Nature of the Causal Relationship Between Academic Achievement and the Risk for Alcohol Use Disorder

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ABSTRACT. Objective: We evaluated the claim that interventions to improve academic achievement can reduce the risk for alcohol use disorder (AUD). **Method:** Using nationwide data for individuals born in Sweden from 1972 to 1981 (n = 930,182), we conducted instrumental variable and co-relative analyses of the association between academic achievement and AUD with a mean 21.4-year follow-up. Our instrument, used in the instrumental variable analyses, was month of birth. Co-relative analyses were conducted in cousins, full siblings, and monozygotic twins discordant for AUD, with observed results fitted to a genetic model. The academic achievement–AUD association was modeled in Cox regression. AUD was assessed using national medical, criminal, or pharmacy registries. **Results:** Later month of birth was significantly associated with poorer academic achievement. Lower standardized academic achievement had a strong relationship with the risk for subsequent AUD registration: hazard ratio (HR) [per *SD*] = 2.14 [2.11, 2.17]. Instrumental variable analysis produced a substantial but moderately attenuated association: HR = 1.52 [1.28, 1.80]. Controlling for modest associations between month of birth and parental education and AUD risk reduced the association to HR = 1.43 [1.20, 1.69]. Our genetic co-relative model fitted the observed data relatively well and estimated the academic achievement–AUD association in monozygotic twins discordant for academic achievement to equal an HR of 1.44 [1.35, 1.52]. Results were broadly similar when analyzed separately in males and females. **Conclusions:** Two distinct methods with different assumptions produced results suggesting that the association observed between academic achievement at age 16 and the risk for AUD into middle adulthood is partly causal, thereby providing support for interventions to improve academic achievement as a means to prevent later AUD risk. (*J. Stud. Alcohol Drugs, 81,* 446–453, 2020)

POOR ACADEMIC ACHIEVEMENT in adolescence, including low grades and school disengagement, is associated with an increased risk for alcohol use and misuse including alcohol use disorder (AUD; Hawkins et al., 1992; Henry et al., 2012; Huurre et al., 2010; Sale et al., 2003; Schulenberg et al., 1994; Spooner, 1999). Students who succeed academically tend to be committed to education—which facilitates a prosocial lifestyle and reduces the risk for a range of externalizing behaviors including problematic alcohol use (Hirschi, 1969). By contrast, those with poor grades and low school attachment are more prone to deviant behaviors including problematic alcohol consumption (Catalano & Hawkins, 1996; Dewey, 1999).

The association between academic achievement and the risk for AUD is of interest, given the prevalence and substantial adverse consequences of AUD. Worldwide in 2004, 3.8% of all deaths and 4.6% of the total burden of disease and injury were attributable to alcohol use (Rehm et al., 2009). In Sweden, AUD strongly predicts the need for social assistance, unemployment, early retirement, reduced income (Kendler et al., 2017), and premature mortality (Kendler et al., 2016), and a substantial proportion of these associations are likely to be causal.

If poor academic achievement contributes to the risk for AUD, then interventions that can improve academic achievement should reduce AUD risk and the associated medical and social problems. There is evidence that some individual and school-based interventions for children and adolescents can enhance academic achievement and reduce alcohol use

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(Dodge et al., 2015; Fletcher et al., 2009; Hawkins et al., 1999; Kellam et al., 2008; Lewis et al., 2012). However, with a few exceptions, such studies have had short follow-up periods and focus on alcohol use in the normative range rather than problematic use or dependency. Thus, the current literature does not provide strong evidence that improving academic achievement would have a long-term impact on the risk for AUD.

Furthermore, although the positive effects from intervention research suggest a causal relationship between academic achievement and the risk for AUD, several confounders (National Research Council & Institute of Medicine, 2000), not always accounted for in these studies, could complicate interpretation. For example, familial factors can increase the risk for both poor academic achievement and AUD (Barnard & McKeganey, 2004; Moss et al., 1995). In addition, reverse causation cannot be ruled out, as alcohol and substance use/misuse during the school years is associated with poor academic achievement (Allison et al., 2009; Cox et al., 2007; Hirschfield, 2018; Horwood et al., 2010).

