International Journal of Methods in Psychiatric Research
Int. J. Methods Psychiatr. Res. 17(S1): S30–S38 (2008)
Published online in Wiley InterScience
(www.interscience.wiley.com) **DOI**: 10.1002/mpr.242

The adolescent origins of substance use disorders

MATT MCGUE,[1,2] WILLIAM G. IACONO[1]

1 Department of Psychology, University of Minnesota, Minneapolis, MN, USA
2 Department of Epidemiology, Southern Denmark University, Odense, Denmark

Abstract
Although early use of alcohol during adolescence has been consistently associated with increased risk of alcoholism in adulthood, the specific mechanisms that underlie this association remain unclear. We describe a program of epidemiological twin-family research that shows that early use of alcohol is best conceptualized as an indicator of a more general propensity to engage in adolescent problem behavior. Adolescent problem behavior, in turn, is a risk factor for a broad range of adult externalizing disorders, of which alcoholism is but one manifestation. These findings are shown to be consistent with a dual-process model whereby early adolescent problem behavior is associated with increased risk of adult psychopathology because both are indicators of a common inherited liability and because early adolescent problem behavior increases the likelihood an adolescent is exposed to high-risk environments. We conclude with a discussion of the importance of cross-cultural research, which may be especially informative for identifying the consequences of early adolescent drinking.

Key words: adolescent drinking, disinhibitory psychopathology, adolescent problem behavior, cross-cultural alcohol research

One of the most robust findings in the alcohol research field is the association between an early initiation of alcohol use and risk of alcohol dependence. In a highly cited influential study on a large US epidemiological sample, Grant and Dawson (1997) reported that individuals who retrospectively report that they had first tried alcohol prior to age 15 were four times more likely to have a lifetime diagnosis of alcohol dependence than individuals you first tried alcohol after the age of 20. This finding has been replicated in several retrospective studies (Dewit et al., 2000; Hingson et al., 2006; McGue et al., 2001a; Prescott and Kendler, 1999) and at least one prospective study (Keyes et al., 2006). Unfortunately, however, all of these studies have been based on US samples, an important limitation to which we will return later.

The consistency and strength of the association between early alcohol use and alcoholism suggest that it has implications for efforts to prevent alcoholism onset. The nature of these implications, however, necessarily depends on the specific mechanisms that underlie the association. Two alternative mechanisms have been proposed. First, early use of alcohol may increase alcoholism risk by altering the course of adolescent development (Dewit et al., 2000). Adolescent alcohol use could alter the course of development by decreasing the likelihood that an adolescent is affiliated with individuals and institutions that value and model sobriety and increasing the likelihood an adolescent is affiliated with individuals that reinforce and model deviance. Early use of alcohol may also influence the course of development indirectly, through its effects on the developing adolescent brain (Tapert et al., 2004). Research with rodents (Spear, 2002) and humans (Brown and Tapert, 2004) suggest that heavy use of alcohol during adolescence can result in neurocognitive changes that increase the likelihood of subsequent abuse of alcohol in adulthood.

Alternatively, others have suggested that the association of early alcohol use with alcoholism risk is spurious, arising because early use of alcohol in adolescence and alcoholism in adulthood are both manifestations of a general inherited liability to disinhibitory psychopathology (Krueger et al., 2002; Young et al., 2000). Support for this proposition came initially from a study of nearly 9000 twins, which showed that early use of alcohol was heritable and its association with alcoholism risk was mediated entirely by genetic factors (Prescott and Kendler, 1999). The common-inherited-liability model also predicts that, because it is an indicator of a general disposition towards disinhibited behavior, early use of alcohol should be a non-specific risk factor for a wide range of behavioral pathologies. Consistent with this expectation, alcohol use prior to age 15 is associated with attention deficit/hyperactivity disorder, conduct disorder, personality measures of impulsivity, psychophysiological indicators of disinhibition, academic underachievement, and abuse of substances other than alcohol, even after its association with alcoholism risk has been taken into account (McGue et al., 2001a).

