Associations Between Adolescent Heavy Drinking and Problem Drinking in Early Adulthood: Implications for Prevention

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ABSTRACT. Objective: We assessed how heavy episodic drinking (HED) in adolescence (Time 1) was related to hazardous drinking as well as symptoms of alcohol problems and dependence in early adulthood (Time 2). The key question was to what extent preventive measures targeted at underage HED may have a potential to reduce problem drinking in early adulthood. **Method:** Data are from the 1992 (Time 1, ages 14–17 years) and 2005 (Time 2) waves of the Young in Norway Longitudinal Study (N = 1,764). In addition to odds ratios and relative risks, we calculated population-attributable fractions to estimate how the prevalence of hazardous drinking and alcohol problems in early adulthood would be affected if adolescent HED at various frequencies were eliminated. The results were adjusted for age, gender, and measures on impulsivity and delinquency. **Results:** The risk of problem drinking at Time 2 increased with increasing frequency of HED at Time 1, but a great deal of discontinuity in drinking behaviors was also observed. The population-attributable fractions indicated that if all instances of HED at Time 1 were eliminated, the expected reduction in hazardous drinking and alcohol problems at Time 2 would be 11% and 15%, respectively. **Conclusions:** Because of a marked discontinuity in drinking behaviors from adolescence to early adulthood, the potential long-term effects of interventions targeted at HED among youth are likely to be limited. (*J. Stud. Alcohol Drugs, 73,* 542–548, 2012)

A SIZEABLE PROPORTION OF DEATHS and disease burden among youth in the developed world is attributable to drinking (Toumbourou et al., 2007). In addition to short-term negative outcomes such as accidents and intoxicated aggression, there is evidence to suggest that brain development during adolescence may be adversely affected by alcohol (Lubman et al., 2007; Monti et al., 2005; Zeigler et al., 2005). Moreover, recent research indicates that extensive drinking during the teen years may enhance the risk for a range of poor outcomes in adulthood, including health problems, low educational attainment, and social adversity (Hill et al., 2000; Odgers et al., 2008; Viner and Taylor, 2007).

Against this background, the widespread concern about young people's use of alcohol seems highly justified. To some extent, this concern also appears to rely on the assumption that early heavy drinking sets the stage for drinking habits later in life. Hence, the long-term ambition of many preventive measures targeted at alcohol use by youth is to reduce the risk of problem drinking in early adulthood as well (e.g., Ferrer-Wreder et al., 2005). However, whether such an aim is likely to be achieved hinges on, among other things, the extent of continuity in drinking during this phase of the life span. The current article scrutinizes this issue from a public health perspective by analyzing panel data on drinking behaviors from a general population study of young people.

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Stability and change in drinking from adolescence to early adulthood

Numerous longitudinal studies based on general population samples have revealed that heavy drinking in the teen years is prospectively related to problem drinking in early adulthood (Bonomo et al., 2004; Cable and Sacker, 2008; Ellickson et al., 2003; Huurre et al., 2010; Jefferis et al., 2005; McCarty et al., 2004; Riala et al., 2004; Viner and Taylor, 2007; Wells et al., 2004). For example, Wells et al. (2004) revealed strong associations between drinking at age 16 and various indicators of problem drinking at ages 21–25. Correspondingly, Huurre et al. (2010) reported that alcohol use in the mid-teens, notably an intoxication-oriented drinking style, predicted high scores on the Alcohol Use Disorders Identification Test (AUDIT) in the early 30s. Moreover, McCambridge et al. (2011) reported, in a summary of this research literature, that all the studies they had reviewed had found significant associations between teenage drinking and adult alcohol problems or dependence. However, a great deal of discontinuity in drinking has been reported as well, particularly in light and moderate drinking (McCartey et al., 2004; Pape and Hammer, 1996) and when a longer time span is considered (Jefferis et al., 2005; Temple and Fillmore, 1985).

There are several plausible explanations for why adolescent heavy drinking is related to problem drinking in early adulthood. First, because of the vulnerability of the developing brain, extensive use of alcohol in adolescence may have neuropsychological (Yücel et al., 2007) and epi-

genetic effects (Wong et al., 2011) that increase the risk of long-lasting alcohol dependence. Second, irrespective of the drinker's age, frequent drinking may lead to alcohol dependence because of the psycho-pharmacological properties of alcohol. Third, if an identity as a heavy drinker is established early in life, excessive drinking habits may be bolstered and maintained through differential associations with heavy-drinking peers (Akers et al., 1979). Stigmatization and social exclusion may reinforce such processes. Last, because of the reinforcing effects of alcohol and the sheer power of habit, a pattern of heavy drinking may persist over time.

