A LONGITUDINAL STUDY OF ALCOHOL USE AND ANTISOCIAL BEHAVIOUR IN YOUNG PEOPLE

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Abstract — Aims: To examine the direction of causation between young people's antisocial behaviour and alcohol (mis)use in the longer and shorter term, together with their joint effects on alcohol-related trouble. Methods: A longitudinal study (2586 pupils) supplied data, allowing exploration of the causal effects of alcohol (mis)use and antisocial behaviour between ages 11 and 15, using structural equation models of longer and shorter-term relationships and joint-effects models in respect of alcohol-related trouble at age 15. This method allowed us to evaluate which of three hypotheses, described as 'disinhibition' [alcohol (mis)use causes or facilitates antisocial behaviour], 'susceptibility' [antisocial behaviour causes alcohol (mis)use] or 'reciprocal' [alcohol (mis)use causes antisocial behaviour and the reverse] receives most support, both overall and by gender, social class, and drinking context. Results: Overall, the results support the susceptibility hypothesis, particularly in the longer-term models. There is no support for 'pure' disinhibition. However, in the shorter-term and joint-effects models (i.e. as the time lag becomes shorter), there is evidence that in some gender, social class, or drinking contexts, in addition to antisocial behaviour causing alcohol (mis)use, the reverse also applies. Conclusions: Antisocial behaviour is the main predictor of alcohol (mis)use and alcohol-related trouble, with alcohol (mis)use impacting only modestly on antisocial behaviour and alcohol-related trouble in the shorter term.

INTRODUCTION

This paper examines the relationship between alcohol (mis)use, antisocial behaviour and alcohol-related trouble (that is, the co-occurrence of alcohol and disruptive behaviour) among young people in the west of Scotland in the mid-late 1990s. Evidence of a dramatic rise in alcohol consumption in this age group (particularly females), in both this geographical area (Sweeting and West, 2003) and the UK in general (Rodham et al., 2005; Plant and Plant, 2006), together with its poor position in comparison to the rest of Europe, (Hibell et al., 2004) has made the reduction in alcohol (mis)use, especially binge drinking and public drunkenness, a priority of the British Government (Cabinet Office, Prime Minister's Strategy Unit, 2004). Similarly, evidence of an increase in conduct disorder in youth (Collishaw et al., 2004) underpins a range of policies to reduce antisocial behaviour. Given the wellestablished association between alcohol (mis)use and antisocial behaviour (Plant et al., 1985; Sanford, 2001; Miczek et al., 2004; Rose et al., 2004), a significant thrust of these involves attempts to reduce young people's alcohol consumption (Marsh and Fox, 1992).

Though well-documented, the relationship between alcohol (mis)use and antisocial behaviour is not simple, different perspectives producing different predictions about the direction of causality. These can be formulated as three basic hypotheses, representing disinhibition, susceptibility, and reciprocal effects respectively, each of which can be applied in both the shorter and longer term.

The first (disinhibition hypothesis) implies that alcohol causes or facilitates antisocial behaviour in the shorter (more immediate) term, as a consequence of its acute effects on the brain, in particular its impact on pre-frontal functioning (Room and Collins, 1988; Bushman and Cooper, 1990;

Contrasting with perspectives that posit an effect of alcohol on antisocial behaviour, the second (susceptibility) hypothesis implies the reverse; people with susceptibility to, or on a trajectory for antisocial behaviour, use alcohol to a greater extent than those who are less susceptible. Thus, antisocial behaviour is the cause of alcohol (mis)use, in either the shorter or longer term. Substance use, including alcohol in adolescence, has been related to characteristics such as under control and aggression at (much) younger ages (Block et al., 1988; Windle, 1990; White et al., 1993; Brook et al., 1996), resulting from a general inability to regulate behaviour. This could arise from a variety of causes including common genetic factors (Clark et al., 2002; Cooper et al., 2003; Stallings et al., 2005) or social factors. Each of these explanations is consistent with the notion of higher order factors representing core psychopathological processes, in this case expressed in externalizing problems (Krueger et al., 1998). Alternatively (or additionally), antisocial behaviour may cause alcohol (mis)use through association with antisocial, alcohol-using peers (Barnow et al., 2002).

Finally, the third (reciprocal hypothesis) implies both that alcohol (mis)use causes antisocial behaviour and that antisocial behaviour causes alcohol (mis)use, thus establishing a feedback loop. In the shorter term, alcohol and antisocial

Graham *et al.*, 1998). For example, a study of young adults concluded that violence among alcohol-dependent individuals occurred when, and possibly because, they were under the influence of alcohol (Arseneault *et al.*, 2000). In the longer term, there is a potential additional impact of chronic alcohol (mis)use on antisocial behaviour through hippocampal damage (impairing memory, learning, and consequently, overall executive functioning) or other neurological impairment (Tapert *et al.*, 2005; Howard, 2006). In addition, excessive alcohol use may be associated with impaired relationships with parents and peers, difficulties in education or work, conflict with law enforcement agencies and a deviant or antisocial friendship network (Brook *et al.*, 1998).