Although the alternative hypotheses for the association of early alcohol use with alcoholism may appear to be mutually exclusive, they need not be. Early use of alcohol may both be an indicator of inherited risk and disrupt the course of adolescent development. In this paper, we summarize a program of research that has sought to explore the possible dual nature of risk associated with early adolescent use of alcohol. We begin with a brief description of the research context upon which our investigations are based, and then address three issues: (1) the non-specific nature of risk associated with early use of alcohol, (2) the heritable basis of early use of alcohol, and (3) the environmental consequences of early alcohol use. We end with a general discussion that includes speculation about the cross-national generalizability of our findings.

Methods

Sample

The Minnesota Twin Family Study (MTFS) is a longitudinal study of a community-based sample of two cohorts of twins and their parents. The older (i.e. age 17) cohort had a mean age of 17.5 years [standard deviation (SD) = 0.45] at the intake assessment and consisted of 626 pairs of like-sex twins [189 monozygotic male (MZM), 100 dizygotic male (DZM), 223 MZ female (MZF) and 114 DZ female (DZF)]. The younger (i.e. age 11) cohort had a mean age of 11.7 years (SD = 0.44) at the intake assessment and consisted of 756 pairs of like-sex twins (254 MZM, 122 DZM, 233 MZF, and 147 DZF). The MTFS sample was identified from birth records from the US state of Minnesota and is broadly representative of families with twins born in that state. A complete description of the recruitment of the MTFS sample as well as evidence of sample representativeness is given in Iacono et al. (1999). The MTFS samples have been followed longitudinally every 3–4 years, with in-person follow-up assessments timed to coincide with major life transitions in the life of American adolescents and young adults (Figure 1). Currently, we are completing the fourth follow-up assessment of the younger cohort, at age 24–25, and the third follow-up assessment of the older cohort, at age 29. There has been limited sample attrition over the successive waves of assessments, with from 80–93% of surviving twins participating at each follow-up assessment. For example, Elkins et al. (2006) report that in the older cohort 83% of males and 93% of females from the older cohort completed their first follow-up assessment, while McGue at al. (2005) reported that 92% of males and 94% of females from the younger cohort completed their first follow-up assessment. Importantly, in all cases non-participants differ minimally, although in some cases significantly, from participants in terms of their status on the intake assessments.

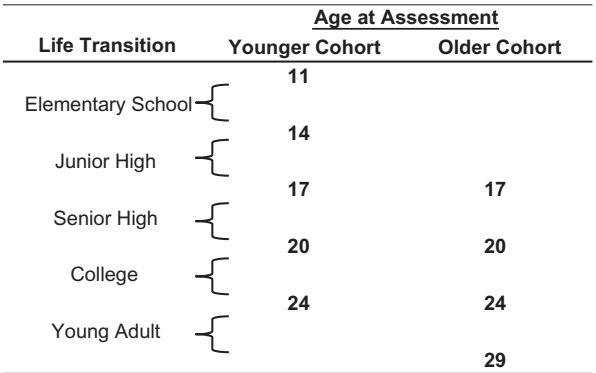

Figure 1. Longitudinal design of the MTFS: The MTFS includes two cohorts of twins assessed every 3–5 years with assessments timed to coincide with major transitions in the lives of US adolescents and young adults.

DOI: 10.1002/mpr

The MTFS thus depends heavily on the classical twin study comparison of monozygotic (MZ) and dizygotic (DZ) twins, which is not without its critics. Nonetheless, research has generally supported the validity of the twin study approach (Kendler, 1993) and has shown that twins are not psychologically different from non-twins (Christensen et al., 2006; Johnson et al., 2002).

Assessment

At intake, twins and their parents completed a day-long, in-person assessment that included a comprehensive clinical interview for common mental and substance use disorders using the Diagnostic and Statistical Manual of Mental Disorders (DSM); self-report measures of personality and substance use; measures of academic aptitude and achievement; a battery of psychophysiological brain-wave and autonomic indicators of risk; and an extensive assessment of environmental risk and protective factors that included measures of peer group characteristics, family functioning, religiousness, and life stress. At each follow-up assessment, measures in each of these domains are repeated, adapted for the changing developmental levels of the participants as necessary. There are several noteworthy features of the MTFS assessments. First, interview assessments are administered by interviewers who have either a bachelor's or master's degree in psychology or related field and who completed an extensive training program and were required to meet proficiency criteria prior to being allowed to interview study participants. Each family member is interviewed independently by a separate interviewer. Second, a consensus team consisting of at least two graduate students with advanced training in descriptive psychopathology and differential diagnosis review all relevant evidence, including audio tapes of interviews as needed, before any symptom is coded positive. Diagnoses and symptom counts according to DSM-III-R, the diagnostic standard current at the time the MTFS was started, are generated by computer algorithm. The reliability of this consensus process for the diagnoses we assess is uniformly high (Iacono et al., 1999).