To address the nature of the association between academic achievement and long-term risk for AUD, we used an instrumental variable analysis (Boef et al., 2016; Tchetgen Tchetgen et al., 2015), which requires the identification of an "instrument"—a variable that both affects the risk factor of interest (here academic achievement) and has no direct effect on the key outcome (here AUD).

Our instrument, chosen for its empirical association with academic achievement, is month of birth. Sweden, like many Western countries, has a cutoff date for school enrollment, so that nearly all students in any class were all born in the same year, therefore differing in age by up to 12 months. Studies in several European countries (Crawford et al., 2011; Eggert et al., 1994; Jürges & Schneider, 2011; Russell & Startup, 1986; Solli, 2017; Strom, 2014) show that, in such classes, presumably because of age-related cognitive maturation, the older students, on average, perform better academically than the younger ones.

In this study, we performed an instrumental variable analysis in which the predictor variable is academic achievement assessed at age 16 in a complete population cohort in Sweden, with greater than a 20-year mean follow-up period. The dependent variable is AUD, assessed from population-wide medical, criminal, and pharmacy registries. By comparing the raw association between academic achievement and AUD with that obtained from the instrumental variable analyses, we were able to estimate the proportion of the observed relationship between the two variables that is likely a result of causal processes. In addition, we also conducted a co-relative analysis (Kendler et al., 2015; Windle & Windle, 2014) of the association between academic achievement and AUD, an entirely different approach to causal inference in observational data. The co-relative analyses estimated, within monozygotic twins, the association between differences in academic achievement and differences in the risk for AUD, thereby controlling for all genetic effects and the impact of rearing in the same household and community (Kendler et al., 1993).

Method

We analyzed information on individuals from Swedish population-based registers with national coverage. These registers were linked, using each person's unique identification number replaced by a serial number to preserve confidentiality. We secured ethical approval for this study from the Regional Ethical Review Board of Lund University. (The authors assert that all procedures contributing to this work complied with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.)

The National School Registry contains academic achievement (i.e., grade point average) for all students in grade nine (usually age 16) from 1988 to 1997, attendance being mandatory for all children in Sweden. Students had an incentive to perform well, because those scoring well were likely to gain admission to desirable secondary schools. We standardized grade score for each year and sex, calling this variable *academic achievement.* From 1988 to 1997, scores were expressed on a 1 to 5 scale, students being assessed by a peer-referencing system. Under this system, grades had minimal inflation over time and were normally distributed. AUD was identified in the following Swedish medical registries: the Swedish Hospital Discharge Register (national coverage for 1987–2015 and partial coverage for 1969–1986); and the Outpatient Care Register (national coverage for 2001–2015). We also used a new Primary Care Registry (coverage for 1998–2016; exact coverage years vary slightly by county), which included individual-level information on diagnoses from visits to primary health care centers in 15 of Sweden's 21 counties. The following diagnostic codes from the *International Classification of Diseases* (ICD) were used: ICD 8: 571A, 291, 303, 980; ICD 9: V79B, 305A, 357F, 571A-D, 425F, 535D, 291, 303, 980; ICD 10: E244, G312, G621, G721, I426, K292, K70, K852, K860, O354, T51, F10.

AUD was also collected from the Crime Register (codes 3005 and 3201, which reflect crimes related to alcohol misuse driving under the influence convictions); the Suspicion Register (codes 0004 and 0005; only those individuals with at least two alcohol-related crimes or suspicion of crimes from both the Crime Register and the Suspicion Register were included); and the Prescribed Drug Register by the drugs disulfiram (Anatomical Therapeutic Chemical [ATC] Classification System N07BB01), acamprosate (N07BB03), and naltrexone (N07BB04).

