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Childhood sexual abuse and the course of alcohol dependence development: Findings from a female twin sample

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Abstract

Childhood sexual abuse (CSA) has been associated with increased risk for alcohol dependence (AD), but the extent to which CSA history may impact transitions in the course of AD development remains unclear. The current study examined the role of CSA in initiation of alcohol use and rate of progression from first drink to AD using a sample of 3,536 female twins (mean age = 21.6 years). Psychiatric diagnoses and alcohol use histories were obtained via telephone interviews using an adaptation of the SSAGA. The contribution of CSA to alcohol outcomes independent of familial influences was estimated by using co-twin AD status to adjust for familial liability to AD. CSA was associated with higher rates of both lifetime alcohol use and AD, but CSA-associated risk for consumption of first alcoholic drink was evident only at ages 12 and 13. Rate of transition from first alcohol use to AD did not differ by CSA status. Findings indicate that CSA and elevated risk for AD may be linked via early age at first drink and that progression from first drink to AD follows a similar course among women with and without histories of CSA.

Keywords

sexual abuse; alcohol dependence; alcohol initiation; twins

1. Introduction

Childhood sexual abuse (CSA) has been linked to increased risk for alcohol related problems in community-based (Dinwiddie et al., 2000;Fergusson et al., 1996;Kendler et al., 2000;Kilpatrick et al., 2000;Molnar et al., 2001;Nelson et al., 2002) as well as clinical samples (Moncrieff and Farmer, 1998; Simpson and Miller, 2001). Estimates from general population-based studies suggest that exposure to CSA is associated with a 1.5 to 3 fold increase in the odds of lifetime alcohol dependence (AD) (Dinwiddie et al., 2000;Fergusson et al., 1996;Kendler et al., 2000;Molnar et al., 2001;Nelson et al., 2002). CSA has also been linked to a range of other alcohol-related outcomes, including negative consequences of alcohol consumption, past month drinking, and an increased likelihood of alcohol use during adolescence (Harrison et al., 1997;Wilsnack et al., 1997).

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The majority of studies cited above have looked simply at the lifetime prevalence of AD or other alcohol related outcomes and have not considered their developmental course, yet drinking trajectories fluctuate considerably over time (Bennett et al., 1999;Jackson et al., 2000) and may differ significantly between individuals who meet lifetime criteria for a given alcohol outcome (Chassin et al., 2002; Schulenberg et al., 1996). Information about the timing of various drinking milestones creates a much richer picture of the progression of alcohol related problems than data on a single feature of the course of alcohol use. The one component of drinking course that has been relatively well studied in relation to CSA history is the onset of alcohol use. The association between CSA and early initiation of alcohol use has been documented in a number of studies, including Bensley and colleagues' (1999) survey of substance use behaviors in 8^{th}, 10^{th}, and 12^{th} graders. Elevated risk for the first heavy drinking episode occurring before age 15 has also been tied to CSA (Edgardh and Ormstad, 2000). Retrospective reports from individuals with substance use disorders (including alcohol abuse and dependence) indicate a link between CSA and early age at first drink as well (Brems et al., 2004;Kilpatrick et al., 2000). Far less is known about the potential role of CSA in shaping other stages of problem alcohol use, but increases to above average levels of alcohol consumption during adolescence have been reported in girls with CSA histories (Pedersen and Skrondal, 1996), suggesting that CSA may also impact transitions later in drinking course.

