptoms of AAD				*p*
	0	1–2	3–4	5+	
Violent offending (mean, SD)					
Ages 17–18	0.99	2.32	4.93	14.00	
	(5.73)	(8.84)	(10.73)	(18.90)	
n	782	138	55	50	
Ages 20–21	0.23	1.86	3.27	3.92	
	(2.27)	(8.24)	(11.22)	(11.40)	
n	749	161	63	38	
Ages 24–25	0.10	1.52	0.43	0.54	
	(0.86)	(7.45)	(1.19)	(1.57)	
n	812	130	39	22	
Ages 29–30	0.37	0.77	0.11	0.63	
	(3.71)	(5.23)	(0.32)	(0.74)	
n	855	97	27	8	
Population-averaged IRR (95% CI)	1	2.28	5.20	11.85	<.0001
	–	(2.15–2.42)	(4.62–5.86)	(9.94–14.17)	
Population-averaged IRR (95% CI) – males	1	2.00	4.00	8.00	<.0001
	–	(1.86–2.16)	(3.46–4.67)	(6.43–10.08)	
Population-averaged IRR (95% CI) – females	1	2.49	6.20	15.44	<.0001
	–	(2.25–2.75)	(5.06–7.56)	(11.39–20.80)	

Test of gender × AAD symptoms interaction: LR$\chi^2(1) = 0.04$, $p > .80$.
Test of age × AAD symptoms interaction: LR$\chi^2(1) = 14.97$, $p < .0001$.

Table 1b
Associations between physical intimate partner violence (IPV) perpetration and concurrently measured alcohol abuse/dependence (AAD) symptoms, ages 20–30.

Age	Number of symptoms of AAD				*p*
	0	1–2	3–4	5+	
IPV perpetration (mean, SD)					
Ages 20–21	0.19	0.29	0.37	0.35	
	(0.77)	(1.19)	(1.13)	(1.32)	
n	749	161	63	38	
Ages 24–25	0.09	0.36	0.44	0.41	
	(0.53)	(1.36)	(1.29)	(1.33)	
n	812	130	39	22	
Ages 29–30	0.12	0.16	0.22	1.13	
	(0.61)	(0.49)	(0.51)	(2.10)	
n	855	97	27	8	
Population-averaged IRR (95% CI)	1	1.64	2.69	4.41	<.0001
	–	(1.50–1.80)	(2.25–3.24)	(3.38–5.83)	
Population-averaged IRR (95% CI) – males	1	1.63	2.66	4.33	<.001
	–	(1.43–1.86)	(2.04–3.46)	(2.92–6.43)	
Population-averaged IRR (95% CI) – females	1	1.73	2.99	5.18	<.0001
	–	(1.50–1.99)	(2.25–3.96)	(3.38–7.88)	

Test of gender interaction: LR$\chi^2(1) = 0.12$, $p > .70$.
Test of age × AAD symptoms interaction: LR$\chi^2(1) = 0.72$, $p > .30$.

Table 2a
Associations between violence victimization and concurrently measured alcohol abuse/dependence (AAD) symptoms, ages 17–30.

Age	Number of symptoms of AAD				*p*
	0	1–2	3–4	5+	
Violence victimization (%)					
Ages 17–18	7.5	19.6	25.5	46.0	
n	782	138	55	50	
Ages 20–21	10.4	16.8	20.6	26.3	
n	749	161	63	38	
Ages 24–25	5.0	13.8	17.9	45.5	
n	812	130	39	22	
Ages 29–30	2.6	7.2	7.4	37.5	
n	855	97	27	8	
Population-averaged OR (95% CI)	1	1.89	3.57	6.75	<.0001
	–	(1.68–2.13)	(2.82–4.54)	(4.74–9.66)	
Population-Averaged OR (95% CI) – males	1	1.98	3.92	7.76	<.0001
	–	(1.71–2.31)	(2.92–5.34)	(5.00–12.33)	
Population-averaged OR (95% CI) – females	1	1.77	3.13	5.55	<.0001
	–	(1.46–2.14)	(2.13–4.58)	(3.11–9.80)	

Test of gender × AAD symptoms interaction: LR$\chi^2(1) = 0.44$, $p > .50$.
Test of age × AAD symptoms interaction: LR$\chi^2(1) = 1.02$, $p > .30$.

