Changes in Externalizing Behaviors After Children First Have an Alcoholic Drink and First Drink Heavily

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ABSTRACT. Objective: Proximal changes in externalizing behaviors before and after children and early adolescents have their first alcoholic drink and first heavy drinking episode are examined using intergenerational, prospective data from the ongoing U.K. national Millennium Cohort Study (10,529 child–parent pairs followed over 35,406 occasions). **Method:** We examined how within-person changes in externalizing behaviors (based on parental reports on the Strengths and Difficulties scale when children were modal ages 5, 7, 11, and 14 years) follow children's age at first alcoholic drink (AFD) and age at first heavy drinking (AFHD), based on confidential child self-reports at ages 11 and 14 years. Analyses controlled for child age, time-varying parent-level confounders (parental education and alcohol abstinence), and time-stable

selection factors. **Results:** Estimates from fixed-effects Poisson models revealed a 5% increase in the expected count of externalizing behaviors after children have their first alcoholic drink (incidence rate ratio [IRR] = 1.05, 95% CI [1.03, 1.07]), and a 13% increase after first drinking heavily (IRR = 1.13, 95% CI [1.09, 1.18]), independent of key time-varying and all time-stable individual differences. **Conclusions:** Early AFHD and unobserved time-stable selection factors partially explain relationships between early drinking and problem behaviors, but early AFD continues to be a significant predictor of externalizing behavior. Although prevention efforts should continue to discourage heavy drinking in childhood and early adolescence, the results suggest that both AFHD and AFD should be delayed. (*J. Stud. Alcohol Drugs*, 80, 472–479, 2019)

EARLY AGE AT first alcoholic drink (AFD) predicts a greater likelihood of tobacco, marijuana, and other drug use; alcohol and drug dependence; drinking and driving; motor vehicle accidents; other injuries; academic and employment problems; victimization; aggression; and criminal and violent behavior (Agrawal et al., 2006; Blonigen et al., 2015; Buchmann et al., 2009; Clark et al., 2005; Colder et al., 2018; Dawson et al., 2008; Grant & Dawson, 1997; Guttmannova et al., 2011; 2012; Hingson et al., 2006; Hingson & Zha, 2009; Odgers et al., 2008; Pedersen & Skrandal, 1998; Pitkanen et al., 2005; Swahn et al., 2008; Warner & White, 2003; Windle et al., 2009). Scholars have questioned whether these links of early first drink with problematic outcomes in adolescence through young adulthood are causal evidence of “consequences” or simply reflect pre-existing differences between children (Brown et al., 2008; Kuntsche et al., 2016; Maimaris & McCambridge, 2014; Prescott & Kendler, 1999; Ystrom et al., 2014). Resolving this issue is urgent (Hingson et al.,

2004), as many prevention efforts aim to delay alcohol onset as a primary goal. Critiques center around two alternative hypotheses: (a) *selection*, or third variable causes, may underlie both early AFD and later health-risk behaviors and poor health (Kuntsche et al., 2016; Zucker et al., 2008), and (b) *heavy drinking in early adolescence* may be the true cause of poor outcomes, not age at initiation of any alcohol use (Kuntsche et al., 2013). Indeed, most theoretical explanations for detrimental effects assume considerable alcohol exposure beyond just one drink (Baumrind & Moselle, 1985; Newcomb & Bentler, 1988).

We use nationally representative, intergenerational data from the ongoing Millennium Cohort Study (MCS), a prospective study of U.K. children and their parents, to examine hypotheses about selection and age at first heavy drinking (AFHD) as alternative explanations for well-documented negative health and social correlates of early AFD. The MCS design overcomes pivotal limitations of prior population-based work where data collection typically began, at the earliest, in early adolescence (Donovan, 2007; Zucker et al., 2008). The large national sample provides a sufficient number of respondents who tried more than a few sips of alcohol at a very young age (Maggs et al., 2019). Furthermore, the prospective design analyzed with fixed-effects models (Wooldridge, 2002) controls for *all* time-stable sources of unobserved heterogeneity when assessing changes in externalizing behaviors after respondents first drink alcohol and first drink heavily. The ability to assess whether observed proximal links between alcohol initiation ages and problem behavior are consistent with causal effects or reflect early-life or ongoing vulnerabilities is unique.