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behaviour may fuel each other, escalating aggression in particular social contexts (Graham *et al.*, 1998, 2000). In the longer term, the consequences of alcohol (mis)use may interact with individual factors (impulsivity, sensation-seeking, aggressive personality), so increasing dysregulation and problems with judgement, leading to further, and worsening alcohol use and antisocial behaviour (Howard, 2006; Measelle *et al.*, 2006).

Each of the above hypotheses relates to the relationship between alcohol (mis)use and antisocial behaviour in general. More specific alcohol-related trouble such as fights, arguments, or involvement with the police due to drinking may be a joint outcome of (tendencies towards) antisocial behaviour in addition to alcohol (mis)use. As such, the same three hypotheses are relevant to its prediction. Thus, the disinhibition hypothesis suggests that alcohol (mis)use is a better predictor of alcohol-related trouble, the susceptibility hypothesis that it is better predicted by antisocial behaviour, and the reciprocal hypothesis that both alcohol and antisocial behaviour predict alcohol-related trouble. In support of the latter, a recent study found that significant predictors of adolescent alcohol-related fighting included frequent and high volume drinking (suggesting disinhibition) and troubles in school such as relationship problems with teachers or peers, or attention difficulties (suggesting susceptibility) (Swahn and Donovan, 2005).

The aim of this paper is to determine which of these competing hypotheses has greater support over both longer and shorter time frames. In addition, it is important to acknowledge social and cultural factors which suggest that the effects of alcohol (mis)use ('drunken comportment' or 'drunken changes-for-the-worse') vary between societies, contexts and, to some degree, over time (MacAndrew and Edgerton, 1969; Room and Collins, 1988; Room, 2001; Abel and Plumridge, 2004). Thus, while the predominant cultural expectancy in the United Kingdom is that alcohol leads to aggression, this is not the case in ('wet') countries such as Spain (Marsh and Fox, 1992). Furthermore, within nations, specific subcultures shape expectancies, a notable example within Englishspeaking countries being macho subcultures celebrating the link between alcohol and violence, in contrast to more commonly held beliefs that drunkenness is not an excuse for bad behaviour (Graham et al., 1998, 2000; Room, 2001; Rolfe et al., 2006). Situational factors are also important; for example, crowded, noisy, smoky, and provocative environments facilitate aggression (Bushman and Cooper, 1990; Graham et al., 1998; Wells et al., 2005).

Evidence that the disinhibiting effects of alcohol are subject to such variation, suggests the relative importance of each hypothesis is likely to vary between population subgroups. Chief among these are gender and social class, which together with drinking context shape both alcohol use and antisocial behaviour, and the interaction between the two. With respect to gender, differences may be biological, females being physiologically less tolerant of alcohol (Schuckit *et al.*, 1998) or socio-cultural (Room and Collins, 1988). Higher correlations between earlier antisocial behaviour and later substance use, suggesting stronger susceptibility effects, have been found in males (Windle, 1990), while stronger relationships between alcohol consumption and aggression have been found in females (Wells *et al.*, 2005), These authors attribute

these stronger disinhibition effects among females to gender differences in ease of intoxication, deviancy associated with heavy drinking or the nature of aggression (Wells *et al.*, 2005). However, one Glasgow-based study found students (of either sex) strongly expected aggression to be a primary consequence of alcohol use, particularly in males (Crawford, 1984). It is possible that the recent increase in drinking among young women may have both increased tolerance to alcohol and changed expectancies.

With respect to social class, evidence of higher levels of alcohol-related violence among working-class youth compared with a pattern of 'social drinking' among their middle-class peers (Room and Collins, 1988; Makkai, 2001) suggests that as a result of social or cultural differences, the former may be more susceptible to both antisocial behaviour and alcohol (mis)use.

It is also possible that class-based variations in the relationship between alcohol and aggression differ by gender, one study finding stronger socio-economic effects for males than females (Wells *et al.*, 2006). However, a British study of adult women who drink found that some working-class women accepted, and to some degree celebrated the role of alcohol-related violence, more middle-class women distanced themselves from direct physical confrontation preferring more indirect methods such as verbal aggression (Day *et al.*, 2003). As with gender, class differences may have diminished over time, however, one recent UK study of heavy adult drinkers suggests this is not the case, with young men and those working in manual occupations particularly prone to alcohol-related violence (Rolfe *et al.*, 2006).

The significance of the three hypotheses may also vary according to the typical contexts in which young people drink. Although parental drinking increases the likelihood that their children will also drink (Wilks *et al.*, 1989), there is some evidence that alcohol problems later in life are reduced among adolescents who drink at home (McKechnie *et al.*, 1977; Ghodsian and Power, 1986). In contrast, alcohol-related problems are more likely among young people who drink in settings away from home, for example in groups or public spaces, such drinking often linked to both fighting and unintentional injuries, particularly when rival peers or peer groups are involved (Brain and Parker, 1997; Pavis *et al.*, 1997; Coleman and Cater, 2005; Wells *et al.*, 2005).