Although CSA has been tied to a range of alcohol related outcomes, these associations may reflect familial risk for alcohol use disorders (AUDs) and other factors linked to increased risks both for exposure to CSA and for the development of alcohol related problems. CSA frequently occurs in families with elevated rates of risk factors for the development of AUDs (Fergusson, et al., 1996;Sheridan, 1995;Vogeltanz, 1999;Walsh, 2003), such as family history of AD (Legrand et al., 1999;Hill et al., 2000) and instability in the family environment (Hussong and Chassin, 1997; Li et al., 2001). Estimates of risk associated with CSA may be confounded by familial liability to AUDs. It is therefore important in studies examining the association of CSA with alcohol related problems to control for potentially confounding background factors. Previous studies have achieved this either by including a range of measured covariates in their analyses (Fergusson et al., 1996;Kilpatrick et al., 2000;Molnar et al., 2001;Mullen et al., 1993) or by using co-twin methodology (Dinwiddie et al., 2000;Kendler et al., 2000;Nelson et al., 2002). These studies have found that the relationship between CSA and alcohol outcomes remains after accounting for parental alcohol related problems and other familial risk factors, consistent with the notion that CSA has a direct effect on alcohol outcomes. Evidence for indirect effects have emerged from several of these investigations as well (Dinwiddie et al., 2000;Fergusson et al., 1996;Nelson et al., 2002), suggesting that although not fully mediating the relationship between CSA and alcohol related problems, family background factors may still play an important role. Few studies examining CSA and initiation of alcohol use have controlled for potential confounders, and in the previously cited Pederson et al. study CSA history was no longer associated with patterns of alcohol consumption after adjusting for parents' alcohol use and smoking and measures of parenting. Thus, while twin and other studies suggest a link between CSA and AD, there is uncertainty concerning the influence of CSA on specific stages in the developmental course of AUDs and the extent to which such associations may be independent of the effects of family background.

The current study aimed to extend the literature on CSA and the course of alcohol related problems by examining the potential influence of CSA on two critical junctures in AD development using a twin design that accounts for familial liability to AD. Both the initiation of alcohol use and AD are commonly studied drinking milestones and, unlike some other intermediate ones (e.g., first intoxication, which occurred on the same occasion as first drink for 48.6% of the current sample) are reached at points in the drinking course that are separated by a substantial period of time. Early age at first drink and AD have also been consistently linked in the literature (DeWit et al., 2000;Grant and Dawson, 1997;Nelson and Wittchen,

1998), although the mechanism underlying this link remains controversial (Prescott & Kendler, 1990). Independent of the debate surrounding causality of their association, both alcohol outcomes have been tied to CSA. Age that the first alcoholic drink was consumed and transition time from first drink to the onset of AD were therefore chosen as the outcomes of interest in the current investigation of CSA in predicting course of AD development. Co-twin AD status was used to adjust for familial liability to AD so that genetic and shared environmental influences on AD as well as on correlated disorders such as conduct disorder and internalizing disorders could be controlled in estimating risk associated with CSA. This approach was chosen over the alternative strategy of using a discordant twin design because the high concordance rates for CSA in twin pairs (Dinwiddie et al., 2000;Kendler et al., 2000) would have resulted in a reduction in sample size (and power) as well as a possible threat to external validity.

2. Method

2.1 Participants

The sample consisted of 3,536 female twins (954 monozygotic (MZ) pairs, 815 dizygotic (DZ) pairs, and 8 singletons) from the Missouri Adolescent Female Twin Study (MOAFTS), a longitudinal study of alcohol related problems and associated psychopathology in adolescent females and women (Heath et al., 2002). Female twin pairs born in Missouri to Missouri-resident parents between July 1, 1975 and June 30, 1985 were identified from birth records and recruitment was conducted using a cohort-sequential design. In the first two years, cohorts of 13, 15, 17, and 19 year-old female twin pairs and their families were ascertained. A new cohort of 13 year-old twins and their families were added in the subsequent two years. Recruitment began in 1995 and continued until 1999. Of the 2,644 families initially targeted for inclusion in the study, parental diagnostic interviews were completed by at least one parent in 2,061 families, representing 78% of eligible families. (For further details on ascertainment, see Heath et al., 2002). Data in the present study were drawn from the fourth and most recent wave of data collection, when participants had a mean age of 21.6 years (range = 18 to 29 years). Approximately 86% of the sample identified as Caucasian and 14% as African-American.

2.2 Procedure

Data collection was performed over the telephone by trained interviewers. An initial screening was conducted with one of the twin's parents to determine zygosity of the twin pair and, when permission was granted, parental diagnostic interviews were scheduled. Interviews with the twins were scheduled after obtaining their verbal consent (and, for those under the age of 18, the consent of parents), in accordance with procedures approved by the Institutional Review Board at Washington University.

2.3 Assessment Battery

Comprehensive diagnostic interviews were conducted using an adaptation of the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA; Bucholz et al., 1994;Hesselbrock et al., 1999), an instrument designed to assess AUDs and related psychiatric disorders in adults. Of relevance to the current study, detailed information about experiences with alcohol as well as childhood sexual abuse histories were queried and DSM-IV diagnoses of alcohol dependence were obtained.