Results

The non-specific nature of risk

Early use of alcohol is associated not only with risk of alcoholism but also with risk for a wide range of psychopathology. We investigated the correlates of early alcohol use in a sample of 1328 male and 1361 female ever-drinking parents from the MTFS (McGue et al., 2001a). Table 1 gives the odds ratios (ORs) relating alcohol use prior to age 15 with risk for the following DSM-III-R diagnoses: alcohol dependence (ALCDP), nicotine dependence (NICDP), drug dependence (DRGDP), antisocial personality disorder (ASPD), and major depressive disorder (MDD). Diagnoses were lifetime at a definite (all symptom criteria met) or probable (all except one symptom criterion met) level of

Table 1. Association of alcohol use prior to age 15 with psychopathology risk in 1328 male and 1361 female Minnesota adults

DSM-III-R diagnosis	OR (95% confidence interval)			
	Male sample		Female sample	
	Uncorrected	Corrected for alcohol dependence	Uncorrected	Corrected for alcohol dependence
Alcohol dependence	2.4 (1.8, 3.1)	–	4.1 (2.7, 6.1)	–
Nicotine dependence	2.1 (1.6, 2.7)	1.7 (1.3, 2.3)	2.6 (1.9, 3.7)	2.1 (1.5, 2.9)
Drug dependence	3.7 (2.8, 4.9)	3.2 (2.4, 4.3)	4.6 (3.2, 6.8)	3.6 (2.4, 5.4)
Antisocial personality	7.5 (4.5, 12.8)	5.8 (3.0, 10.3)	4.9 (0.8, 29.8)	2.5 (0.4, 6.6)
Major depression	1.4 (0.9, 2.0)	1.3 (0.9, 1.8)	1.5 (1.0, 2.1)	1.3 (0.9, 1.8)

Note: Odds ratios (ORs) give the relative increase in odds of having the disorder associated with first use of alcohol before the age of 15 as compared to first use after age 15 or not at all. Diagnoses are lifetime at a definite (all criteria met) or probable (one symptom short) level of certainty.

DOI: 10.1002/mpr

certainty. As can be seen, use of alcohol before the age of 15 is associated with a significantly increased risk of not only ALCDP, but also the other disinhibitory psychopathology diagnoses: NICDP, DRGDP, and ASPD. The association of early alcohol use with MDD, an internalizing form of psychopathology, is not as strong as its associations with indicators of disinhibitory psychopathology. Importantly, the associations with disinhibitory psychopathology remain significant even after their association with ALCDP has been statistically controlled.

Adolescent problem behaviors do not typically occur in isolation. Adolescents who drink are also more likely to smoke, use illicit drugs, and break parent or socially proscribed rules than are adolescents who do not drink (Jessor et al., 1991). The general nature of adolescent problem behavior suggests that it is essential that the risk associated with any specific behavior not be evaluated in isolation. We consequently investigated whether rate of adult alcoholism was elevated specifically among early drinkers or more generally among adolescents who engaged early in other problem behaviors (McGue and Iacono, 2005). Table 2 summarizes our findings for ALCDP in a sample of 1252 17-year-old twins (i.e. from the 626 pairs of like-sex twins that constitute the older twin cohort from the MTFS). Because 17-year olds who started drinking more than two years earlier will have had a longer period of time to progress to problem drinking than their age mates who had started drinking later, it is not surprising to find that use of alcohol prior to age 15 is strongly associated with ALCDP risk by age 17 (OR > 6.0 in both the male and female samples). More interesting is the finding that other adolescent problem behaviors are associated with ALCDP risk. In fact, the ORs for early smoking and early illicit drug use were substantially larger and the ORs for early troubles with police and sexual intercourse were nearly as large as those for early alcohol use. Significantly, the OR for every problem behavior except one (early sexual intercourse in females), remained significant after controlling for early use of alcohol. That is, adolescents who smoke, use illicit drugs, have troubles with police or sexual intercourse prior to age 15 are more likely to be alcohol dependent by age 17 even if they did not try alcohol prior to age 15.