In order to test the relative importance of the three hypotheses in both the shorter and longer term, and their applicability to different sociodemographic sub-groups and drinking contexts, longitudinal data are required. To date, only a few studies meet that requirement and fewer still have used path analysis to investigate the causal relationship between alcohol (mis)use and antisocial behaviour. One such study, conducted by White et al. (1993) on 218 US males from ages 12 to 18, examined the causal relationship between aggression (similar to antisocial behaviour), alcohol use and alcohol-related aggression (similar to alcohol-related trouble). Significant paths were found between previous aggression and later alcohol use and alcohol-related aggression, the evidence supporting the susceptibility hypothesis. Though exemplary for its time, the study was based on a relatively small, allmale sample (prevalence rates for aggressive behaviour and alcohol-related aggression being too low among females to

permit meaningful analysis), and notably did not include sociodemographic or contextual variables.

The longitudinal study reported here examines the causal pathways between alcohol (mis)use, antisocial behaviour and alcohol-related trouble in a larger sample of young people of both genders between the ages of 11 and 15 in the West of Scotland. We test both longer-term (between ages 11–13 and 13–15) and shorter-term (at ages 13 and 15) relations between alcohol (mis)use and antisocial behaviour and, in addition, the joint effects (at age 15) of alcohol (mis)use and antisocial behaviour on alcohol-related trouble. Using latent path analysis, we evaluate which of the three hypotheses (disinhibition, susceptibility, or reciprocal) receives most overall support, by each gender, and in different social classes and drinking contexts. The specific questions addressed are:

- (i) What are the causal sequences of alcohol (mis)use, antisocial behaviour and alcohol-related trouble?
- (ii) Are there differences in the causal sequences of alcohol (mis)use, antisocial behaviour and alcohol-related trouble in the longer when compared with the shorter term?
- (iii) Are there differences in the causal sequences according to gender, social class (non-manual compared with manual) and drinking context (alcohol provided by parents compared with other contexts)?

METHODS

Participants

The school-based 'West of Scotland 11–16 Study' (West and Sweeting, 1996) recruited a cohort of children during their final year of primary schooling (age 11, in 1994–95), following-up at ages 13 (1996) and 15 (1999). At each age, respondents completed health and lifestyle questionnaires, and at age 15 (approximately 1 week later), a psychiatric interview, the Voice-DISC (West *et al.*, 2003). This interview is an interactive computerized (voice) version of the Diagnostic Interview Schedule for Children (Costello *et al.*, 1984), a widely used and well-validated instrument for the identification of DSM-IV (American Psychiatric Association, 1994) psychiatric disorders in children and young people. The prevalence of diagnoses in this sample has been reported (West *et al.*, 2003).

A total of 2586 (1335 males and 1251 females) respondents (93% of the issued sample) completed questionnaires at age 11, 2371 at age 13, and 2196 (79%) at 15, of whom 1860 also completed Voice-DISC. Missing values in subgroup analyses further reduced the numbers. As with all longitudinal studies, there was differential attrition, details of which are available (Sweeting *et al.*, 2001). To address attrition bias, we use full information and maximum likelihood methods to include cases with missing data (Arbuckle and Wothke, 1999).

MEASURES

Latent variables [alcohol (mis)use, antisocial behaviour, and alcohol-related trouble] used in the analyses are constructed

from the following indicator variables (all measures were ordered scales or re-coded as such with scale options shown in Table 1).

Alcohol (mis)use (ages 11, 13, and 15) comprised three indicators from the self-complete questionnaires: drinking frequency, based on roughly equivalent distributions of ageappropriate responses; drunkenness; and prior drinking. Antisocial Behaviour (ages 11, 13, and 15) also comprised three indicators from the questionnaires: truancy ('If I get the chance to skip school I do'), predicted trouble with the police by age 21; and an antisocial identity scale, comprising three summed items ('I get into fights/take risks/am a rule-breaker'), $\propto = 0.65$, 0.72, and 0.69 at ages 11, 13, and 15, respectively. To validate our antisocial behaviour dimension, each of the three indicators at age 15 was correlated with the Voice-DISC diagnosis of conduct disorder, the results showing that conduct disorder was predicted equally well or better by each indicator (e.g. 'police trouble by age 21', r = 0.338), as by individual items within the Voice-DISC module considered to be exemplary indicators of conduct disorder and antisocial personality (e.g. 'physically cruel to animals', r = 0.334). Alcohol-related trouble (age 15) comprised five summed items from the Voice-DISC alcohol abuse section relating to: getting into trouble with the police due to drinking; drinking in situations where you could get hurt; getting into a physical fight while drinking; arguing with family or friends because of drinking; and missing school or work in order to drink, or due to hang-

Social class was based on the occupation of the head of the household at age 11 and derived predominantly from parental information, supplemented by that of the child (West *et al.*, 2001). Occupations were coded by reference to the standard UK classification (Office of Population Census and Surveys, 1990) and categorized non-manual versus manual.