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Table 2b
Associations between physical intimate partner violence (IPV) victimization and concurrently measured alcohol abuse/dependence (AAD) symptoms, ages 20–30.

Age	Number of symptoms of AAD				*p*
	0	1–2	3–4	5+	
IPV Victimization (mean, SD)					
Ages 20–21	0.22	0.38	0.37	0.54	
	(0.90)	(1.30)	(1.18)	(1.82)	
n	749	161	63	38	
Ages 24–25	0.18	0.63	0.59	1.14	
	(0.87)	(2.14)	(1.39)	(2.83)	
n	812	130	39	22	
Ages 29–30	0.37	0.71	0.81	1.25	
	(2.27)	(2.29)	(2.72)	(2.82)	
n	855	97	27	8	
Population-averaged IRR (95% CI)	1	1.60	2.56	4.10	<.0001
	–	(1.49–1.72)	(2.22–2.96)	(3.31–5.09)	
Population-averaged IRR (95% CI) – males	1	1.51	2.28	3.44	<.01
	–	(1.39–1.65)	(1.93–2.72)	(2.69–4.49)	
Population-averaged IRR (95% CI) – females	1	1.58	2.50	3.38	<.001
	–	(1.37–1.82)	(1.88–2.89)	(2.57–6.03)	

Test of gender × AAD symptoms interaction: LR$\chi^2(1) = 0.04$, $p > .80$.
Test of age × AAD symptoms interaction: LR$\chi^2(1) = 1.40$, $p > .20$.

that were 2.15 times (95% CI: 1.26–3.65) greater; and (c) odds of violence victimization that were 1.91 times (95% CI: 1.00–3.58) greater.

2. For the associations between IPV victimization and AAD symptoms, controlling for confounding reduced the associations between IPV victimization and AAD symptoms to statistical non-significance ($p > .10$). The results of these analyses suggest that the observed associations between IPV victimization and AAD symptoms could be largely explained by a range of sources of confounding that influenced both IPV victimization and AAD symptoms.

3.4. Population attributable risk (PAR)

As noted in Section 2, estimates of the PAR for the role of AAD in violence perpetration, IPV perpetration and violence victimization were calculated using methods described by Coughlin et al. (1994), after adjustment for fixed effects and time-dynamic confounding factors. These analyses showed that 9.3% of violence perpetration 6.3% of violence victimization, and 4.6% of IPV perpetration could be accounted for by the presence of AAD.

4. Discussion

In this paper we have used data gathered over the course of a 30-year longitudinal study to examine the linkages between alcohol misuse and a range of violence outcomes spanning perpetration and victimization during the period ages 17–30. The findings of this analysis and their implications are outlined below.

First, in agreement with a range of research examining the links between alcohol misuse and violence outcomes (Bureau of Justice Statistics, 2009; Fergusson and Horwood, 2000; Follingstad et al., 1999; Foran and O'Leary, 2008; Jewkes, 2002; McClelland and Teplin, 2001; Mericle and Havassy, 2008; Miller et al., 2006; Parkhill et al., 2009; Scott et al., 1999; White and Widom, 2003; Wolitzky-Taylor et al., 2008), there were consistent bivariate associations between measures of AAD symptoms and each of the four measures of violence outcome (violent offending; violence victimization; IPV perpetration; IPV victimization). Individuals with five or more AAD symptoms had risks of involvement in violence that ranged from 4.10 to 11.85 times higher than those with no AAD symptoms. In addition, there was little evidence to suggest that these effects differed by gender, with the analyses revealing no evidence of statistically significant interactions between gender and AAD symptoms in predicting violent outcomes, indicating that the strength of association between AAD and outcomes was similar for females and males. However, for the measure of violent offending, there was a statistically significant ($p < .0001$) age × AAD symptoms interaction, which was likely due to lower rates of both offending and AAD symptoms at age 30.