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Dominant view suggests early drinking is harmful

A variety of causal mechanisms could explain deleterious effects of early AFD: (a) neurotoxic effects of alcohol, leading to neuropsychological and behavioral inhibition deficits, (b) changed expectancies regarding benefits and costs of alcohol and other substances, (c) increased interest in older friends and more adult-like roles and leisure activities, (d) selection of deviant peer groups and substance-using friends, and (e) transition to riskier drinking or other problem behaviors (Brown et al., 2000; Newcomb & Bentler, 1988; Windle et al., 2009; Zucker, 2008). These plausible mechanisms, combined with extensive evidence linking early AFD with heightened risk for myriad short- and longer-term health risk behaviors (Chassin et al., 2016), strongly support the argument that early drinking may be detrimental to healthy development. Therefore, numerous individual, family, school, and community-level prevention efforts and policy initiatives aim to delay onset of alcohol use (U.S. Department of Health and Human Services, 2013). However, key scholars have questioned whether purported harms of AFD might (a) be spurious or (b) reflect effects of *heavy* early adolescent drinking.

Critique 1: The purported developmental harms of AFD are the result of selection

Research is equivocal whether observed effects of early AFD are causal or are instead spurious reflections of early-life confounders that predict precocious timing of AFD and negative youth adjustment (Kuntsche et al., 2016; Maimaris & McCambridge, 2014). In this view, early AFD may exert no causal effect on subsequent problem behaviors but rather simply be a marker of genetic and early-life family risk factors, such as neural deficits, difficult temperament, socioeconomic adversity, and parental heavy drinking. Unfortunately, little research has explored the consequences of drinking on problematic outcomes *during childhood and adolescence* but often assesses problematic outcomes later in life. This exacerbates the problem, making it more difficult to assess whether these effects are causal or spurious.

Although early initiation is a frequently studied risk factor for deleterious adult outcomes, a systematic narrative review concluded that rigorous evidence for causal effects was meager (Maimaris & McCambridge, 2014; see also Kuntsche et al., 2016; Prescott & Kendler, 1999). Only five cohort studies with adequate controls for background risk factors were identified, and only one revealed long-term effects of early AFD on adult alcohol problems (Pedersen & Skrondal, 1998). Furthermore, in most studies, individuals age 18 years and older were asked to recollect their initiation age retrospectively (Donovan, 2004; 2007), increasing potential forward telescoping bias (i.e., reporting ever-older ages of initiation at later waves) and denial of drinking at follow-up

despite previous reports of AFD (Bailey et al., 1992; Parra et al., 2003; Sartor et al., 2011).

Moreover, externalizing behaviors in childhood increase the likelihood of early AFD (Buchmann et al., 2009; Maggs et al., 2019; McGue et al., 2001; Odgers et al., 2008). Some research shows that AFD effects on adult drinking problems disappear when controls for externalizing behaviors are included (e.g., King & Chassin, 2007; Rossow & Kuntsche, 2013; Warner & White, 2003), but little research tests whether early AFD increases the proximal risk of problem behaviors with controls for early-life confounders. Most recently, Blonigen and colleagues (2015) used a person-centered design to show heightened rates of adolescent aggression following early AFD; however, they did not control for unobserved early-life selection factors that may be spuriously related to both AFD and problem behaviors.

Critique 2. AFHD, and not AFD, is the true cause of short- and longer-term harms

An alternative explanation argues that drunkenness or intoxication in childhood or early adolescence, rather than early light drinking, accounts for apparent AFD harms (Asbridge et al., 2016; Kuntsche et al., 2013; Warner & White, 2003). Potential consequences of adolescent heavy drinking include injury, alcohol-related accidents and deaths, alcohol poisoning, suicide, homicide, and physical and sexual assault (Hingson et al., 2004; U.S. Department of Health and Human Services, 2013). Even if catastrophic injury is avoided, a single heavy drinking episode can lead to neurocognitive and immune functioning decrements (Bala et al., 2014; Zeigler et al., 2005). As such, it is feasible that a primary causal mechanism by which early AFD could lead to later-life health and social problems is via increased likelihood of heavy drinking in adolescence (Dawson et al., 2008).