Drinking context was represented by a questionnaire item at age 15, in which drinkers indicated where they had obtained their most recent alcoholic drink. This variable was dichotomized: provided by parents (which we take to indicate parental approval—22.7%) versus other source (shop, bar or pub, club, siblings, friends, stolen from home, other).

Statistical analysis

Structural equation models using latent variable paths are appropriate techniques for charting longitudinal pathways (in our case between alcohol (mis)use and antisocial behaviour). We modelled longer-term (2-year cross-lagged paths—ages 11–13 and 13–15) and shorter-term (simultaneous—ages 13 and 15) relationships between alcohol (mis)use and antisocial behaviour and, in addition, the joint effects (crosssectional—age 15) between alcohol (mis)use, antisocial behaviour and alcohol-related trouble. (Note that although described as 'cross-sectional', the Voice-DISC measure of alcohol-related trouble was generally obtained about a week later than the age 15 measures of alcohol use and antisocial behaviour.) Using multiple indicators of alcohol (mis)use and antisocial behaviour allowed measurement error to be incorporated. Adequate indicator-latent variable loading (typically>0.70) indicated good reliability. Correlations were

Table 1. Frequencies of alcohol (mis)use and antisocial behaviour indicators at ages 11, 13, and 15, and alcohol-related trouble items at age 15

	Age 11		Age 13		Age 15	
Alcohol (Mis)Use	N	(%)	N	(%)	N	(%)
Alcohol frequency						
Never had a drink (age $11 = \text{never tasted}$)	602	33.0	499	27.4	197	10.8
1-2 per year (age $11 = $ tasted only)	863	47.4	768	42.2	477	26.2
Fortnightly (age $11 = 1-2$ per year)	293	16.1	199	10.9	374	20.5
Monthly	42	2.3	196	10.8	333	18.3
Weekly or more	22	1.2	160	8.8	441	24.2
Ever been really drunk						
Never had a drink	1465	80.4	499	27.4	197	10.8
No	311	17.1	834	45.8	542	29.7
Yes (at least once)	46	2.5	489	26.8	1083	59.4
Length of time drinking						
Never	1465	80.4	499	27.4	197	10.8
Within a year of survey	191	10.5	595	32.7	429	23.5
1–2 years prior to survey	139	7.6	411	22.6	427	23.4
More than 2 years prior to survey	27	1.5	317	17.4	769	42.2
Antisocial behaviour						
Miss school						
Strongly disagree			734	40.3	622	34.1
Disagree	1459	80.1	792	43.5	859	47.1
Agree	363	19.9	233	12.8	291	16.0
Strongly agree	_	_	63	3.5	50	2.7
Trouble with the police by age 21						
Very untrue	1195	65.6	793	43.5	856	47.0
Untrue	492	27.0	720	39.5	666	36.6
True	100	5.5	229	12.6	223	12.2
Very true	35	1.9	80	4.4	77	4.2
Take risks						
Very untrue	380	20.9	167	9.2	87	4.8
Untrue	774	42.5	652	35.8	590	32.4
True	575	31.6	845	46.4	985	54.1
Very true	93	5.1	158	8.7	160	8.8
Get into fights	,,,	0.1	100	0.7	100	0.0
Very untrue	560	30.7	528	29.0	648	35.6
Untrue	762	41.8	727	39.9	829	45.5
True	435	23.9	511	28.0	298	16.4
Very true	65	3.6	56	3.1	47	2.6
Rule breaker	05	5.0	20	5.1	.,	2.0
Very untrue	761	41.8	537	29.5	483	26.5
Untrue	937	51.4	936	51.4	913	50.1
True	110	6.0	291	16.0	365	20.0
	1117	O.U	7.71	11111		

allowed both between errors from identical indicator variables over time and contemporaneous disturbances (cross-lagged models only). The most parsimonious model provides the best-fit statistic and accounts for most of the variation between the variables. Thus, when comparing differences in fit between models, the fit statistic determines the relative superiority of each model. The chi-square statistic is our primary method of discrimination, though we also report the comparative fit index (CFI). Due to the complex nature of modelling used in this study we make no adjustment for multiple testing.

In addition to a null hypothesis of no significant critical paths between alcohol (mis)use and antisocial behaviour, three outcomes corresponding to our hypotheses are possible:

- Disinhibition hypothesis: Paths alcohol (mis)use ⇒ antisocial behaviour are significant while antisocial behaviour ⇒ alcohol (mis)use are not; In the joint-effects models, paths alcohol (mis)use ⇒ alcohol-related trouble are significant while antisocial behaviour ⇒ alcohol-related trouble are not.
- Susceptibility hypothesis: Paths antisocial behaviour ⇒ alcohol (mis)use are significant, while paths alcohol

Table 1. (Continued)