In this context it is also worth noting a related strand of research that has provided solid evidence of an association between an early onset of drinking and problem drinking later in life. In their much-cited study, Grant and Dawson (1997) found that ". . . the odds of lifetime alcohol dependence were reduced by 14% with each increasing year of age at first use" (p. 108). Several researchers have interpreted such findings in causal terms and proposed that preventive measures should be aimed at delaying the onset of drinking to reduce the risk of alcohol problems in adulthood (see Rossow, 2006, for a review). However, others have questioned the notion of causality and offered alternative explanations of the association at issue.

Besides the possible causal effects of early drinking mentioned above, continuity in heavy drinking may also be attributable to stable underlying influences. For instance, several studies have shown that individual characteristics such as impulsivity (Acton, 2003; Dawe et al., 2004) and behavioral undercontrol (e.g., delinquency) (Donovan and Jessor, 1985; Windle, 1990) both increase the risk of excessive use of alcohol in adolescence as well as early adulthood.

Aim

The primary aim of the current study was to assess how the frequency of intoxication in adolescence (Time 1) is related to hazardous drinking as well as symptoms of alcohol problems and dependence in early adulthood (Time 2). Our modeling includes controls for factors that may affect drinking behavior at both Time 1 and Time 2, i.e., age and gender, and measures on impulsivity and general delinquency.

Method

Data

Data were obtained from the Young in Norway Longitudinal Study, which has been described in detail elsewhere (Strand and von Soest, 2007). The data we required were collected in the first (1992) and fourth (2005) waves. In the following, we will refer to the 1992 survey and the 2005 survey as Time 1 and Time 2, respectively. The initial sample was established by selecting schools from a national register of all junior and senior high schools, and the sampling procedures were designed to obtain a representative cross section of this student population in Norway. All students in the selected schools were included in the first survey (Time 1), whereas the follow-up in 2005 (Time 2) was confined to respondents who initially attended 7th or 10th grade. At Time 1, the questionnaires were distributed and completed in the classroom. At Time 2, the respondents could choose to fill out a postal or web-based questionnaire or be interviewed by phone. The overall response rate was 67%. We restricted the analyses to respondents aged 14–17 years at Time 1 (n = 1,751) and, hence, below the minimum legal age for purchasing alcohol in Norway. The respondents' mean age was 15.5 years at Time 1 and 28.6 years at Time 2 (SD = 1.2), and a bare majority (56%) were female.

Attrition

The following characteristics at Time 1 predicted subsequent attrition: gender (being male), frequent involvement in deviant behaviors, low parental socioeconomic status, poor school grades, low parental monitoring, urban or suburban residence, and the participant's prediction that he or she will be doing manual work at 40 years of age (Pedersen, 2007; Storvoll and Wichstrøm, 2003). Moreover, compared with population data from official registers, the sample had a lower prevalence of crime charges (Pedersen and Skardhamar, 2010).

Measures

At Time 1, heavy episodic drinking (HED) was measured using the following question: "During the past 12 months, have you drunk so much that you felt clearly intoxicated?" There were six response options: *never* (coded 0), *once* (1), *2–5 times* (3.5), *6–10 times* (8), *11–50 times* (30), and *more than 50 times* (55).

At Time 2 we used the AUDIT, which is a well-established test for screening problem drinking and alcohol problems (Babor et al., 2001). It comprises the following 10 items: (1) How often do you have a drink containing alcohol? (2) How many drinks containing alcohol do you have on a typical day when you are drinking? (3) How often do you have six or more drinks on one occasion? (4) How often during the last year have you found that you were not able to stop drinking once you had started? (5) How often during the last year have you found that you failed to do what was normally expected from you because of drinking? (6) How often during the last year have you found that you needed a first drink in the morning to get yourself going after a heavy drinking session? (7) How often during the last year have you found that you had a feeling of guilt or remorse after drinking? (8) How often during the last year have you been unable to remember