2.3.1 CSA—Childhood sexual abuse experiences were assessed in two sections of the interview, the Traumatic Events and Early Childhood Experiences modules. Participants were coded as positive for a childhood sexual abuse history if they a) reported in the Traumatic Events module that they had been raped or sexually molested prior to age 16 or b) endorsed forced sexual contact with a family member or another adult five or more years older before

the age of 16 in the Early Childhood Experiences module. CSA was reported by 11.5% of the sample and first occurred on average at 8.2 years of age.

2.3.2 Alcohol Use—Age at first drink was defined as the age at which the first full alcoholic drink, i.e. a standard can or bottle of beer, a glass of wine or a shot of liquor, was consumed. Consumption of one or more drinks over the lifetime was reported by 85.3% of participants.

2.3.3 Alcohol Dependence—Age of onset of alcohol dependence (AD) was defined as the age at which full DSM-IV criteria for AD (i.e., three or more AD symptoms occurring during the same 12 month period) were first met. AD criteria were met by 7.9% of the sample.

2.4 Data Analysis

2.4.1. CSA and Lifetime Risks—Logistic regression analyses were initially conducted to test for associations between CSA and lifetime prevalence of both alcohol use and AD, without adjusting for the potential effects of familial liability on alcohol outcomes. A second set of logistic regressions that adjusted for co-twin AD status were then conducted. Zygosity and an interaction term reflecting zygosity by co-twin AD status, which provided an estimate of the degree to which familial risk may be attributed to genetic versus environmental factors, were also included in the models. (The strategy of using co-twin AD status is conservative and may lead to an underestimation of the impact of CSA on alcohol outcomes. Because of the high concordance rates of CSA in twin pairs (Dinwiddie et al., 2000;Kendler et al., 2000), adjusting for co-twin AD status may also partially adjust for exposure to CSA.)

2.4.2 CSA and Speed of Transitions—Although the logistic regression analyses described above quantify the association between CSA and lifetime risks for alcohol use and AD, they do not address the key issue of whether exposure to CSA alters the speed of transition from non-use to first alcohol use and from the onset of alcohol use to the onset of AD. To examine this issue, two models were constructed that captured the role of CSA in transitions during AD development by using CSA to predict a) age at first alcoholic drink and b) time from first alcoholic drink to onset of AD. Cox proportional hazards models were chosen to conduct multivariate regression with time-to-event data (Cox, 1972) given that not all of the twins had lived through the periods of risk for first alcohol use and for the development of AD. The advantage of this approach is that the individual's data up until the time of censorship is used in the calculation of hazard ratios. In an effort to ensure that the sequences of CSA events and alcohol outcomes were modeled accurately (e.g., that only CSA preceding age at first drink was characterized as a risk factor for initiating alcohol use) a “person year” data set was created using SAS, version 9.1 (SAS Institute Inc., 2002) to represent time-varying covariates. Data were constructed such that each line of data represented a single year of life for every individual. For cases that were positive for CSA, CSA was coded as absent in each year up to the age that the abuse occurred and present from that year onward. Analyses were conducted with STATA, version 8.2 (Statacorp, 2005). Standard deviations of means and confidence intervals for Cox regression models were adjusted for family clustering using Huber-White robust standard errors.

2.4.2.1 Time to first drink Variables representing CSA and co-twin AD status as well as zygosity and the interaction of zygostity with co-twin AD status were entered into the model. Zygosity and zygosity by co-twin AD status did not produce significant hazard ratios and were dropped in the final model. The proportional hazards assumption that risk remains constant over time was assessed using the Grambsch and Therneau test of the Schoenfeld residuals (Grambsch and Therneau, 1994). Proportional hazard assumptions were violated for CSA and co-twin AD status. In order to adjust for violations, risk period was split into 11 sub-divisions (up through age 11, separate sub-divisions for each year from ages 12 through 20, and age 21

and up) and interaction terms were created between each of the 11 sub-divisions and the two covariates. Each interaction term was entered into the model one at a time to test for changes in the proportional hazard estimations for CSA and for zygosity. Inclusion of the interaction terms reflecting CSA status at age 12, CSA status at age 13, CSA status at age 17, and CSA status at age 20 resulted in a non-significant outcome in the test of proportional hazard assumptions for CSA. Inclusion of the interaction term reflecting co-twin AD status by the risk period through age 12 resulted in a non-significant outcome in the test of proportional hazard assumptions for co-twin AD status.