	Age 11		Age 13		Age 15	
Alcohol (Mis)Use	N	(%)	N	(%)	N	(%)
Alcohol-related trouble in the last year						
Trouble with police						
Never	_	_	_	_	1703	92.3
Once	_	_	_	_	86	4.7
More than once		_	_	_	57	3.1
Hurt self	_	_	_	_	_	_
Never		_	_	_	1799	97.5
Once	_	_	_	_	26	1.4
More than once	_	_	_	_	21	1.1
Fight(s)		_	_	_		
Never	_	_	_	_	1758	95.2
Once	_	_	_	_	65	3.5
More than once		_	_	_	23	1.2
Argument(s)	_	_	_	_	_	_
Never	_	_	_	_	1649	89.3
Once		_	_	_	64	3.5
More than once	_	_	_	_	133	7.2
Skipped school	_	_	_	_	_	_
Never	_	_	_	_	1781	96.5
Once	_	_	_	_	44	2.4
More than once		_	_	_	21	1.1

(mis)use \Rightarrow antisocial behaviour are not; in the joint-effects models, paths antisocial behaviour \Rightarrow alcohol-related trouble are significant, while paths alcohol (mis)use \Rightarrow alcohol-related trouble are not.

• Reciprocal hypothesis: All critical paths are significant.

RESULTS

Univariate results

Table 1 presents the frequencies of the indicators at each wave. As expected, the frequency of drinking and drunkenness [alcohol (mis)use indicators] increased with age. By contrast, most antisocial behaviour indicators remained relatively stable, although the percentage reporting taking risks and rule-breaking increased between ages 11 and 13. At age 15, only a small percentage of respondents reported involvement in alcohol-related trouble.

Longer-term associations between alcohol use and antisocial behaviour

Figure 1 presents the results of the longer-term (cross-lagged) models in standardized form for males (first path estimates) and females (second), with solid lines representing significant, and broken lines non-significant paths. The models for both genders have excellent fit (CFI>0.90), and echoing the univariate results, show considerable stability for antisocial behaviour. Alcohol (mis)use also shows stability, much more so between ages 13 and 15, though for females the path between ages 11 and 13 is less stable, reflecting the fact

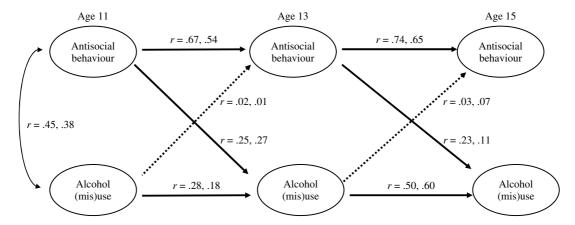
that they start alcohol (mis)use later than males, but catch up by 15. The cross-loading paths demonstrate much greater evidence for the susceptibility than disinhibition hypothesis, all paths from antisocial behaviour to alcohol (mis)use being significant for both genders, and of greater magnitude than the converse alcohol to antisocial behaviour paths.

Shorter-term associations between alcohol use and antisocial behaviour

Figure 2 presents the overall results of the shorter-term (simultaneous) models in standardized form for each gender. Again, solid lines represent significant paths, with the dash-dot line from alcohol (mis)use to antisocial behaviour indicating significant paths for one gender only. The models again have excellent fit. At both 13 and 15, the paths from antisocial behaviour to alcohol (mis)use are stronger than the converse, supporting the susceptibility hypothesis. However there is evidence for a small reciprocal effect for males at age 13 and a more substantial one for females at 15 (the alcohol (mis)use \Rightarrow antisocial behaviour path, r = 0.26, being of almost the same magnitude as the antisocial behaviour \Rightarrow alcohol (mis)use path, r = 0.29), providing evidence of shorter-term effects of alcohol (mis)use to antisocial behaviour as well as vice versa.

Joint effects of alcohol use and antisocial behaviour on alcohol-related trouble

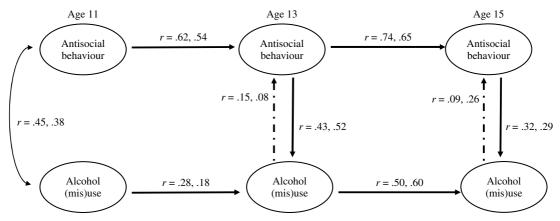
Figure 3 presents the results for the joint-effects (cross-sectional) models of alcohol (mis)use and antisocial behaviour on alcohol-related trouble at age 15 (analyses restricted



Model (male): $\chi^2 = 247.4$, df = 106, P = 0.000, CFI = 0.97

Model (femalz): $\chi^2 = 333.7$, df = 106, P = 0.000, CFI = 0.99

Fig. 1. Longer-term relationships between antisocial behaviour and alcohol (mis)use at ages 11, 13, and 15 (cross-lagged models).



Model (male): $\chi^2 = 248.0$, df = 106, P = 0.000, CFI = 0.99 Model (female): $\chi^2 = 325.1$, df = 106, P = 0.000, CFI = 0.97

Fig. 2. Shorter-term relationships between antisocial behaviour and alcohol (mis)use at ages 11, 13, and 15 (simultaneous models).

to drinkers). Again, models for both males and females have excellent fit, the predictive paths demonstrating greater support for the susceptibility than disinhibition hypothesis. Thus, while the alcohol (mis)use to alcohol-related trouble path is significant and of similar magnitude for both males (0.17) and females (0.13), the parameter estimates (0.36 for each gender) for antisocial behaviour to alcohol-related trouble are highly significant and of greater magnitude. However, estimates are probably attenuated because of the lack of adjustment for measurement error in alcohol-related trouble.