what happened the night before because you had been drinking? (9) Have you or someone else been injured as a result of your drinking? (10) Has a relative or friend or a doctor or other health worker been concerned about your drinking or suggested you cut down? Items 1–8 had five response options, which we coded in accordance with prevailing guidelines as follows (Babor et al., 2001): *never* (coded 0), *less than monthly* (1), *a few times a month* (2), *a few times a week* (3), and *daily or almost daily* (4). Items 9–10 had three response options: *never* (coded 0); *yes, but not in the last year* (2); and *yes, during the last year* (4). The complete AUDIT is typically converted into an additive scale with a maximum score of 40. However, it is generally recognized that AUDIT does not tap a single dimension of problem drinking (Bergman and Källmén, 2002). Moreover, the majority of those who score positive on AUDIT have no positive score on the alcohol problem or dependence items (i.e., Items 4–10), only on the consumption items (i.e., Items 1–3). Therefore, we constructed two dichotomous outcomes that, on the basis of the factor analytical structure, were suggested by Bergman and Källmén (2002): *hazardous consumption* (Items 1–3) and alcohol-related problems/dependence (Items 4–10, hereafter referred to as *alcohol problems*).

In the general population, the prevalence of positive cases using AUDIT-10 is typically around 20% when applying the conventional cutoff of ≥8 (Hradilova Selin, 2006). In the present sample of fairly young people, a prevalence close to that (19.3%) was obtained by choosing a cutoff of ≥10. We thus chose cutoffs for hazardous consumption (≥7) and alcohol problems (≥4) that yielded prevalences as close as possible to 20% (22.1% and 18.1%, respectively).

Impulsivity was measured at Time 2 using six items based on Eysenck's Personality Questionnaire (Eysenck and Eysenck, 1978) and the Barratt Impulsiveness Scale (Patton et al., 1995), including statements such as "I make up my mind quickly" and "I act on the spur of the moment." The response options ranged from 1 (*corresponds very poorly*) to 4 (*corresponds very well*). An additive index was used in the analyses (Cronbach's $\alpha = .72$). Delinquent behavior was also assessed at Time 2. A sum score was constructed based on 11 dichotomous measures of lifetime involvement in delinquency and norm-violating activities, such as theft, violent behavior, and false insurance claims.

Statistical analyses

The statistical analyses can be described through the following steps. First, we estimated logistic regression models, one for each of the two outcomes. In addition to HED at Time 1, the models included the following covariates as controls: age, sex, impulsivity, and delinquent behavior (the latter two measured at Time 2). Second, the estimated odds ratios (OR) associated with the various levels of HED were converted into relative risks (RR) following standard procedures (Zhang and Yu, 1998).

Third and finally, we calculated population-attributable fractions (PAFs). These fractions express the expected percentage reduction of cases if exposure at a specific level is eliminated. We followed the standard procedure for calculating PAFs when there are multiple exposure levels (Hanley, 2001). The PAF for the exposure level *i* was thus calculated according to the following:

$$PAF_i = p_i \times (RR_i - 1)/RR_i,$$

where p_i is the percentage of cases at exposure level *i,* and RR_i is the relative risk at exposure level *i.*

Two clarifications may be in order. First, the reason for converting OR into RR is that the latter is the appropriate risk measure to use when calculating PAF. It is not uncommon to substitute OR for RR in this context. However, the

Table 1. Associations between frequency of heavy episodic drinking (HED) in adolescence (Time 1) and hazardous consumption and alcohol problems in early adulthood (Time 2), controlling for age, gender, impulsivity, and delinquency: Logistic regression analyses (n = 1,751)

		Hazardous consumption at Time 2			Alcohol problems at Time 2		
Variable	%	*B*	*SE*	OR [95% CI]	*B*	*SE*	OR [95% CI]
Frequency of HED at Time 1							
No times	58.9			1.00			1.00
Once	7.9	0.03	0.25	1.03 [0.64, 1.67]	-0.04	0.27	0.96 [0.57, 1.62]
2–5	14.2	0.17	0.21	1.18 [0.63, 1.67]	0.37	0.21	1.45 [0.96, 2.18]
6–10	6.6	0.24	0.28	1.27 [0.74, 2.18]	0.53	0.27	1.70 [0.99, 2.89]
11–50	10.9	0.69	0.22	2.00 [1.29, 3.10]	0.59	0.23	1.81 [1.15, 2.90]
>50	1.5	1.04	0.46	2.83 [1.14, 7.00]	1.10	0.47	3.00 [1.18, 7.60]
Age		-0.35	0.06	0.71 [0.63, 0.79]	-0.24	0.06	0.79 [0.70, 0.89]
Gender, male = 1		1.42	0.03	4.15 [3.17, 5.44]	0.75	0.14	2.11 [1.59, 2.78]
Impulsivity		0.69	0.15	2.00 [1.48, 2.65]	0.28	0.03	1.32 [1.24, 1.41]
Delinquency		0.19	0.03	1.21 [1.14, 1.29]	0.95	0.16	2.58 [1.24, 1.41]

Notes: OR = odds ratio; CI = confidence interval.