In this study, we also showed that each of the early adolescent problem behaviors, in addition to being associated with ALCDP, is significantly associated with risk for NICDP, DRGDP, ASPD, and MDD at age 17. In order to assess the aggregate association of adolescent problem behavior with disinhibitory psychopathology, we further fit a latent variable model to the data. In this model, early adolescent problem behavior was quantified as the number of problem behaviors prior to age 15 and disinhibitory psychopathology was operationalized as the latent factor underlying symptoms of NICDP, ALCDP, DRGDP, and ASPD. The

Table 2. Association of problem behaviors prior to age 15 with alcohol dependence at age 17 in 578 male and 674 female Minnesota twins

Early problem behavior	OR (95% confidence interval)			
	Male sample		Female sample	
	Uncorrected	Corrected for early alcohol use	Uncorrected	Corrected for early alcohol use
Alcohol use	6.4 (3.9,10.6)	–	6.5 (3.7, 11.4)	–
Smoking	11.9 (5.1, 27.8)	7.5 (3.1, 17.9)	15.5 (6.6, 36.7)	10.6 (4.4, 25.8)
Illicit drug use	13.5 (5.5, 33.2)	6.9 (2.7, 18.1)	6.9 (3.3, 14.4)	3.1 (1.4, 7.0)
Police troubles	4.4 (2.5, 7.8)	3.0 (1.7, 5.5)	6.6 (2.6, 16.6)	3.6 (1.3, 9.9)
Sexual intercourse	6.4 (2.6, 15.6)	3.9 (1.5, 10.4)	4.5 (2.1, 10.0)	1.9 (0.8, 4.6)

Note: Odds ratios (ORs) give the relative increase in odds of having a diagnosis of alcohol dependence associated with first expression of each of the problem behaviors before the age of 15 as compared to not having engaged in that behaviour before. Alcohol dependence diagnoses are lifetime at a definite (all criteria met) or probable (one symptom short) level of certainty according to DSM-III-R criteria.

DOI: 10.1002/mpr

correlation between early adolescent problem behavior and adult disinhibitory psychopathology as significant and strong ($r = 0.75$), demonstrating that early adolescent problem behavior is a major risk factor for adult psychopathology. Taken together, our research implies that the association of early alcohol use with alcoholism risk should be conceptualized broadly rather than narrowly. Adolescent alcohol use is an indicator of a broader array of adolescent problem behaviors, each of which is independently predictive of alcoholism risk. Alternatively, alcoholism in adulthood, at least early-onset alcoholism, is in part an indicator of a broader spectrum of externalizing/disinhibitory disorders, each of which is associated with early adolescent problem behavior.

The association of early adolescent problem behavior with inherited risk

Prescott and Kendler (1999) hypothesized that the association of early use of alcohol with alcoholism risk arises because both are indicators of a general and heritable underlying dimension of vulnerability to disinhibitory psychopathology. Consistent with their hypothesis, we have found that early use of alcohol is familial and heritable, especially in boys (McGue et al., 2001b). We have also investigated the association of early use of alcohol with one of the major biological risk factors for alcoholism in particular and disinhibitory psychopathology in general, the P3 amplitude assessed in a visual odd-ball event-related potential paradigm (Iacono et al., 2002). Consistent with a biological hypothesis, reduced P3 amplitude is associated not only with early use of alcohol but also with the other indicators of early adolescent problem behavior (Iacono and McGue, 2006).

We have also investigated whether early adolescent problem behavior is heritable by determining whether MZ twins are more correlated for adolescent problem behavior than DZ twins (McGue et al., 2006). In the male sample, the correlation for the log-transformed early problem behavior score was 0.57 for MZ ($N = 183$) and 0.50 for DZ ($N = 96$). The comparable correlations in the female sample were 0.60 ($N = 212$) and 0.48 ($N = 105$), respectively. The minimal differences between the MZ and DZ correlations in the two samples suggest that heritable factors account for a relatively small proportion of variance in the early problem behavior index. Nonetheless, just like Prescott and Kendler (1999), we found that the relationship between the early problem behavior and disinhibitory psychopathology was primarily genetically mediated.