Little prospective research has tested whether AFHD is a stronger predictor of harm than AFD. International cross-sectional studies suggest that AFHD is a stronger predictor than AFD of smoking, marijuana use, injuries, fights, low academic performance, and problematic alcohol use during adolescence (Asbridge et al., 2016; Kuntsche et al., 2013). In addition, in the long term, Warner and White (2003) found that feeling drunk at first use predicted later life drinking problems, but early onset drinking did not. Because initiation of any use is required for initiation of heavy drinking, it is necessary to distinguish their links with problem behaviors.

Current study

To evaluate the validity of these critiques, we first describe patterns of AFD, AFHD, and externalizing behaviors through childhood and early adolescence. To minimize recall and shared-method bias, we rely on confidential child reports (assessed at modal ages 11 and 14 years) of the AFD (more

than a few sips) and AFHD (five or more drinks), as well as prospective parental reports of children's externalizing behaviors on four occasions through age 14 years. The repeated survey design assesses age at initiation relatively close in time to both alcohol transitions and permits identification of links between changes in alcohol use and changes in key time-varying outcomes. No other nationally representative contemporary U.K. study has equivalently detailed data from infancy to early adolescence.

Furthermore, because data on earlier contexts and characteristics are rarely available in large representative studies, it is difficult to disentangle alcohol effects from developmental/social contexts or to rule out selection effects (King & Chassin, 2007; Windle et al., 2009). Modeling AFD and AFHD from age 5 years through 14–15 years using fixed-effects regressions permits the estimation of how AFD and AFHD influence changes in externalizing behaviors while controlling for key theoretically driven early-life factors (e.g., socioeconomic adversity, difficult infant temperament). Furthermore, the within-person design avoids arbitrary dichotomized age thresholds for “early” drinking (which may vary within families and communities). By comparing children with themselves over time (via fixed-effects models), we assessed whether externalizing behaviors increased after the child first has a drink and first drinks heavily, controlling for age, time-stable unobserved heterogeneity, and other time-varying confounders.

Method

Participants

The MCS follows a nationally representative stratified random cluster sample of children born between September 2000 and January 2002. Eligible infants were living in England, Northern Ireland, Scotland, and Wales at 9 months of age, based on records compiled by Health Visitors lists and the Child Benefit register, both universal programs (Plewis, 2007). The MCS oversampled electoral wards outside England, as well as wards in England with high levels of child poverty and high concentrations of Indian, Pakistani, Bangladeshi, Black African, and Black Caribbean families (based on census reports). At Wave 1, parents from 18,552 families participated in structured in-person interviews, representing 91% of eligible children. An additional 1,389 families were added at Wave 2 (at child's modal age of 3 years). New families included children who would have been eligible in Wave 1 but had registered late for universal Child Benefit (Ketende, 2008).

Professional interviewers conducted in-home assessments with MCS children and parents in five subsequent waves (when children were approximately ages 3, 5, 7, 11, and 14 years). MCS children also completed confidential surveys at ages 11 and 14. Approximately 76% of eligible children who

had not emigrated, permanently withdrawn, or died completed the survey at age 14. Mostafa and Ploubidis (2017) found that attrition by age 14 was more likely among boys, Whites, and children residing in a single-parent household and with parents of lower social class or lower levels of educational attainment.

In this study, we used data from 10,529 MCS children to create a person-wave data set that included 35,406 observations (an average of 3.4 waves for each child). Although most children were observed at ages 5, 7, 11, and 14 years (approximately 75% of respondents), some variation in child age at assessment occurred (e.g., 3% of person-period waves occurred at age 6; 7% at age 10 years; and 5% at age 13 years). Less than 1% of MCS children completed interviews when they were ages 8, 12, and 15; therefore, some age categories were collapsed (e.g., ages 14 and 15). The large sample, combined with the age variation in the timing of interviews at each wave, allowed us to match the timing of AFD and AFHD with changes in problem behaviors during childhood and early adolescence.