Comparing models in sub-groups

Table 2 shows results corresponding to the disinhibition, susceptibility and reciprocal hypotheses for the longer-term (upper section), shorter-term (middle section) and joint-effects (lower section) models. The overall 'baseline' figures for males and females in the first row of each section correspond

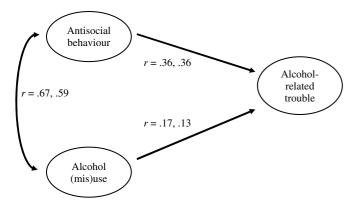
to those in Figs. 1-3, with subsequent rows representing separate subgroup analyses. The 'baseline' columns refer to the overall chi-square of each model including all four crossloading paths. Models with fewer paths can be compared with these 'baseline' figures. The 'disinhibition' columns show the chi-square differences between the 'baseline' model and a model with the disinhibition paths [alcohol (mis)use \Rightarrow antisocial behaviour] removed. A significant difference indicates that the omitted paths are required to improve the model fit, thus providing evidence in support of the disinhibition hypothesis. Similarly, significant results in the 'susceptibility' columns indicate that the antisocial behaviour ⇒ alcohol (mis)use paths are required by the model. If both the 'disinhibition' and 'susceptibility' columns are significant, this provides evidence for the reciprocal hypothesis, since paths representing both alcohol (mis)use ⇒ antisocial behaviour and antisocial behaviour \Rightarrow alcohol (mis)use are required. Finally. if neither column is significant, this supports the null hypothesis of no significant critical paths between alcohol (mis)use and antisocial behaviour. In order to summarize the results,

Table 2. Comparison of longer-term (cross-lagged), shorter-term (simultaneous), and joint-effects (cross-sectional) models in respect of relationships between alcohol (mis)use and antisocial behaviour or alcohol-related trouble for males and females, and each subgroup

	Males			Females				
	N	Baseline χ ²	Disinhibition $\Delta \chi^2$	Susceptibility $\Delta \chi^2$	N	Baseline χ ²	Disinhibition $\Delta \chi^2$	Susceptibility $\Delta \chi^2$
Longer term (cross-lagged) models								
Overall	1335	247.4	0.8	60.3***	1251	333.7	2.0	41.8***
Social class								
Non-manual	542	181.3	0.0	41.8***	486	223.6	0.4	22.0***
Manual	695	153.3	2.3	21.0***	653	236.8	3.1	24.6***
Alcohol from								
Parents	211	132.1	0.6	3.7	226	166.5	0.4	7.8*
Others	730	224.9	2.7	29.6***	755	227.8	0.3	34.0***
Shorter-term (simultaneous) models								
Overall	1335	248.0	7.7*	84.3***	1251	325.1	18.6***	68.0***
Social class								
Non-manual	542	181.6	0.4	55.0***	486	226.7	6.1*	36.0***
Manual	695	153.4	12.1**	28.5***	653	238.6	12.7**	37.0***
Alcohol from								
Parents	211	141.8	2.7	8.9*	226	182.5	0.7	15.9***
Others	730	224.9	8.2*	45.0***	755	227.5	1.4	60.9***
Joint-effects (cross-sectional) models								
Overall	1113	79.1	13.0***	52.2***	1078	59.8	5.3*	37.0***
Social class								
Non-manual	492	42.3	13.1***	15.0***	440	28.3	1.2	19.3***
Manual	565	47.9	2.3	37.0***	558	32.8	5.5*	13.4***
Alcohol from								
Parents	211	6.8	2.3	0.3	226	8.1	0.5	1.4
Others	730	34.6	9.3**	12.1***	755	19.4	11.2**	5.9*

^{*} P = 0.05; ** $P \le 0.01$, *** P = 0.001.

^a Estimation problems encountered but reasonable parameter estimates obtained.



Model (male): $\chi^2 = 79.0$, df = 12, P = 0.000, CFI = 0.97 Model (female): $\chi^2 = 59.8$, df = 12, P = 0.000, CFI = 0.98

Fig. 3. Joint effects of antisocial behaviour and alcohol (mis)use on alcoholrelated trouble at age 15.

Table 3 shows the hypothesis supported by each model for males and females.

For each of the longer-term, shorter-term, and joint-effects models, each hypothesis was tested in respect of four different sub-groups (non-manual and manual social class; most recent alcohol from parents or another source) among both males and females. The upper sections of Tables 2 and 3 show that in the case of the longer-term models, all but one of these comparisons support the susceptibility hypothesis, no support being found for disinhibition. A single comparison supports the null hypothesis; no significant critical paths between alcohol (mis)use and antisocial behaviour (note effect sizes are low) occur among males who had been given alcohol by parents.