The database included all individuals born in Sweden between 1972 and 1981 who had not died or emigrated before age 16 and were registered in the National School Register

the year they turned 15, 16, or 17 years old. The database also included their first year (if any) of registration of AUD. Individuals with AUD registered at age 16 or earlier were excluded from the analyses. Although the school year in which academic achievement was evaluated was normally made up of individuals who turned 16 between January 1 and December 31, approximately 3% of individuals were instead evaluated the year they turned 15 or 17. The month of birth of these individuals was not randomly distributed; more individuals born in January were registered with academic achievement the year they turned 15. More individuals born in December were registered with academic achievement the year they turned 17.

Furthermore, individuals registered with academic achievement at age 15 had a mean academic achievement of +0.74 *SD,* whereas individuals registered at age 17 had a mean academic achievement of -0.91 *SD.* As the association between month of birth and academic achievement was central to our further analyses, we modeled this nonrandomness. Using all individuals regardless of age at academic achievement, we fitted a regression model predicting academic achievement based on month of birth and age at registration. In the below analyses we used, for individuals registered the year they turned 15 or 17, their predicted value, and for all others their true value.

Analysis

First, we used a Cox proportional hazards model to investigate the risk of AUD as a function of low academic achievement, from the year of academic achievement registration until the end of follow-up (AUD registration, death, emigration, or December 31, 2015). The hazard ratio (HR) represents the increased risk for AUD for a 1-*SD* decrease in academic achievement. This is the crude association between academic achievement and AUD used for comparison.

We then used an instrumental variable approach to control for unmeasured confounding, using month of birth as an instrument. Below, we evaluate empirically the suitability of month of birth as an instrument. We used a two-stage-regression model adapted to a Cox-regression framework. The first step was a linear regression of academic achievement on month of birth. The predicted values were thereafter used in a Cox regression model as exposure variable (instead of the actual academic achievement value). As the unobserved and observed confounders should be equally distributed among the predicted values of academic achievement, the resulting HRs for academic achievement should be controlled for unmeasured and measured confounding. To obtain the 95% confidence intervals (CIs), we used nonparametric bootstrap with 1000 replications.

Methodological concerns with using month of birth as an instrument focus on potential differences between children born at different times of the year. Indeed, as seen in Appendix Table 1, our sample shows a small but significant decrease in AUD among parents with children born in March to May, $\chi^2(11) = 253.4$, $p < .0001$, and a significantly increased parental educational level for parents of children born in March, April, May, September, and October, $F(11, 929308) = 8.1$, $p < .0001$). (A supplemental appendix appears as an online-only addendum to this article on the journal's website.) As these results might violate a key instrumental variable assumption (month of birth has no direct effect on AUD risk), we conducted further analyses, adding controls for parental AUD and educational status in both the first and second stages of the instrumental variable analysis.

In the second analysis, we compared the results from the instrumental variable approach with those from a co-relative design. In the co-relative design, we examined whether the regression results (i.e., the crude association between academic achievement and AUD) reflect confounding by familial risk factors. From the Swedish Multi-Generation and Twin Registers, we identified all monozygotic (MZ) twin, full-sibling, and cousin pairs. Using stratified Cox proportional hazards models, with a separate stratum for each relative pair, we refitted the analysis (i.e., the risk of AUD as a function of academic achievement). The HR is then adjusted for a range of unmeasured genetic and environmental factors shared within the relative pair. MZ twins share 100% of their genes and a large part of environmental factors, suggesting that the HR for MZ twins is controlled for all possible confounding by genes and shared environment.

Last, we combined all four samples (i.e., population, MZ twins, full siblings, and cousins) into one data set in which we performed two analyses. The first allowed all parameters for each sample to be independent (i.e., similar to four separate analyses). In the second, we modeled the association between academic achievement and AUD with two parameters: one main effect and one as a linear function of the genetic resemblance (i.e., 0 for the population, .125 for the cousins, .5 for the siblings, and 1 for the MZ twins). The HR for the second parameter gives an indication of the size of the familial confounding. If the second model fitted the data well, as indexed by the AIC, we also obtained an improved estimation of the association among all relatives, but especially MZ twins, where the data were sparse. All analyses were also done separately by sex. All statistical analyses were performed using SAS Version 9.4 (SAS Institute, Inc., Cary, NC).