The association of early adolescent problem behavior with environmental risk

The seemingly anomalous finding that early problem behavior is only weakly heritable yet its relationship with disinhibitory psychopathology is primarily mediated by genetic factors may be a consequence of gene-environment interplay. That is, we hypothesize that early problem behavior is both a marker of inherited risk and contributes to a cascade of experiences that increase the likelihood that inherited risk manifests phenotypically as disinhibitory psychopathology. To evaluate this hypothesis, we recently explored the relationship of early adolescent problem behavior with contextual risk in a prospective study of 692 male twins (Keyes et al., 2006). Early adolescent problem behavior was operationalized similarly to operationalizations in our earlier studies by taking the number of the following behaviors the respondent reported having engaged in prior to age 15: tobacco use, alcohol use, marijuana use, other illicit drug use, police troubles, and sexual intercourse. Contextual risk was operationalized as the sum of the standardized scores from four measures of the adolescent's environment obtained at age 14: conflict with parents, academic engagement, affiliation with positive peer models (reverse scored), and affiliation with negative peer models. Consistent with our conceptualization of this being an index of environmental risk, the twin correlation for the contextual risk score was only slightly greater among MZ [$r = 0.66$, 95% confidence interval (CI) = 0.57, 0.74] than DZ ($r = 0.49$, 95% CI = 0.30, 0.64) twins.

Also as expected, early problem behavior and contextual risk were substantially correlated ($r = 0.53$, 95% CI = 0.46, 0.59), indicating that adolescents who engage in problem behaviors prior to age 15 are more likely to be exposed to high-risk environments. But how are each associated with psychopathology risk? Disinhibitory psychopathology at age 17–18 was measured by the number of DSM-IV symptoms of ALCDP, NICDP, DRGDP, and adult antisocial behavior (the latter corresponding to the adult symptoms of ASPD). This disinhibition composite was significantly correlated with both the early problem behavior ($r = 0.60$, 95% CI = 0.54, 0.65) and the contextual risk ($r = 0.51$, 95% CI = 0.44, 0.57) indexes. Importantly, both indexes contributed independently to the prediction of symptoms of

DOI: 10.1002/mpr

disinhibition (multiple $r = 0.65$, 95% CI = 0.60, 0.70). Multivariate biometric analysis of the twin data provided additional support for our dual pathway hypothesis. Replicating our earlier research (McGue et al., 2006) as well as the initial study by Prescott and Kendler (1999), we found that the relationship between early problem behaviors and disinhibitory psychopathology was predominantly genetically mediated. The relationship between contextual risk and disinhibitory psychopathology reflected, however, both genetic and environmental pathways.

Discussion

A series of large community-based epidemiological studies by our group, as well as similar research by others, leads to the following empirically-supported conclusions:

(1) In the US, alcohol use prior to age 15 is a robust predictor of risk of alcoholism in adulthood.
(2) Alcohol use prior to age 15 is also a robust predictor of other forms of adult disinhibitory psychopathology, even when its association with alcoholism is taken into account.
(3) Other forms of early adolescent problem behavior (e.g. smoking, troubles with police) are predictive of adult alcoholism and other disinhibitory psychopathology risk, even when their association with early alcohol use is taken into account.
(4) Early adolescent problem behavior is modestly heritable, yet its association with disinhibitory psychopathology is primarily genetically mediated.
(5) Early adolescent problem behavior increases the likelihood that adolescents are exposed to environments that increase risk of disinhibitory psychopathology.