2.4.2.2 Time from first drink to AD onset Time to AD onset was defined as years since first alcohol use. In addition to CSA and co-twin AD status, two dummy variables, reflecting the lowest and highest thirds of the distribution for age at first drink, were created to represent age at first drink and were entered into the model to adjust for variability in proximity of age at first drink to the risk period for development of AUDs. (Categorical variables were created both for ease of interpretation and for potential use in constructing interaction terms representing age at first drink in combination with other covariates.) “Early age at first drink” was defined as first alcohol use at age 15 or younger and “late age at first drink” as first alcohol use at age 18 or older. Zygosity by co-twin AD status was inestimable and zygosity did not produce a significant hazard ratio. Both were dropped in the final model. Tests revealed that the proportional hazard assumptions were not violated.

3. Results

3.1 Prevalence and Age of Onset For Alcohol Use and Dependence By CSA Status

Exposure to childhood sexual abuse was associated with increased rates of both lifetime alcohol use and alcohol dependence. Lifetime alcohol use was reported by 90.1% of CSA-positive participants versus 85.3% of CSA-negative participants, (OR=1.45; CI=1.04-2.04). Prevalence of AD was 15.6% among twins with CSA histories versus 7.0% of those without, (OR=2.87; CI=2.14-3.86). CSA was also associated with a younger age of onset for first alcohol use, 15.7 versus 16.5 years of age, $F(1, 1843)=29.74$; $p< 0.001$, but not for onset of AD, 18.3 versus 18.8 years of age, $F(1, 266)=1.36$; $p= 0.244$.

3.2 CSA as a Predictor of Lifetime Alcohol Use and AD, Adjusting For Familial Liability to AD

History of CSA was next assessed as a predictor of lifetime alcohol use using a logistic regression model that included co-twin AD status, zygosity, and the interaction between zygosity and co-twin AD status to adjust for potentially confounding familial influences that may predispose to both CSA and alcohol dependence. Results revealed that CSA history was associated with an increased likelihood of having consumed alcohol over the lifetime, (OR=1.47; CI=1.03-2.10). Zygosity by co-twin AD status was significant as well (OR=7.36; CI=1.55-34.89), indicating that the relationship between co-twin AD status and lifetime alcohol consumption was stronger for MZ versus DZ twins. A logistic regression using CSA status to predict AD revealed a significant association between CSA status and AD. After adjusting for co-twin AD status, zygosity, and the interaction between zygosity and co-twin AD status, CSA history was associated with elevated risk for AD (OR=2.02; CI=1.45-2.80). The interaction between zygosity and co-twin AD status was also significant (OR=3.88; CI=1.47-10.24), suggesting that the association of co-twin AD status and lifetime alcohol consumption was stronger for MZ versus DZ twins.

3.3 Association Between CSA and Transition to First Alcohol Use, Adjusting For Familial Liability to AD

Whereas the above results show that CSA predicts lifetime alcohol consumption, they do not address the issue of whether CSA is associated with earlier age at first use. Results of the first

survival analysis, which used CSA to predict age at first drink, are reported in Table 1. The hazard ratio for CSA was not significant (HR=1.08; CI=0.94-1.24), but as noted earlier, interaction terms representing CSA status at ages 12, 13, 17, and 20 were added to the model to adjust for proportional hazard assumption violations and revealed age-specific associations between CSA and risk for alcohol use initiation. CSA was associated with increased risk for initiation at ages 12 (HR=2.58; CI=1.69 – 3.94) and 13 (HR=1.69; CI=1.23 – 2.34) and lower risk for initiation at age 20 (HR=0.51; CI=0.31 – 0.85). Findings also indicated significant risk conferred by familial liability to AD, as measured by co-twin AD status. Co-twin AD was associated with a two fold increase in risk for early age at first drink (HR=2.27; CI=2.01 – 2.57) and the interaction term representing co-twin AD status and risk for first alcohol use prior to age 12 was significant as well (HR=2.13; CI=1.26 – 3.59).