Results

Instrumental variable analysis

Included in our instrumental variable analyses were 930,182 subjects (476,190 males and 453,992 females) with academic achievement scores at a mean (*SD*) age of 16.0 (0.3) and a mean year of birth of 1976 (2.9). Their average

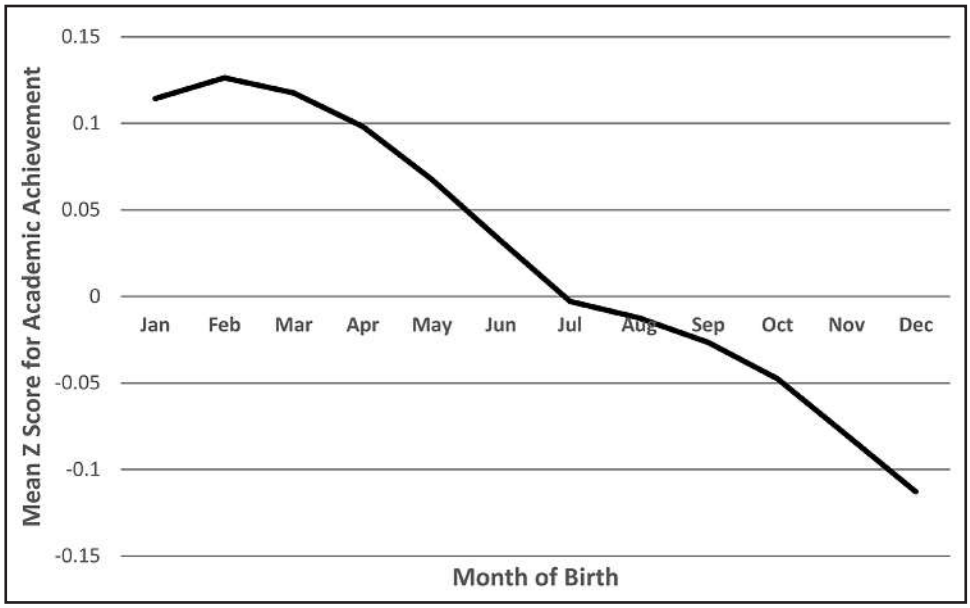

FIGURE 1. The association between month of birth and academic achievement assessed at age 16 in our sample of 930,182 Swedish adolescents. A linear model predicted that being 1 month younger reduced academic achievement in this sample by an average of 2.25% of an *SD.* As detailed in the Method section, these results included a statistical correction for the 3% of the sample who were assessed at ages 15 or 17. The *y* axis depicts *SD* units.

age at follow-up was 37.4 (4.8) (range: 16–43). Of these, 2.6% (23,774) were subsequently registered for AUD at a mean age of 27.4 (6.2). The mean (*SD*) of follow-up time was 21.4 years (4.9). The average academic achievement of those with a subsequent AUD registration was -0.74 (1.0) *SD* below the population mean.

The ability of an instrumental variable to provide information about causal influences requires that it should both meaningfully predict the independent variable (here low academic achievement) and show minimal association with the dependent variable (here AUD) except that mediated through the independent variable. Aside from an anomaly in January births, there is a clear monotonic association between our adjusted estimate of month of birth and academic achievement (Figure 1). Modeled as a linear effect, the regression coefficient [95% CI] was estimated at -0.0225 [-0.0231, -0.0219], so that being 1 month younger reduced the level of academic achievement by 2.3% of 1 *SD.*

The raw association between month of birth and AUD was modest but statistically significant in predicting that later months of birth predicted AUD risk: HR [95% CIs] = 1.009 [1.006, 1.013]. However, in accord with the assumptions of the instrumental variable model, when academic achievement was added to the model, the association between month of birth and AUD risk was reduced and changed sign, so that later months of birth were now slightly predictive of a lower AUD risk: HR = 0.995 [0.991, 0.999].