The middle sections of Tables 2 and 3 show the results of subgroup comparisons for the shorter-term models. Of eight comparisons, none support the disinhibition hypothesis, four support susceptibility, and four the reciprocal hypothesis (that is, both disinhibition and susceptibility). The reciprocal hypothesis receives support among females, regardless of social class, and among males from manual backgrounds. However, among males from non-manual backgrounds, the susceptibility hypothesis is supported. With respect to drinking context, the susceptibility hypothesis receives support

Table 3. Summary of comparison of longer-term (cross-lagged), shorter-term (simultaneous), and joint-effects (cross-sectional) models, showing hypothesis supported for males and females, and each subgroup

	Disinhibition	Susceptibility	Reciprocal	Null
Longer term				
(cross-lagged)				
models				
Overall	_	M, F	_	_
Social class	_	_	_	_
Non-manual	_	M, F	_	_
Manual	_	M, F	_	_
Alcohol from				
Parents		F	_	M
Other source	_	M, F	_	_
Shorter-term				
(simultaneous)				
models				
Overall	_	_	M, F	_
Social class	_	_	_	_
Non-manual	_	M	F	_
Manual	_	_	M, F	_
Alcohol from	_	_	_	_
Parents	_	M, F	_	_
Other source	_	F	M	_
Joint-effects				
(cross-				
sectional)				
models				
Overall	_	_	M, F	_
Social class	_	_	_	_
Non-manual	_	F	M	_
Manual	_	M	F	_
Alcohol from	_	_	_	_
Parents	_	_	_	M, F
Other source	_	_	M, F	_

among females regardless of source, and among males who had most recently obtained alcohol from parents. However, among males who had obtained alcohol from another source, the reciprocal hypothesis is supported.

The results of the subgroup comparisons for the jointeffects models of alcohol (mis)use and antisocial behaviour on alcohol-related trouble at age 15 are shown in the lower sections of Tables 2 and 3. Again, none of the eight comparisons support the disinhibition hypothesis. The susceptibility hypothesis is supported in two, the reciprocal in four, and the null in two. An interaction effect between social class and gender is suggested; the susceptibility hypothesis receives support among females from non-manual but males from manual backgrounds, but the reciprocal hypothesis among males from non-manual and females from manual backgrounds. This suggests that the alcohol (mis)use \Rightarrow alcohol-related trouble path is stronger in middle-class males and working-class females. With regard to context, the null hypothesis is supported in respect of those (both males and females) given alcohol by parents, and the reciprocal among those who most recently obtained it from another source.

Overall pattern

In combination, these results imply a gradual shift in support from susceptibility in the longer term through reciprocal in the shorter-term and joint-effects models, as would be expected if alcohol (mis)use had some (more immediate) effect on antisocial behaviour. Importantly, however, where a reciprocal effect exists, the paths representing the susceptibility hypothesis are generally stronger than those representing disinhibition.

DISCUSSION

Using data on young people in the west of Scotland, this paper has tested three competing hypotheses about the relationships between alcohol (mis)use and antisocial behaviour both in the longer and shorter term, and of alcohol (mis)use, antisocial behaviour and alcohol-related trouble in a joint-effects model. Note that the study used data collected at ages 11–15, thus, any purchase or public drinking of alcohol is 'under-age'.

Overall, the results strongly support the susceptibility hypothesis; that is, they reveal antisocial behaviour to be a substantive cause of, or predisposing factor to, alcohol (mis)use, a pattern observed in most sub-groups. There were no comparisons which supported 'pure' disinhibition. In every case where there was some evidence for a disinhibition effect [alcohol (mis)use predicting antisocial behaviour] antisocial behaviour still predicted alcohol (mis)use. These findings echo the conclusion drawn by White et al. (1993) from a similar, American, longitudinal study of adolescent males. However, it is notable that the susceptibility hypothesis received most support in the longer-term models and least in the joint-effects analyses. Thus, paths representing a disinhibition effect began to emerge as the time lag became shorter. These results are compatible with recent qualitative work on alcohol and violence suggesting that alcohol has a 'magnifying' effect, either amplifying underlying aggressive tendencies or conferring extra salience to relatively minor irritations (Rolfe et al., 2006).

Notwithstanding this overall conclusion, there were exceptions to the general trend, and different patterns according to gender, social class, or drinking context which merit comment. There was evidence of gender differences according to social class in both the shorter term and joint-effects models. While the susceptibility path was significant in all groups, a disinhibition effect was also significant among all except middle-class males in the shorter term, but among middleclass males and working-class females in the joint-effects models. Thus different patterns were seen in the shorterterm and joint-effects models, suggesting different effects for certain class and gender groups (particularly middle-class males), depending on whether the analysis focused on antisocial behaviour more generally, or alcohol-related trouble more specifically. It has been suggested that fights after drinking may be less strongly associated with social background than non-alcoholic aggression or delinquency (Wells et al., 2006).