3.4 Association Between CSA and Transition From First Alcohol Use to AD, Adjusting For Familial Liability to AD

As seen in Table 2, after adjusting for age at first drink and co-twin AD status, CSA history did not predict time from first alcohol use to onset of AD, (HR=0.93; CI=0.67-1.27), but results revealed co-twin AD status to be a potent predictor of rapid progression from first drink to AD (HR=4,063.27; CI=566.07 – 29, 166.51) and indicated that the transition time is longer for early initiates of alcohol use (0.61; CI=0.46 – 0.80) compared with those initiating alcohol use at 16 and 17 years of age.

4. Discussion

Expanding on previous efforts that have established a link between CSA and elevated risk for AD, the current study addressed potential differences in the impact of CSA on alcohol outcomes at two stages of the development of AD. The association between CSA and two key transition points in the course of the disorder, initiation and progression from first drink to AD, were examined using a genetically-informed design that adjusted for potential confounds of familial liability to AD and produced estimates of the contribution of CSA to alcohol outcomes that were independent of familial influences.

Consistent with the literature linking CSA to lifetime alcohol use (Bensley et al., 1999;Harrison et al., 1997) and AD (Dinwiddie et al., 2000;Kendler et al., 2000;Molnar et al., 2001;Nelson et al., 2002), CSA was associated with increased likelihood of lifetime alcohol use and was a significant predictor of lifetime diagnosis of AD, even after adjusting for variance in outcomes attributable to familial liability to AD. The significant interactions between co-twin AD status and zygosity are consistent with previous reports of genetic influences on alcohol use and dependence (Heath and Martin, 1994;Heath et al., 1997;Liu et al., 2004;Prescott and Kendler, 1999), and highlight the importance of adjusting for such potentially confounding effects in analyses examining the impact of CSA on alcohol related outcomes.

Initial analyses that did not adjust for familial liability indicated earlier first use among women with CSA histories, echoing findings in the small but consistent literature that ties CSA to early initiation of alcohol use (Edgardh and Ormstad, 2000;Harrison et al., 1997;Kilpatrick et al., 2000;Kendler et al., 2000). However, once familial liability to AD was accounted for, variability in the relationship between CSA and initiation of alcohol use over the period of risk became apparent. Elevated risk of first use was found to be specific to ages 12 and 13, suggesting that the impact of CSA on risk for first alcohol consumption is greatest early on in the period of risk for alcohol initiation. (CSA was in fact associated with decreased risk for first use at age 20, likely due to the fact that the majority of individuals with CSA histories who drank during their lifetimes had already initiated alcohol use by that age.) Familial liability to AD also appeared to have a greater impact on initiating alcohol use earlier in the risk period, in keeping with studies that have revealed differential impact of genetic liability over the course

of development (Koopmans and Boomsma, 1996) and have found associations between genetic liability to alcohol use disorders and early initiation (Johnson et al., 1996;McGue et al., 1992). In contrast, rate of progression from first drink to AD was not associated with CSA status, suggesting that CSA as a risk factor has its effects primarily by increasing risk of early-onset alcohol use.

4.1 CSA and AD

Prevalence of lifetime AD was elevated among participants with CSA histories, yet the rate of transition from first drink to AD did not differ by CSA status, suggesting distinctions between the impact of CSA on progression of the disorder versus overall likelihood of meeting diagnostic criteria. Of note, risk for first alcohol use conferred by CSA history was significant for early age at first drink and early age at first drink has been closely tied to increased risk for AD in the larger literature (DeWit et al., 2000;Grant and Dawson, 1997;Nelson and Wittchen, 1998). Taken together, they suggest that CSA and AD may be linked via early age at first alcohol use, although whether the association between early use and AD is a causal one cannot be addressed by the current findings. Adjustment for familial liability to AD in the present study rules out the possibility that this link is simply reflecting family history of alcohol related problems as a risk factor common to CSA, early alcohol use, and development of AD. It appears therefore that CSA increases the likelihood of early alcohol use (a marker for AD risk), but is not associated with the rate of progression from initiation of alcohol use to AD.