In our entire sample, lower academic achievement had a strong relationship with the risk for subsequent AUD registration: HR [per *SD*] = 2.14 [2.11, 2.17]. Our instrumental variable analysis produced an appreciably weaker but still substantial association HR = 1.52 [1.28, 1.80], consistent with partial familial confounding. To explore the sensitivity of our estimates to potential biases in our month of birth instrument, we reran the model controlling for parental educational attainment and AUD status. The predictive relationship between low academic achievement and risk for AUD was slightly further reduced in magnitude: HR = 1.43 [1.20, 1.69].

We then repeated these analyses in the male and female subjects in our cohort. As seen in Table 1, the results in males closely mirrored the findings in the entire sample, with HR estimates of 2.27 [2.23, 2.30] in the entire sample, 1.45 [1.18, 1.77] in the instrumental variable analysis with month of birth, and 1.36 [1.12, 1.66] when we corrected for potential biases. For females, the overall association was slightly weaker than that seen in men (2.00 [1.94, 2.03]), but the instrumental variable results both without and with correction were less attenuated (but less precisely known) than in males: 1.81 [1.32, 2.45] and 1.67 [1.23, 2.28], respectively.

Co-relative analysis

The sample sizes of subjects in these analyses are seen in Table 2, which also provides the observed HRs between low academic achievement and the risk for AUD in the entire sample and then in first cousins, full siblings, and MZ twin pairs discordant for their level of academic achievement for our entire sample and then for males and females separately. In

Table 1. Instrumental variable analysis of the association between academic achievement and the risk for alcohol use disorder (AUD)

Variable	AA (crude) HR [95% CI]	Instrumental variable HR [95% CI]	Instrumental variable controlling for parental educational and risk for AUD HR [95% CI]	MZ twins expected from co-relative design HR [95% CI]
AUD	2.14 [2.11, 2.17]	1.52 [1.28, 1.80]	1.43 [1.20, 1.69]	1.44 [1.35, 1.52]
AUD—males	2.27 [2.23, 2.30]	1.45 [1.18, 1.77]	1.36 [1.12, 1.66]	1.66 [1.49, 1.84]
AUD—females	2.00 [1.94, 2.03]	1.81 [1.32, 2.45]	1.67 [1.23, 2.28]	1.30 [1.12, 1.51]

Notes: AA = academic achievement; HR = hazard ratio; CI = confidence interval; MZ = monozygotic.

the three well-powered samples (population, cousins, and full siblings), the HRs were progressively lower for more closely related pairs of relatives for the entire cohort as well as for the males and females separately. However, the observed associations within monozygotic twin pairs were quite variable and were estimated very imprecisely because of a small sample of informative pairs. In the right side of this table, we applied our genetic model to these four estimates. The predicted HRs were quite close to those observed for the population, cousins, and full siblings but not for the MZ twins where they were known more precisely, and the results were now in line with the expectations from the other groups. As measured by the Akaike information criterion (Akaike, 1987), the genetic model fit better than the observed estimates for females and only slightly worse for males and the entire sample. The key estimates for the academic achievement–AUD association in discordant MZ pairs was 1.44 [1.35, 1.52] for the entire sample and 1.66 [1.49, 1.84] and 1.30 [1.12, 1.51] in males and females, respectively.

In Table 1, we compared the estimated HRs from MZ twins with the results obtained from the instrumental variable analyses with month of birth. For the entire sample, the two estimates were quite similar. For males and for females, the co-relative estimate was, respectively, somewhat higher and lower than that seen from the instrumental variable analyses, although in both cases the confidence intervals widely overlapped.

Discussion

The goal of this study was to clarify the causal nature of the association between academic achievement in adolescence and the risk for subsequent AUD using two methodologically distinct methods of causal inference applied to observational data (i.e., instrumental variable and co-relative analyses). Examining the entire sample, both methods, despite having quite divergent underlying assumptions, produced convergent evidence that a considerable proportion of the observed association between low academic achievement and subsequent risk for AUD is likely causal. Both methods estimated that an increase of 1 *SD* of academic achievement at age 16 should decrease long-term risk for AUD by approximately 45%. When analyzed separately by sex, the overall association of low academic achievement with AUD was clearly stronger in males than in females. Although the results for our instrumental variable and co-relative analyses differed somewhat between the sexes, the overall conclusions from these two sets of analyses suggested that a similar degree of the observed academic achievement–AUD association in the two sexes was likely causal.