Further analyses showed that, after controlling for a range of potential sources of confounding, including non-observed fixed confounding factors and time-dynamic covariate factors, the associations between AAD symptoms and violent offending, violence victimization, and IPV perpetration remained statistically significant, with those reporting five or more AAD symptoms having risks of violent offending, violence victimization and IPV perpetration that ranged from 1.91 to 3.58 times greater than individuals with no

Table 3
IRRs and ORs (and 95% CI) for the associations between violence perpetration/victimization and concurrently measured symptoms of alcohol abuse/dependence (AAD), 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+	
Violent offending (ages 17–30) IRR	1	1.53	2.34	3.58	<.01
	–	(1.17–2.01)	(1.37–4.04)	(1.60–8.12)	
Physical IPV perpetration (ages 20–30) IRR	1	1.29	1.66	2.15	<.01
	–	(1.08–1.54)	(1.17–2.37)	(1.26–3.65)	
Violence victimization (ages 17–30) OR	1	1.24	1.54	1.91	<.05
	–	(1.00–1.53)	(1.00–2.34)	(1.00–3.58)	
Physical IPV victimization (ages 20–30) IRR	1	0.90	0.81	0.73	>.30
	–	(0.80–1.02)	(0.64–1.04)	(0.51–1.06)	

Note: Statistically significant ($p < .05$) 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.

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symptoms. On the other hand, controlling for a range of sources of potential confounding factors explained the associations between AAD symptoms and IPV victimization.

The results of the present study suggest that the associations between AAD and violent behaviors may be partially spurious and arise from observed and non-observed sources of confounding (Greenland and Morgenstern, 2001; Ward, 2009; Ward and Johnson, 2008). Specifically, control for confounding using fixed effects regression models and time-dynamic confounding factors reduced the associations between symptoms of AAD and violence outcomes quite substantially. However, even after such control significant associations remained between violence offending, IPV perpetration, violence victimization and symptoms of AAD. These findings are consistent with the conclusion that alcohol misuse plays a causal role in increasing risks of these outcomes (Gustafson, 1993; Kallmen and Gustafson, 1998; Vengeliene et al., 2008). Estimates of the population attributable risk (PAR) suggest that alcohol misuse explains in the region of 5–10% of the risk of violence offending, IPV perpetration and violence victimization in the cohort. However, AAD was not related to IPV victimization after control for confounding factors, suggesting that alcohol misuse plays no role in increasing the risk of IPV victimization. One possible explanation for the differences in these patterns of findings is that the measures of violence perpetration largely reflect impulsive outbursts of violent behavior while the individual is under the influence of alcohol intoxication (Gustafson, 1993; Kallmen and Gustafson, 1998; Vengeliene et al., 2008), and the measure of violence victimization may also reflect exposure to assault under circumstances in which both victims and perpetrators were intoxicated (Scott et al., 1999; Young et al., 2008). On the other hand, physical IPV victimization, which consists largely of minor physical violence in the context of an intimate partnership, and for which there may be a wider range of opportunities and circumstances for violence, is less likely to be the result of intoxication on the part of the victim. In general, the results of the study suggests that, for violent offending, IPV perpetration, and violence victimization, the adoption of laws and policies aimed at reducing alcohol consumption, and the use of treatments to address alcohol abuse and dependence issues amongst high-risk individuals, will reduce the overall incidence of each of these outcomes in the population as a whole. It is unclear, however, the extent to which such laws, policies, and interventions would reduce the risk of violence perpetration and victimization at the individual level. Further research is needed to better estimate the impact of reductions in alcohol consumption and alcohol-related problems on individuals' exposure to violence perpetration and victimization.

An important finding of this analysis was that the linkages between alcohol misuse and violence outcomes were evident for both males and females. The present results suggest that the associations between alcohol misuse and violence outcomes did not differ according to gender, and the strength of associations between alcohol misuse and violence were comparable for males and females. These findings suggest that increasing levels of alcohol consumption and alcohol misuse amongst females may explain to some extent recent observations of increases in crime and related antisocial behaviors amongst women (Moretti and Odgers, 2002; Odgers and Moretti, 2002; Rossegger et al., 2009). Furthermore, these findings suggest that public health interventions aimed at reducing alcohol misuse may be effective in reducing the risk of violence perpetration and victimization for both males and females.

These conclusions need to be considered in the light of possible limitations of the study. These limitations include the fact that the study was based on a specific cohort studied in a specific social context; that the measures of AAD symptoms and violence outcomes were obtained via self-report; and that sample loss over time was not entirely random. As such these variables may be subject to errors of measurement that may compromise the estimation of model parameters. Notwithstanding these limitations, the weight of the evidence from this and other research studies suggest that the misuse of alcohol is related to increased risks of both violent crime and domestic violence. These trends appear to be similar for both males and females.

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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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