TABLE 2. Relative risks (RR) and population-attributable fractions (PAF) of hazardous consumption and alcohol problems in early adulthood (Time 2) by frequency of heavy episodic drinking (HED) in adolescence (Time 1), controlling for age, gender, impulsivity, and delinquency

Frequency of HED at Time 1	Hazardous consumption at Time 2		Alcohol problems at Time 2		
	RR	PAF	RR	PAF	*n*
No times	1.00	–	1.00	–	1,032
Once	1.02	0.17	0.96	0.00	138
2–5	1.14	1.74	1.35	4.32	248
6–10	1.20	1.20	1.54	3.03	116
11–50	1.66	6.38	1.61	5.66	190
>50	2.06	1.45	2.30	1.71	27
Sum	–	10.95	–	14.72	1,751

difference between OR and RR is substantial if (as in the present application) OR exceeds 2.5, and p_0 is larger than 0.1 (Zhang and Yu, 1998). Second, the estimate of, for example, PAF_5 in the present application answers the question: How much would the prevalence of problem drinking at Time 2 decrease if the individuals in the top category of HED (Level 5) at Time 1 had not been drinking heavily at all?

Results

The first column in Table 1 reveals that a majority (59%) of the respondents had not engaged in HED at Time 1, whereas less than 2% had done so more than 50 times. Further, the table shows that HED in adolescence (Time 1) and all control variables were significantly associated with both hazardous drinking and alcohol problems in early adulthood (Time 2).

The PAFs (Table 2) provide additional insight into the nature of the associations. It appears that if HED at Time 1 in the top category (i.e., >50 times) is eliminated (i.e., if the individuals in this category instead had been in the lowest category), a 1.5% reduction in hazardous drinking at Time 2 could be expected. Extending the reduction in HED at Time 1 to the next highest frequency category (11–50 times), and thus including the top 13%, would yield an additional expected decrease of about 6%. Finally, if HED at Time 1 is eliminated altogether, the reduction in hazardous drinking at Time 2 would be 11%. The corresponding analyses of alcohol problems at Time 2 revealed a similar pattern, although the impact of HED at Time 1 was somewhat stronger. More precisely, the results indicated that an elimination of highly frequent HED (i.e., >50 times) at Time 1 is expected to reduce the prevalence of alcohol problems at Time 2 by 2%. Further, a scenario implying no HED at all at Time 1 is expected to reduce the prevalence of alcohol problems at Time 2 by approximately 15%.

The outcomes reported above suggest that the stability in drinking from adolescence to early adulthood was fairly low.

TABLE 3. Hazardous alcohol consumption in early adulthood (Time 2) by frequency of heavy episodic drinking (HED) in adolescence (Time 1): Crosstabulation with row percentages (**in boldface**) and column percentages (*in italics*) (*n* = 1,751)

Frequency of HED at Time 1	Hazardous consumption at Time 2		Total
	No	Yes	
No times	**79.6**	**20.4**	**100.0**
	60.0	*55.1*	*58.9*
Once	**77.5**	**22.5**	**100.0**
	7.8	*8.1*	*7.9*
2–5 times	**79.4**	**20.6**	**100.0**
	14.4	*13.3*	*14.2*
6–10 times	**77.6**	**22.4**	**100.0**
	6.6	*6.8*	*6.6*
11–50 times	**71.6**	**28.4**	**100.0**
	9.9	*14.1*	*10.9*
>50 times	**63.0**	**37.0**	**100.0**
	1.2	*2.6*	*1.5*
Total	**78.1**	**21.9**	**100.0**
	100.0	*100.0*	*100.0*

The cross-tabulation in Table 3 indeed confirms that this was the case. Thus, the majority (63%) of the respondents in the heaviest drinking category at Time 1 did not score as hazardous drinkers at Time 2. Conversely, 55% of the hazardous drinkers at Time 2 reported no HED at all when they were assessed as teenagers.

Discussion

Our analyses of panel data from a general population study revealed that HED at ages 14–17 was significantly related to measures on hazardous consumption and alcohol problems at ages 27–30. Although precise comparisons with previous studies are hampered by differences in measures and follow-up periods, the effect sizes that we report (i.e., the OR in Table 1) are consistent with reasonably comparable studies (e.g., Huurre et al., 2010; Jefferis et al., 2005; McCarty et al., 2004; Viner and Taylor, 2007).