In considering these conclusions, it is important to note that our research has focused primarily on adolescents and young adults. Consequently, our findings apply specifically to early-onset disorders. The nature of risk for late-onset disorders, where internalizing processes may come to play an increasingly important role (Dixit and Crum, 2000), may be quite different. In any case, these empirical conclusions are consistent with a dual pathway model of the association of early adolescent problem behavior with adult disinhibitory psychopathology (Figure 2). Specifically, we hypothesize that

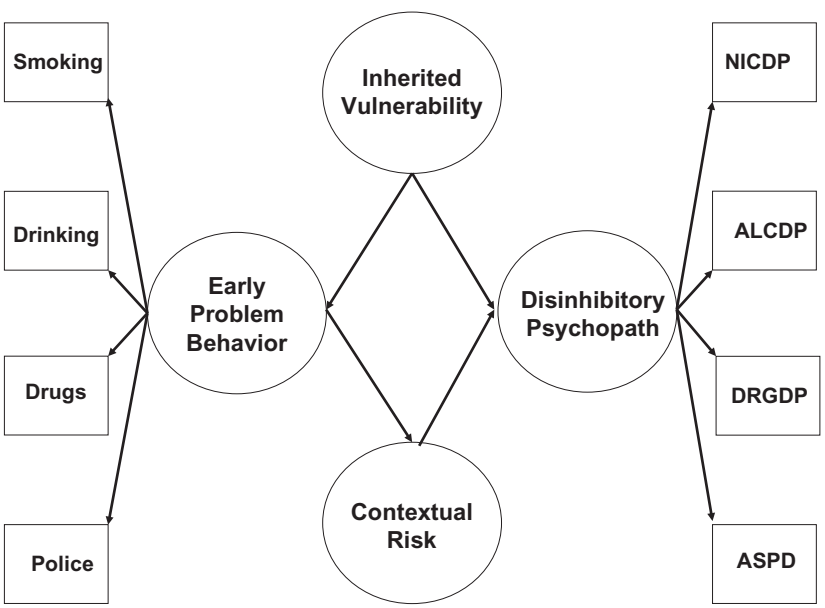

Figure 2. A heuristic dual-process model: We hypothesize that the association of early alcohol use with alcoholism risk narrowly, and of early problem behavior with disinhibitory psychopathology broadly reflects two processes. First, risk and outcome are both manifestations of an underlying inherited vulnerability to disinhibited psychopathology. Second, the early expression of problem behavior increases the likelihood an adolescent is exposed to a high-risk environment.

DOI: 10.1002/mpr

early adolescent problem behavior appears to be associated with disinhibitory psychopathology because both are manifestations of a general highly heritable vulnerability to disinhibitory psychopathology and because the expression of early adolescent problem behavior increases the likelihood that the developing adolescent is exposed to high-risk environments. Characterizing the prevention implications of the original observation that teens who start drinking early are at heightened risk for developing alcoholism will require additional evaluation of alternative models for this association.

Cross-national generalizability

A significant limitation of research relating early adolescent alcohol use with adult risk for alcoholism and related forms of disinhibitory psychopathology is that it has been based overwhelmingly on US samples. The age at which adolescents can legally purchase alcohol varies markedly across cultures (Ahlström and Österberg, 2004), providing a unique opportunity to explore the consequences of alternative policies governing teen drinking that has yet to be fully exploited. In the US, the legal age to purchase alcohol is 21, while in many European countries it is 18 or younger. Those who hypothesize that early adolescent drinking results in elevated rates of adult alcoholism because adolescent drinking causes neurological damage, for example, might expect alcoholism rates to be higher in cultures that condone adolescent drinking versus those that do not. Alternatively, those who subscribe to a common-inherited liability model might expect a diminished association of adolescent drinking with adult disinhibited psychopathology in cultures that allow adolescents to drink, because in these cultures teen drinking may be less an indicator of rebelliousness. Indeed, it is even possible that early teen drinking that is legal and part of normal family life may lower risk of adult alcoholism by providing some teens with responsible models of drinking behavior.

Unfortunately, comprehensive cross-cultural data relating teen drinking policies with adult rates of alcoholism does not exist, in part because epidemiological alcoholism data gathered using a common methodology from multiple cultures does not exist (Rehm et al., 2005). Although uniform cross-cultural data on problem drinking is definitely needed, we have undertaken a very preliminary analysis of the association of teen drinking policies with drinking behavior using per capita alcohol consumption as the outcome. We classified European countries in terms of the minimum age