There was also evidence of different effects according to where respondents reported, at age 15, that they had most recently obtained alcohol, a measure which we take to represent drinking context. There was no support for a

disinhibition effect in either the shorter-term or joint-effects models, among those who had recently been given alcohol by parents. In the absence of a better measure, we assume this represents those drinking with parental approval. In contrast, for those who had obtained alcohol from another source, disinhibition effects were evident among males in the shorter term and both males and females in the joint-effects analyses. This evidence of disinhibition is consistent with those studies which have suggested that alcohol-related problems are more likely among young people who drink in settings away from home (McKechnie *et al.*, 1977; Ghodsian and Power, 1986; Brain and Parker, 1997; Wells *et al.*, 2005). This has been attributed to the lack of adult supervision and restriction of inappropriate behaviour, together with the greater amounts of alcohol consumed in such circumstances (Wells *et al.*, 2005).

While gender differences were seen in respect of both class and recent drinking context, there was little evidence of a tendency towards stronger disinhibition effects among females, as might be suggested by evidence that females in general are more prone to the physiological effects of alcohol (Schuckit *et al.*, 1998), at least in relation to conventionally measured antisocial behaviour and alcohol-related trouble. This suggests that the explanations for gender differences may lie elsewhere, for example, alcohol-related expectancies (Engineer *et al.*, 2003). One recent study summarized the gendered nature of British alcohol-related expectancies in the phrase 'Men become violent, women become lippy' (Rolfe *et al.*, 2006).

Limitations

This is a longitudinal study, focussing on the relationships between alcohol (mis)use, antisocial behaviour and alcoholrelated trouble over particular time periods. The evidence shows that different time lags lead to different conclusions. A crucial factor is the time lag between actual alcohol (mis)use, antisocial behaviour, and alcohol-related trouble, and when these behaviours were reported in our surveys. Since we could not measure either alcohol (mis)use or disruptive behaviour at the time they occurred, relying instead on individuals' later reports, it is likely that both the shortterm and joint-effects models do not capture immediate reactions, but rather, describe recent patterns of alcohol (mis)use on antisocial behaviour or alcohol-related trouble. Given experimental evidence of the disinhibiting effects of alcohol, it is probable that the alcohol (mis)use to alcoholrelated trouble effect is underestimated. Alternatively, in the cross-sectional analysis, it is possible that respondents are prone to post-hoc reconstruction, and therefore, show bias in attributing their behaviour to alcohol. However, any study which attempted to record immediate reactions would be ethically difficult (allowing potentially dangerous antisocial behaviour), and either require an experimental (automatically limiting generalizability) or observational design. The current method, while imperfect, gets about as close to an optimal design as is ethically and practically possible in a community based cohort study.

It is also possible that the results may not generalize beyond the west of Scotland, or the UK, given the particularly high levels of alcohol consumption among contemporary British youth and the widespread belief in the UK that alcohol leads to violence. Thus, our results may provide evidence for a disinhibition effect specific to British youth.

Finally, some limitations of the modelling method should be noted, particularly in respect of the possibility of model misspecification, omitting key variables or failing to incorporate important correlated errors (Shadish *et al.*, 2002). We report average rather than individual behaviour, which will obfuscate rare exceptions to a trend such as that expressed by extremely disruptive individuals. Thus, it may be that major differences in the more extreme groups have been masked by responses of more average individuals.

CONCLUSION

The results of this study of alcohol (mis)use, antisocial behaviour, and alcohol-related trouble in young people suggest that antisocial behaviour is a substantive cause of, or predisposing factor to (under-age) alcohol (mis)use over both the longer and shorter term. However, the effects of alcohol (mis)use on antisocial behaviour or alcohol-related trouble begin to emerge as the time lag reduces. The results also suggest that future studies should address further the issue of variability between sub-groups in respect of the disinhibiting effects of alcohol, in particular those who drink away from home.

Although the dramatic rise in consumption is certainly a cause for concern, it is important not to over-stigmatize alcohol use since most young people consider drinking and learning to drink to be a normal part of development. Strategies advocating abstinence are likely to fail (Coleman and Cater, 2005). Other alcohol policies are inconsistent, the advocacy of tolerance zones for under-age drinkers being clearly incompatible with strict enforcement of age restrictions (Coleman and Cater, 2005). In the light of the failure of current UK policies to reduce alcohol use among under-age drinkers, some commentators have argued for an alternative approach, that of increasing the price of alcohol (Plant and Plant, 2006; Sweeting, 2006). Judging the effectiveness of different strategies requires both a comprehensive evaluation of their relative efficacy and monitoring for unintended consequences. Paradoxically, an unintentional effect of the publicity associated with mass campaigns (British Medical Journal, 2006) may be to reinforce the very assumptions they intend to challenge.

Despite the current assumption (in the UK, at least) that alcohol is a major cause of antisocial behaviour, the majority of young people perceive relatively few, or minor problems in relation to their own alcohol consumption and feel such 'minor difficulties' are a relatively small price to pay for the enjoyment associated with drinking (Coleman and Cater, 2005). Objective evidence suggests that this is an unrealistic perception. As evidenced by alcohol-related disorder and accidents, the burden of short-term harm is borne particularly by young British drinkers (Plant and Plant, 2006). Our study suggests that this is especially true of those with a predisposition to antisocial behaviour.

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