Time-varying measures

Externalizing behaviors. Parents used the Strengths and Difficulties questionnaire (R. Goodman, 1997, 2001; www.sdqinfo.com) at four waves from modal age 5–14 years to describe their child's externalizing behaviors. This measure comprises 10 items (A. Goodman et al., 2010) indicating the extent to which the child fights with or bullies other children, often has temper tantrums, is easily distracted, does not stop and think before acting, etc. Responses range from 0 (*not true*) to 2 (*certainly true*). Items were summed within waves ($M_{\text{across waves}} = 4.74$, $SD = 3.54$; Cronbach's α ranged from .78 to .81). For most children in the MCS, externalizing behaviors generally drop sharply after age 3 and then remain relatively stable (Staff et al., 2015). Table 1 shows descriptive statistics by age and percentage of the analytic sample observed at each age.

Drinking behaviors. Age at first drink (AFD) was based on child reports indicating the age they first had an alcoholic drink (more than a few sips) (Maggs et al., 2019). To increase reliability and validity, best practices of simple language and self-administered surveys were used (Brenner et al., 2003). To minimize recall bias, we relied on AFD responses from age 11 years if children indicated previously consuming alcohol; otherwise, we relied on AFD responses from age 14 years. Children also indicated the age they first had five or more alcoholic drinks at a time. A *drink* was defined as half a pint of lager, beer, or cider, one alcopop, a small glass of wine, or a measure of distilled spirits (Maggs et al., 2015). Similar to AFD, AFHD episode is based on self-reports from age 11 if already experienced; otherwise, we relied on AFHD responses from age 14. Table 1 shows percentages of children who previously had a first drink or

TABLE 1. Descriptive statistics, by age

Age	Child problem behaviors ^a	Child alcohol use ^b		Parent relationship to child ^c			Parental educational level ^c			Parental drinking ^c	
	Externalizing behaviors	First drink	First heavy drinking episode	Mother	Father	Other	Low (no degree)	Middle (NVQ 1 to 3)	High (NVQ 4 or 5)	Not drinking alcohol	% of total occasions ^d
5	4.50	0.8%	0.03%	97.5%	2.4%	0.1%	9.9%	49.4%	40.7%	19.8%	21.9%
6	4.55	1.0%	0.2%	97.8%	2.0%	0.1%	8.5%	49.6%	42.0%	18.0%	3.8%
7–8	4.44	2.0%	0.1%	97.0%	2.7%	0.3%	9.2%	47.9%	42.9%	19.9%	21.6%
10	4.41	11.7%	0.4%	96.4%	3.3%	0.3%	8.7%	45.1%	46.2%	24.4%	8.9%
11–12	4.17	15.3%	0.7%	95.8%	3.6%	0.7%	8.8%	44.9%	46.3%	22.9%	17.3%
13	4.32	37.8%	6.0%	94.6%	4.8%	0.7%	8.8%	43.6%	47.6%	24.0%	6.5%
14–15	4.14	48.4%	11.1%	93.2%	5.8%	1.0%	9.6%	43.8%	46.6%	21.7%	20.0%

Notes: $n = 10,529$ (35,406 occasions). NVQ = National Vocational Qualification. ^aMean count of externalizing behaviors by child age. ^bCumulative percentage of children who had a first drink or first heavy drinking episode by child age. ^cPercentages of parental relationship to child, parental educational level, and parental drinking, by child age. ^dPercentage of total observations by child age.

first heavy drinking episode by each age. By ages 14 to 15, just less than half of MCS children had consumed a drink of alcohol, and approximately 11% had consumed five or more drinks at a time.

Although good reliability and validity of self-reported alcohol use among elementary school-age children (Zucker et al., 1995) and adolescents (Brenner et al., 2003) has been demonstrated, test–retest reliability of AFD over longer periods has been questioned (Kuntsche et al., 2016). In unlisted analyses, we compared child AFD responses at modal ages 11 and 14 years. Of the approximately one half of children who reported drinking alcohol at either wave, about 76% had consistent reports. Of the 24% of children whose AFD reports were inconsistent, 11.7% reported an older AFD at age 14 compared with age 11 (i.e., forward telescopers) or reported drinking at age 11 but never having had a drink at age 14 (7.6%). The remaining 4.6% reported a younger AFD at age 14 compared with age 11. Importantly, early drinkers would have been missed if we relied solely on AFD reports at the later wave, and many respondents would be categorized with an older AFD.