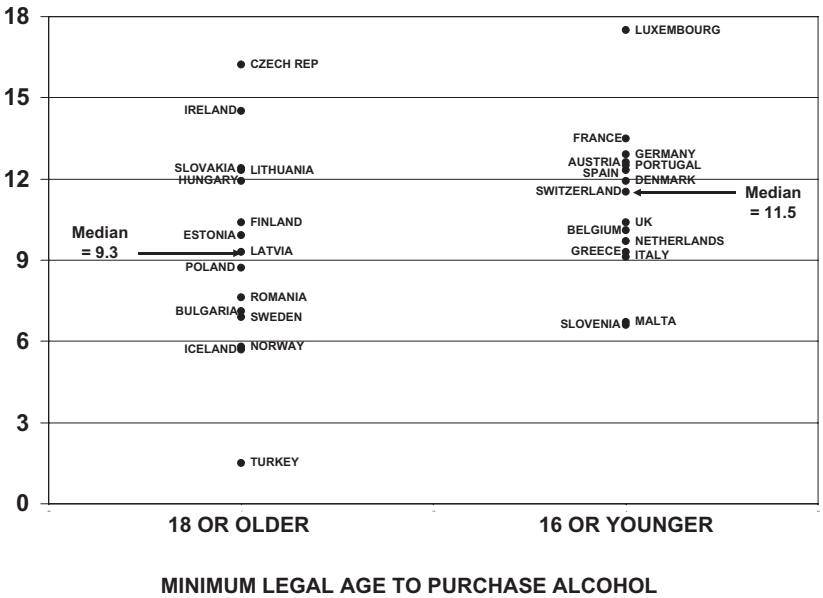

Figure 3. European teen drinking policies and consumption rates: Countries are classified according to the minimum age at which an adolescent can legally purchase some form of alcohol, which is related to 2001 annual per capita adult (age 15 or older) alcohol consumption in liters of pure alcohol based on World Health Organization (2001) data.

DOI: 10.1002/mpr

an individual could legally buy alcohol (either beer, wine or spirits) in some venue (off- or on-site) as age 16 or younger versus age 18 or older according to the information summarized by Ahlström and Österberg (2004). Adult per capita alcohol consumption was taken from data compiled by the World Health Organization (2001) and is reported as annual per capita (age 15 and older) alcohol consumption in liters of pure alcohol. As shown in Figure 3, younger minimum ages are associated with higher levels of alcohol consumption. Although this pattern of data is consistent with earlier drinking ages resulting in higher levels of adult consumption, there are many factors that severely limit our ability to unambiguously interpret the basis for the association. For example, the association could simply reflect that earlier minimum ages increase a country's consumption rates by increasing the pool of drinkers but do not otherwise affect the amount individual drinkers consume. Alternatively, cultures that permit teens to drink may do so, in part, because drinking is an accepted and central aspect of cultural life. In any case, the data summarized in Figure 3 suggest that a more comprehensive and rigorous analysis of the relationship of teen drinking policies and drinking behavior across cultures is warranted.

Acknowledgments

This paper is an extended and modified version of an oral presentation at the BMBF Conference "Understanding Addiction: Mediators and Moderators of Behavior Change Processes", Dresden, May 25–26, 2006.

This paper is based on a presentation given at the conference on, 'Understanding Addiction: Mediators and Moderators of Behaviour Change Process' held in Dresden, Germany in May 2006. The research described was supported in part by USPHS grants numbers AA09367 and DA05147.

Declaration of interest statement

The authors have no competing interests.

References

Ahlström SK, Österberg EL. International perspectives on adolescent and young adult drinking. Alcohol Res Health 2004; 28: 258–68.

Brown SA, Tapert SF. Adolescence and the trajectory of alcohol use: basic to clinical studies. Ann NY Acad Sci 2004; 1021: 234–44.

Christensen K, Petersen I, Skytthe A, Herskind AM, McGue M, Bingley P. Birth weight in twins and singletons and school performance in adolescence. Br Med J 2006; 333: 1095–7.

Dewit DJ, Adlaf EM, Offord DR, Ogborne AC. Age at first alcohol use: a risk factor for the development of alcohol disorders. Am J Psychiatry 2000; 157: 745–50.

Dixit AR, Crum RM. Prospective study of depression and the risk of heavy alcohol use in women. Am J Psychiatry 2000; 157(5): 751–8.

Elkins IJ, King SM, McGue M, Iacono WG. Personality traits and the development of nicotine, alcohol, and illicit drug disorders: prospective links from adolescence to young adulthood. J Abnormal Psychol 2006; 115(1): 26–39.