4.2 CSA and Course of AD Development

Findings suggest that women with histories of CSA are at increased risk for alcohol use and AD, but the impact of CSA is not stable over the course of AD development, with peaks occurring early in the period of risk for alcohol use and misuse. Observed fluctuations in CSA-associated risk may reflect diminished impact of CSA on alcohol related outcomes over time. That is, the closer proximity in time between CSA events and initiation of alcohol use versus AD onset explains distinctions in outcomes at these two stages. Changes in the potency of risk factors during the course of development have long been discussed in the developmental psychopathology literature (Cicchetti and Rogosch, 2002) and evidence for age period-specific predictors of the course of alcohol use disorders has emerged recently as well (Sher et al., 2004). Results may also reflect differences in the relevance of certain risk factors at varying stages of AD development. Evidence from twin studies examining milestones in the development of substance use disorders, for example, indicate that genetic, shared environmental, and unique environmental contributions vary by stage of development of the disorder (Agrawal et al., 2005;Kendler et al., 1999;Pagan et al., 2006). In the present study, the differences in impact of CSA-associated risk between initiation and progression to AD may simply reflect the relative increase in potency of other risk factors later in the course of AD development.

Despite its contribution in revealing mechanisms underlying the association between CSA and the development of AD, certain limitations in the present investigation suggest the need for further research in this area. First, assessment of sexual abuse in the Early Childhood Experiences module included the term “forced” in questions regarding abuse by a family member, which may have led to underreporting. However, participants could also meet CSA criteria by endorsing molestation or rape prior to age 16 in the Traumatic Events module, which was administered first and therefore would not have been affected by the phrasing of questions in the Early Childhood Experiences module. Second, age that sexual abuse occurred was only available for first and most recent CSA events and severity of abuse history was not assessed. Gathering a complete history of CSA, including frequency and duration of abuse episodes and number of CSA events, would make it possible to determine whether proximity of abuse to the age when alcohol is readily accessible and use is developmentally normative has an impact

on alcohol outcomes. In addition, potential moderating effects of CSA severity, which have been found for AD (Fergusson et al., 1996;Nelson et al., 2002), could be tested on features of the course of AD development. Third, confidence in the reliability of drinking histories would be enhanced through the use of prospectively collected data or the supplementation of self-reports with corroborating information from other sources in future studies. Fourth, given the lack of consistency across gender in the prevalence of CSA and associated outcomes, findings may not generalize to males with sexual abuse histories. Finally, extending the period of observation to later adulthood when all respondents have passed through the age of risk for initiation and development of alcohol related problems would reduce the likelihood that findings characterize a subset of individuals with an early onset trajectory of AD. It would also create the opportunity to examine the role of CSA in shaping features of course in later stages of AD, such as duration of problem use, frequency of relapse, and rates of recovery. Through these efforts, a more complete picture of the course of AD among individuals with histories of CSA can be created and applied to the development of prevention and intervention efforts for this high-risk population.

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Table 1

Hazard ratios from Cox proportional hazards model predicting time to first drink

	Hazard Ratio (95% CI)
CSA status: present	1.08 (0.94 – 1.24)
CSA status by risk for initiation of alcohol use at [a] :	
Age 12	2.58 (1.69 – 3.94)
Age 13	1.69 (1.23 – 2.34)
Age 17	0.71 (0.51 – 1.00)
Age 20	0.51 (0.31 – 0.85)
Co-twin AD status	2.27 (2.01 – 2.57)
Co-twin AD status by risk of initiation prior to age 12[b]	2.13 (1.26 – 3.59)

[a] Interaction terms included in the model to adjust for violation of proportional hazard assumption for CSA.

[b] Interaction term included in the model to adjust for violation of proportional hazard assumption for co-twin AD status.

Table 2

Hazard ratios from Cox proportional hazards model predicting time from first drink to onset of AD

	Hazard Ratio (95% CI)
CSA status: present	0.93 (0.67 – 1.27)
Co-twin AD	4,063.27 (566.07 – 29, 166.51)
Age at first drink[a]	
Early age at first drink (<= 15 years)	0.61 (0.46 – 0.80)
Late age at first drink (>= 18 years)	1.29 (0.83 – 2.03)

[a] Hazard Ratios relative to average age at first drink (16 to 17 years)