Parent-level confounders. In all regressions, we included time-varying measures of the parental respondent's relationship to child, educational level, and alcohol use. Dummy variables contrasted whether the parental respondent at each wave was the mother (>96% of occasions), father, or other primary caregiver. Educational qualifications were based on Makepeace et al.'s (2003) categorization of National Vocational Qualification (NVQ) levels. At each wave, we distinguished parents who had attained postsecondary degrees (NVQ4, NVQ5) versus lower qualifications (NVQ1 through NVQ3) or no educational diploma. On average across waves, approximately 43% of parents had postsecondary degrees, 48% had lower level credentials, and 9% had none. Last, parental respondent alcohol use distinguished between waves respondents reported "never" consuming alcohol (~21% of occasions) to waves they reported at least some alcohol use. Note that the time-varying educational qualification and

parental alcohol use measures are almost exclusively mother reports.

Analytical strategy

Fixed effects models assessed individual changes in externalizing behaviors after children consumed their first alcoholic drink and first drank heavily. In general, fixed effects models eliminate bias from unobserved characteristics by first subtracting each child's over-time mean values on both the outcome (i.e., externalizing behaviors) and key predictor variables (i.e., AFD, AFHD) from the observed values at each wave (Wooldridge, 2002). These within-person deviations in externalizing behaviors are then regressed on AFD and AFHD, with age and other time-varying confounders being controlled for. Analyses exclude children (6%) with no change in externalizing behaviors. Because of clustered-count outcome data, all models used the command "xtpoisson, fe" in STATA Version 15 (StataCorp LP, College Station, TX), with robust standard errors to adjust for within-individual correlation.

A small amount of data was missing in some waves regarding the parent's relationship to child (0.1%), educational level (3% of waves), and alcohol use (1%). To retain these cases, we imputed 20 data sets using chained regressions (using the "MI" command in STATA 15) and used the "mi estimate" command to combine results across these data sets.

Results

In Table 2, we present incidence rate ratios (IRRs) and 95% confidence intervals (CIs) from fixed-effects Poisson models predicting within-person changes in externalizing behaviors after children had their first alcoholic drink (Model 1) and first drank heavily (Model 2). When we controlled for child's age at assessment, parent-level confounders, and all time-stable sources of unobserved heterogeneity, the expected count of externalizing behaviors increased by 7% (IRR = 1.07, 95%

TABLE 2. Incidence rate ratios (IRRs) and 95% confidence intervals (CIs) from fixed effects Poisson regression models predicting changes in externalizing behavior by age at first drink and age at first heavy drinking episode

Time-varying predictors	Outcome: Changes in externalizing behavior from ages 5–15					
	Model 1		Model 2		Model 3	
	IRR	[95% CI]	IRR	[95% CI]	IRR	[95% CI]
Child alcohol use						
Had first drink	1.07***	[1.05, 1.09]			1.05***	[1.03, 1.07]
Had first heavy drinking episode			1.16***	[1.11, 1.20]	1.13***	[1.09, 1.18]
Age (vs. 11–12)						
5	1.09***	[1.07, 1.11]	1.08***	[1.06, 1.10]	1.09***	[1.07, 1.11]
6	1.06**	[1.02, 1.10]	1.05**	[1.02, 1.09]	1.06**	[1.02, 1.10]
7–8	1.08***	[1.06, 1.09]	1.07***	[1.05, 1.08]	1.07***	[1.06, 1.09]
10	1.02	[0.99, 1.04]	1.02	[0.99, 1.04]	1.02	[0.99, 1.04]
13	0.96*	[0.93, 0.99]	0.97*	[0.94, 1.00]	0.96**	[0.93, 0.99]
14–15	0.96***	[0.94, 0.98]	0.97***	[0.95, 0.98]	0.95***	[0.94, 0.98]
Parental relationship to child						
Father (vs. mother)	0.95	[0.90, 1.00]	0.95	[0.90, 1.00]	0.95*	[0.90, 1.00]
Other (vs. mother)	1.13	[0.98, 1.31]	1.12	[0.97, 1.30]	1.12	[0.98, 1.31]
Parental education						
Middle (vs. low)	0.93*	[0.87, 0.99]	0.93*	[0.87, 1.00]	0.93*	[0.87, 0.99]
High (vs. low)	0.92*	[0.85, 0.98]	0.92*	[0.85, 0.98]	0.92*	[0.85, 0.98]
Parental alcohol use						
Not drinking (vs. some drinking)	0.99	[0.97, 1.02]	0.99	[0.97, 1.02]	0.99	[0.97, 1.02]