Grant BF, Dawson DA. Age at onset of alcohol use and its association with DSM-IV alcohol abuse and dependence: results from the National Longitudinal Alcohol Epidemiologic Survey. J Substance Abuse 1997; 9: 103–10.

Hingson RW, Heeren T, Winter MR. Age at drinking onset and alcohol dependence: Age at onset, duration, and severity. Arch Pediatr Adol Med 2006; 160: 739–46.

Iacono WG, McGue M. Association between P3 event-related brain potential amplitude and adolescent problem behavior. Psychophysiology 2006; 43(5): 465–9.

Iacono WG, Carlson SR, Malone SM, McGue M. P3 event-related potential amplitude and the risk for disinhibitory disorders in adolescent boys. Arch Gen Psychiatry 2002; 59(8): 750–7.

Iacono WG, Carlson SR, Taylor J, Elkins IJ, McGue M. Behavioral disinhibition and the development of substance use disorders: findings from the Minnesota Twin Family Study. Development and Psychopathology 1999; 11: 869–900.

Jessor R, Donovan JE, Costa FM. Beyond adolescence: problem behavior and young adult development. Cambridge: Cambridge University Press, 1991.

Johnson W, Krueger RF, Bouchard TJJ, McGue M. The personalities of twins: just ordinary folks. Twin Research 2002; 5: 125–31.

Kendler KS. Twin studies of psychiatric-illness – current status and future-directions. Arch Gen Psychiatry 1993; 50(11): 905–15.

Keyes MA, Iacono WG, McGue M. Early onset problem behavior, young adult psychopathology, and contextual risk. Twin Research and Human Genetics 2007; 10(1): 45–53.

Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M. Etiologic connections among substance dependence, antisocial behavior, and personality: modeling the externalizing spectrum. J Abnormal Psychol 2002; 111(3): 411–24.

McGue M, Iacono WG. The association of early adolescent problem behavior with adult psychopathology. Am J Psychiatry 2005; 162(6): 1118–24.

McGue M, Iacono WG, Krueger R. The association of early adolescent problem behavior and adult psychopathology: a multivariate behavioral genetic perspective. Behav Genet 2006; 36(4): 591–602.

McGue M, Elkins I, Walden B, Iacono WG. Perceptions of the parent-adolescent relationship: A longitudinal

DOI: 10.1002/mpr

investigation. Developmental Psychology 2005; 41: 971–84.

McGue M, Iacono WG, Legrand LN, Elkins I. The origins and consequences of age at first drink. I. Associations with substance-use disorders, disinhibitory behavior and psychopathology, and P3 amplitude. Alcoholism Clin Exp Res 2001a; 25: 1156–65.

McGue M, Iacono WG, Legrand LN, Elkins I. Origins and consequences of age at first drink. II. Familial risk and heritability. Alcoholism: Clin Exp Res 2001b; 25: 1166–73.

Prescott CA, Kendler KS. Age at first drink and risk for alcoholism: a noncausal association. Alcoholism Clin Exp Res 1999; 23: 101–7.

Rehm J, Room R, van den Brink W, Jacobi F. Alcohol use disorders in EU countries and Norway: an overview of the epidemiology. Eur Neuropsychopharmacology 2005; 15: 377–88.

Spear LP. The adolescent brain and the college drinker: biological basis of propensity to use and misuse alcohol. J Stud Alcohol Suppl 2002; 14: 71–81.

Tapert SF, Caldwell L, Burke C. Alcohol and the adolescent brain: human studies. Alcohol Res Health 2004; 28(4): 205–12.

World Health Organization. Adult per capita alcohol consumption, 2001. Available: http://www3.who.int/whosis/alcohol/ [14 December 2006].

Young SE, Stallings MC, Corley RP, Krauter KS, Hewitt JK. Genetic and environmental influences on behavioral disinhibition. Am J Med Genet (Neuropsychiatric Genetics) 2000; 96(5): 684–95.

Correspondence: Matt McGue, Department of Psychology, University of Minnesota, 75 East River Rd, Minneapolis, MN 55455, USA.
Telephone (+1) 612-625-8305
Fax (+1) 612-626-2079
Email: mcgue001@umn.edu

DOI: 10.1002/mpr