However, a shift in risk assessment occurred when we expanded previous research by estimating PAFs—which are more relevant when considering possible implications for alcohol policy and prevention. These estimates indicated that if all episodes of HED by underage youth were completely eliminated, the expected reduction in hazardous drinking in young adulthood would be about 10%. The corresponding estimate for alcohol problems was about 15%.

More than 40% of the respondents in our sample reported HED at Time 1, and the low attributable fractions reflect the fact that the discontinuity in drinking behaviors from adolescence to early adulthood was rather substantial. Thus, most of the hazardous drinkers in early adulthood (Time 2) reported no HED in adolescence, and even among the heaviest adolescent drinkers, a solid majority were not hazardous drinkers at Time 2.

Implications for prevention

Based on evidence of statistically significant associations between adolescent drinking and adult problem drinking, Cable and Sacker (2008) claimed that early interventions may be required to reduce alcohol-related problems later in life. Others have stated more explicitly that priority should be given to groups with an excess risk of problem drinking in adulthood, such as early initiators of alcohol use and youth who get drunk frequently (Bonomo et al., 2004; Ellickson et al., 2003; Huurre et al., 2010; Riala et al., 2004). It has also been proposed that "efforts to prevent and treat adolescent problem drinking are likely to have an impact on adult drinking patterns and may therefore have longstanding effects on public health" (McCarty et al., 2004, p. 718).

However, our findings suggested that the potential long-term effect of early interventions targeting excessive drinking is likely to be limited. Even if an intervention succeeded in curbing all adolescent HED, the estimated reduction in problem drinking in early adulthood was quite small (10%–15%). Moreover, eliminating all episodes of heavy drinking among adolescents is hardly possible, implying that the real-life outcome would be even smaller. Thus, school-based programs and other educational approaches to reduce underage drinking rarely work as intended, and favorable long-term effects are particularly hard to achieve (Babor et al., 2010; Foxcroft et al., 2002, 2003; Pape, 2009).

The policy implication of our results accords with the so-called prevention paradox: Despite heavy drinkers having the highest risk of alcohol-related problems, most of these problems are found in the group of light and moderate drinkers. The risk is certainly smaller in the latter group, but this is made up by its sheer size (Kreitman, 1986). Linking up with the present study, the prevention paradox as well as the marked discontinuity in drinking that we observed suggest that priority should be given to preventive measures targeting the entire adolescent population. Nondrinkers are of particular importance here, as this group seems to comprise a large recruitment basis for heavy drinkers later in life. These considerations are pointers to general measures that have proved to be efficient, such as minimum legal age limits, high alcohol excise taxes, and restrictions on outlet density and sales hours (Babor et al., 2010).

Methodological considerations

Our outcome measures at Time 2 were both based on the AUDIT, which has excellent psychometric properties (Reinert and Allen, 2007). However, HED at Time 1 was gauged by a single indicator—that is, frequency of self-perceived intoxications. Lintonen and Rimpelä (2001) examined the validity of this commonly used measure in a general population survey of teenagers. They collected detailed information about the intake of alcohol at the last drinking event to estimate the maximum blood alcohol concentration and found that this estimate correlated strongly with the respondents' perceived intoxication at that event. Other studies suggest that the frequency of intoxications is the best predictor of social and behavioral problems related to drinking (Bailey, 1999; Hauge and Irgens-Jensen, 1986; Midanik, 1999, 2003). Moreover, because it may be especially hard for adolescents to estimate their consumption in terms of standard units, Lintonen and Rimpelä (2001) argued that one should rely on self-perceived intoxication as a simple and valid indicator of youthful heavy drinking.

However, measurement errors are unavoidable, implying a risk of underestimating the continuity of drinking. The panel attrition may also have induced a downward bias because the factors that predicted attrition (e.g., being male, involvement in deviant behaviors, poor school grades) are likely to be associated with problem drinking at Time 2. On the other hand, other sources of bias may work in the opposite direction. More specifically, the association at issue may to a certain extent reflect the impact of stable factors that affect both adolescent and young adult drinking behaviors. We did control for two such factors (impulsivity and delinquency), but ideally they should have been measured at Time 1 rather than Time 2. More importantly, additional confounders probably exist. For instance, a recent twin study suggested that a sizable part of the association between early drinking and later alcohol dependence is attributable to genetic factors (Sartor et al., 2009). Because of the lack of sufficiently detailed information to evaluate the magnitude of these various sources of bias, it is hardly possible to assess their net effect.

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