Note: $N = 9,702$ (34,229 occasions).

* $p < .05$; ** $p < .01$; *** $p < .001$.

CI [1.05, 1.09]) after children consumed their first alcoholic drink, compared with when they had not yet consumed alcohol. Although variation in parental alcohol abstinence had little association with externalizing behaviors, the expected count of externalizing behaviors was significantly lower after children turned 10 years of age and when parental education was higher (vs. lower). Moving beyond AFD, the expected count of externalizing behaviors increased by 16% (IRR = 1.16, 95% CI [1.11, 1.20]) after children experienced their first heavy drinking episode, compared with when they had not yet drunk heavily. In Model 3, effects of AFD and AFHD were reduced in magnitude when both were included as predictors simultaneously, although AFHD still carried more than double the risk of externalizing behaviors as AFD (IRR for AFD = 1.05; IRR for AFHD = 1.13).

Alternative model specifications

In alternative models (available in Supplemental Tables A, B, C, and D), we addressed four additional research questions. (Supplemental material appears as an online-only addendum to the article on the journal's website.) *First, would links of AFD/AFHD with externalizing behaviors be stronger if unobserved time-stable factors were not controlled for?* To address this question, we re-estimated Model 3 shown in Table 2 using a random effects model. Random effects models are similar to fixed effects models in that they can assess time-varying changes in AFD/AFHD and externalizing behaviors. However, random effects models do not control for unobserved time-stable factors (Wooldridge, 2002). The effects of AFD and AFHD are slightly larger in this random-

effects model versus the prior fixed-effects model; however, we believe this is likely the result of the latter model specification controlling for time-stable unobserved factors. To better justify our model, we also performed the Hausman test (Hausman, 1978), which tests the hypothesis that there is no correlation between person effects and the dependent variable. This test was rejected, and thus we focused our interpretation on the fixed effects model specification.

Second, do the effects of AFD/AFHD on externalizing behaviors vary by gender? Despite similarities by gender in AFD (47% of girls and 50% of boys had a first drink by age 14/15 years) and AFHD (11% of both girls and boys had drunk heavily by age 14/15 years), the average count of externalizing behaviors was higher among boys at all ages. To compare the generality of findings by gender, we re-estimated Model 3 shown in Table 2 for girls and boys separately and compared the equality of the estimates using z tests (Clogg et al., 1995). As presented in Supplemental Table B, the effects of AFD/AFHD did not differ significantly by gender.

Third, do rapid AFD to AFHD transitions predict changes in externalizing behaviors? In analyses using Poisson fixed-effects models (Supplemental Table C), we included a time-varying measure indicating transitioning from AFD to AFHD in 1 year or less (coded 1 = yes, 0 = no). By age 14 years, approximately 5.7% of children had experienced a rapid AFD to AFHD transition. When children experienced a rapid transition from AFD to AFHD, the expected count of externalizing behaviors increased by 21% (IRR = 1.21, 95% CI [1.15, 1.27]). The magnitude of this estimate was similar to AFHD effects shown in Model 2 (Table 2).