Finding that academic achievement is related to AUD is congruent with several sociological theories. According to the social control theory (Hirschi, 1969) and the Social Development Model (Cranford, 2014), students who perform well academically will likely develop positive attachments to school and commitment to education, which in turn facilitates commitment to prosocial lifestyles. This positive orientation reduces deviant behaviors and the risk for AUD. The results also support efforts to improve the academic achievement of adolescent students to prevent risk for AUD. This is important; although treatment of AUD can be effective, preventing it is more cost-effective (National Research Council & Institute of Medicine, 2009; U.S. Department of Health and Human Services, 2016).

Schools are an optimal setting for delivering such programs, given their natural focus on academic achievement and their ability to reach most adolescents. Many school-based programs have been demonstrated to improve academic achievement (e.g., see https://ies.ed.gov/ncee/wwc/FWW), and a few have examined and found effects on alcohol use or misuse (Dodge et al., 2015; Hawkins et al., 1999; Kellam et al., 2008; Lewis et al., 2012). Such interventions may provide individual students with remedial education (e.g., tutoring), or they may be more comprehensive, changing school policies or climate to provide more positive learning environments, coaching teachers in classroom management or cooperative learning strategies, and/or promoting parental involvement in school.

As an example, the Raising Healthy Children intervention offers prevention programming for students, teachers, and parents in elementary and secondary schools. An evaluation found that students who received such services in Grades

TABLE 2. Results of the co-relative analysis of the association between academic achievement and alcohol use disorder

Sample	Sample size, *n*	Alcohol use disorder	
		Observed HR [95% CI]	Predicted HR [95% CI]
Population	929,437	2.14 [2.11, 2.17]	2.13 [2.10, 2.15]
Cousins	523,245	1.97 [1.93, 2.02]	2.03 [2.00, 2.05]
Full siblings	305,337	1.77 [1.72, 1.83]	1.75 [1.70, 1.80]
Monozygotic twins	2,193	1.95 [0.91, 4.16]	1.44 [1.35, 1.52]
AIC		673,925.97	673,927.52
Males			
Population	475,519	2.27 [2.23, 2.30]	2.26 [2.22, 2.29]
Cousins	136,971	2.08 [2.00, 2.15]	2.17 [2.14, 2.21]
Full siblings	80,189	1.99 [1.88, 2.11]	1.93 [1.84, 2.03]
Monozygotic twins	981	1.22 [0.52, 2.87]	1.66 [1.49, 1.84]
AIC		434,559.46	434,560.78
Females			
Population	453,918	2.00 [1.94, 2.03]	2.00 [1.95, 2.04]
Cousins	125,251	1.90 [1.81, 2.01]	1.89 [1.85, 1.94]
Full siblings	73,071	1.60 [1.48, 1.72]	1.61 [1.50, 1.73]
Monozygotic twins	1,212	4.09 [0.64, 26.1]	1.30 [1.12, 1.51]
AIC		186,374.07	186,371.89

Notes: HR = hazard ratio; CI = confidence interval; AIC = Akaike information criterion.

1–6 in Seattle, WA, reported significantly less frequent drinking at Grade 10 compared with those in the control condition (Hawkins et al., 1999). Furthermore, the program is cost-beneficial (Washington State Institute for Public Policy, 2017). Increasing the use of these types of programs has a potential to decrease health disparities in the society and are needed to produce significant increases in academic achievement and reductions in AUD. However, most are designed for elementary and middle schools, and our results suggest that more interventions should be developed and tested for high school students.

Income inequality is lower in Sweden than in many other countries of the world including the United States and United Kingdom (Aaberge, 1999). How might our findings extrapolate to countries with greater social inequality and poorer access to social services? Although this question can be answered definitively only by additional research, we would speculate that the effects of academic achievement on AUD rates might be even stronger. In such countries, those who perform poorly in school and adopt more antisocial lifestyles would have access to fewer high-quality jobs and remedial and treatment opportunities than are provided in Sweden.