Last, does early AFD/AFHD increase the risk of children reaching clinical thresholds of externalizing behaviors? At each wave we created a time-varying measure assessing whether children exceeded the clinical threshold for conduct problems or hyperactivity/inattention, based on high scores on the Strengths and Difficulties (about 14% of occasions; see Goodman 1997). Fixed- and random-effects logistic regression models then assessed whether AFD and/or AFHD predicted increased odds of meeting clinical thresholds, and a Hausman test (Hausman, 1978) favored the fixed effects model specification. In the fixed-effects analyses (which controlled for all time-stable factors), the odds of clinical levels of externalizing behaviors increased by 26% (odds ratio = 1.26, 95% CI [1.07, 1.49]) after the first drink, with age and parent-level confounders being controlling for. The first heavy drinking episode was associated with a 65% increase in this risk (odds ratio = 1.65, 95% CI [1.25, 2.18]). This supplemental evidence suggests that AFHD predicted greater increases in the risk of clinical thresholds of externalizing behaviors than AFD.

Discussion

Previous research on potential harms of early AFD on short- and longer-term problem behaviors remains limited by selection issues and scant scholarly attention paid to early heavy drinking. Little work is based on prospective data collected from sufficiently large samples (Kuntsche et al., 2016; Maimaris & McCambridge, 2014). Thus, the lack of controls for confounders precludes clear conclusions about the impact of AFD on youth adjustment. Based on the many possible mechanisms by which alcohol may be dangerous, early AFHD should be more harmful to youth development and health than AFD alone (Kuntsche et al., 2013), but to our knowledge this hypothesis has not been tested previously with nationally representative samples from any country with prospective controls from childhood.

Using intergenerational, prospective, nationally representative data from a recent U.K. birth cohort of children and parents, we found that children experienced a significant increase in rates of externalizing behaviors after they had a first drink *and* after they first engaged in heavy drinking. The current research has several advantages over existing studies: (a) the within-person analytic design allowed us to control for stable-individual differences and thus rule out a large share of selection influences, (b) the multi-informant design limited shared-method bias, and (c) the assessments of AFD/AFHD at modal ages 11 and 14 years reduced recall bias. These advantages lend support to the notion that AFD/AFHD in childhood may increase the proximal likelihood of problem behaviors in the early-life course.

In our results, AFHD evidenced consistently stronger

links with changes in problem behaviors compared with AFD, consistent with between-person findings in cross-sectional research (Asbridge et al., 2016; Kuntsche et al., 2013). Numerous mechanisms may underlie these findings, including harms to neurocognitive functioning, learning to appreciate short-term rewards of heavy drinking, and developing ties with deviant peers who also engage in heavy drinking and other problem behaviors. Testing such plausible processes linking AFHD to short- and longer-term problems behaviors is beyond the scope of this manuscript and awaits further exploration.

Although AFHD is a stronger predictor of problem outcomes, as some scholars have argued (Kuntsche et al., 2013; Warner & White, 2003), our results document that children show elevated rates of problem behaviors after initiating alcohol use even without progressing to heavy drinking. A possible reason for this finding is that our measure of AFD is based on drinking “more than a few sips” and thus excludes occasions when children have a sip of alcohol with parents or during a religious ceremony. It is also important to note that approximately one in seven MCS children had their first drink by age 11 years (Maggs et al., 2015), and thus AFD in this context might be better described as *very* early drinking (Maggs et al., 2019). Last, the prospective assessment of AFD in this study minimizes recall bias, and therefore “forward-telescoping” teenagers are not incorrectly grouped with teenagers whose AFD occurred at a later, more developmentally normative age.

Limitations

Despite its many strengths (i.e., prospective, multi-informant design; strong controls for selection influences), our study has some limitations. First, although we controlled for several time-varying parent-level confounders and all time-stable individual differences (both observed and unobserved), other time-varying unobserved factors may be spuriously related to early AFD/AFHD and problem behaviors, such as changes in school achievement or peer dynamics. Second, the MCS experienced nonrandom attrition; however, our analyses still included more than 10,500 children followed over 35,000 observations. Third, analyses were based on self-reported data, although the repeated assessments and multiple informants helped to reduce concerns about validity and potential bias. Fourth, only one parent reported on the child’s externalizing behaviors at each wave, although on 96% of occasions this was the mother and we controlled for changes in who provided this report. Last, we did not address longer term effects of AFD/AFHD on young adult alcohol use and problem drinking. The MCS is ongoing; therefore, as more waves are collected, researchers can test whether inverse links between AFD/AFHD and adult alcohol problems are mediated by an uptick in problem behaviors in adolescence.

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