Causal inference from observational data such as those examined here should always be considered tentative. However, confidence in results can be increased through multiple inferential methods (Kendler & Gardner, 2010), recently termed *triangulation* (Munafò & Davey Smith, 2018). In fact, the larger the difference in methods, the stronger the resulting confidence can be. Our two methods are quite divergent in both implementation and theoretical assumptions.

Our findings can be usefully compared with similar analyses recently completed examining the outcome of drug abuse in Sweden (Kendler et al., 2018). The population association with low academic achievement was slightly stronger for drug abuse than for AUD (2.33 [2.30, 2.35] vs. 2.14 [2.11, 2.17]), as was the estimate for causal effects from instrumental variable analyses: drug abuse (2.04 [1.75, 2.33]) and AUD (1.43 [1.20, 1.69]). A similar pattern was seen from the estimates from MZ twins from the co-relative analyses: drug abuse (1.79 [1.64, 1.92]) and AUD (1.44 [1.35, 1.52]). These results suggest that not only is the association with academic achievement for AUD somewhat weaker than that seen for drug abuse at the population level but also a somewhat lower proportion of that association is likely causal for AUD, compared with what was found for drug abuse.

Limitations

These results should be interpreted in the context of four potentially significant methodological limitations. First, AUD was assessed using information entirely from Swedish registries. Although such administrative data have important advantages (e.g., no refusals or reporting biases), they cannot be expected to identify cases in the same manner as interview-based assessments. Our subjects with AUD are on average probably more severe than those meeting *Diagnostic and Statistical Manual for Mental Disorders, Fifth Edition* (DSM-5; American Psychiatric Association, 2013), criteria for AUD at interview, although the lifetime prevalence of alcohol dependence in nearby Norway is only moderately higher than the estimates obtained in Sweden (Kringlen et al., 2001). It is also possible that a bias might arise, because individuals with AUD and a high academic achievement are less likely to be detected by the medical or criminal registries.

Second, our instrument—month of birth—had some potential limitations. Three percent of the sample were not tested at age 16. We statistically corrected for this anomaly. Furthermore, month of birth was weakly predicted by parental education and the risk for AUD. The strength of the association between academic achievement and AUD declined modestly when we included these variables as covariates.

Third, our co-relative design does not control for environmental confounders specific to individuals that could affect both academic achievement and AUD risk. Fourth, although instrumental variable analyses should protect us against the impact of reverse causation (i.e., prior heavy alcohol use predicting both poor academic achievement and the risk for AUD) (Cingolani & de Crombrugghe, 2012; Yu, 2018), we evaluated this bias by re-analyzing our data including a 2-year buffer period in which we censored AUD registrations soon after the assessment of academic achievement, as early heavy alcohol use should predict early AUD registration. The estimated academic achievement–AUD HRs decreased very slightly, both from the instrumental variable (from 1.52 to 1.46 [1.22, 1.75] and the co-relative analyses (from 1.44 to 1.42 [1.34, 1.51], suggesting that any appreciable bias from reverse causation was unlikely.

Last, we did not seek to elucidate possible mediational paths from late month of birth in each school year to AUD. One particularly plausible mediator would be ADHD, which has been shown in several studies to occur more frequently in the younger children in each school grade (Karlstad, 2017; Whitely, 2017) and increases the risk for AUD (Charach, 2011).

Conclusions

Two analytical methods based on divergent assumptions—instrumental variable and co-relative analyses—both suggest that a meaningful proportion of the association between academic achievement at age 16 and subsequent risk for AUD over a follow-up period of 21 years was likely causal. When analyzed separately by sex, we saw that the overall association of low academic achievement with AUD was stronger in males than in females. Although the results for our two specific methods differed somewhat between the sexes, the overall results suggest that a similar degree of the association in the two sexes was likely causal. Consistent with intervention studies, most of which had much shorter follow-ups and softer outcomes (e.g., substance use rather than dependence), our findings suggest that programs that improve academic achievement in adolescence should result in meaningful reductions in the subsequent risk for AUD.

Conflict-of-Interest Statement

The authors have no conflicts of interest to declare.

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