Elaborating the association of age at first drink with risky drinking: results from a cross-sectional survey

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ABSTRACT
Objective: An association between early drinking onset and subsequent heavy drinking has been established, but interpretation is problematic: is the onset of drinking or of intoxication important; is early onset a marker for vulnerability or a causal risk factor; what role does recall bias play? We (1) compare the associations of onset indicators with subsequent risky drinking, (2) assess whether drinking onset has a stronger association with subsequent risky drinking than relative onset (deviation from cohort’s median), and (3) study recall bias by examining within-cohort temporal changes in reported ages of drinking onset.

Method: The Finnish Drinking Habits Survey, a cross-sectional general population survey of 15–79-year-old Finns collected in 2016. Additionally, the four previous survey waves from 1984 to 2008 were used. Risky drinking was defined using the Alcohol Use Disorders Identification Test (AUDIT), and the age of first drink (AFD) and of intoxication (AFI) were obtained by retrospective recall.

Results: All three indicators showed statistically significant associations with AUDIT, but AFI had the strongest association. Absolute age of onset had a slightly stronger association with AUDIT than relative age of onset. Recall bias of AFD was found for each birth cohort, as the reported AFD within each cohort increased over time.

Conclusions: Onset of intoxication has stronger association with risky drinking than the onset of drinking. No gain was seen in measuring relative rather than absolute age of onset, which suggests that age of onset is not merely a marker of vulnerability to risky drinking.

Introduction
Rates of underaged alcohol use have decreased in the 21st century in Finland and in many other countries in Europe (ESPAD Group, 2020) and beyond (Raninen and Livingston 2018). Nevertheless, alcohol use continues to be an important cause of adolescents’ deaths from, e.g. motor vehicle accidents, violence and suicide (Gore et al. 2011). Also, adolescent alcohol use has been linked to many physiological harms at the individual level, e.g. injuries, poorer mental health (McKinnon et al. 2016; Silins et al. 2018) and behavioural problems, e.g. gambling and other substance use (Dowling et al. 2017; Hall et al. 2016).

Numerous studies have demonstrated a correlation between early age at first drink (AFD) and problematic alcohol use in adulthood (Grant & Dawson, 1997; Guttmannova et al. 2011; Kuntsche et al. 2016). If this connection is causal, the aforementioned declining trends in youth drinking are important not only in their own right but also from the viewpoint of preventing alcohol-related harm later in life. However, it is still under debate whether early AFD in itself has a causal relationship with adverse outcomes, as longitudinal research evidence on this is scant and not always supporting a causal connection (Maimaris and McCambridge 2014; Meque et al. 2019). An alternative hypothesis is a ‘marker hypothesis’ or ‘shared vulnerability’ hypothesis, according to which age of onset of alcohol use is ‘not a direct risk factor for alcoholism, but an alternative manifestation of vulnerability to problematic alcohol involvement’ (Prescott and Kendler 1999).

The concept of AFD has also inherent limitations, such as problematic reliability and inconsistent definition (Kuntsche et al. 2016; Rossow and Kuntsche 2013). In many studies, age of onset is enquired from adults, and in these cases recall bias affects the results. Additionally forward telescoping has been observed when people report AFD, meaning that the reported age is more recent than it actually was (Shillington et al. 2012). Telescoping effect in self-reported AFD has been found both at the individual level in follow-up studies (Shillington et al. 2012) and in population samples in cross-sectional studies (Golub, Johnson, and Labouvie 2000). The inconsistent definition means that AFD has varied in the literature ‘from having one sip to the consumption of several full drinks’ (Kuntsche et al. 2016).

In contrast to the inconsistent findings between early AFD and adverse outcomes later in life, age at first intoxication (AFI) has been found to be a more robust predictor of adult substance use disorders (Newton-Howes et al. 2019) and even risk of death by age 30 (Levola et al. 2020). The...
association between early AFI and adverse outcomes related to alcohol use disorder and substance use disorder remained statically significant even after adjusting for various confounding variables. Other studies have also found that early AFI may be associated with heavy drinking and related problems later in life (Warner, White, and Johnson 2007; Warner and White 2003) and with problem behaviour among adolescents (Kuntsche et al. 2013). In addition, a short length of time (delay) from the first alcoholic drink to first alcohol intoxication has been shown to be associated with increased frequency of heavy drinking (Morean, Corbin, and Fromme 2012; Morean et al. 2014).

In this study, we use cross-sectional and repeated cross-sectional surveys to explore the association between age of onset of drinking and adult risky drinking from three points of view. With these data, we cannot resolve the question of causality, but we may contribute to shedding light on the question and to a better understanding of the observed associations. First, we will revisit the question of the strength of age at first drink, age at first intoxication (ascertained retrospectively), and the progression time from the former to the latter (delay) as predictors of subsequent risky drinking, as studies using all three indicators simultaneously are rare. Secondly, we will revisit the question of recall bias: how do the cohorts’ reported average ages of drinking onset change from one time point to another?

With the third viewpoint, we aim to assess the benefit of defining the age of onset in relative terms. The notion of early or late age of onset is dependent on individual and cultural factors (Kuntsche et al. 2016). What is early for one country, time period or cohort may be late for another. In contrast, the absolute age of onset of drinking does not depend on context. Specifically, we assess how the relative position within one’s own birth cohort in the age of onset compares with absolute age of onset as a predictor of subsequent risky drinking. By defining the age of onset as a relative position within one’s birth cohort, we also aim to contribute to assessing the merit of the marker hypothesis. We assume that in case the association is due to a causal, biological pathway, absolute onset age should matter more, and in case the association arises because age of onset is a marker of latent vulnerability to alcohol problems, we would expect relative onset age, i.e. an early age of onset compared to peers in the same cohort, to play a stronger role.

**Methods**

**Data**

The main data set was based on the 2016 Finnish Drinking Habits Survey (FDHS), a general population survey of 15–79-year-old Finns, with 2,285 respondents and a 59% response rate. The survey was carried out as face-to-face interviews and the fieldwork was conducted by Statistics Finland. Young adults aged 18–25 were given a two-fold selection probability in the random sampling compared to other age groups. When respondents with missing data on AUDIT score or missing data on both AFD and AFI were left out (of whom 57 per cent were abstainers), the data set consisted of 2,007 respondents. An additional data set, for analysing recall bias, was constructed by adding data from the four previous FDHS waves collected in 2008 (n = 2,725), 2000 (n = 1,932), 1992 (n = 3,446), and 1984 (n = 3,624), with response rates of 74, 78, 87 and 94 percent, respectively. Weights were based on post-stratification for sex, age and geographical region and were used in the analyses to correct for deviations from population representation caused by the over-sampling of young adults and by differential non-response.

**Measurement**

Risky drinking was assessed using the 10-item Alcohol Use Disorders Identification Test (AUDIT) (Babor et al. 2001; Saunders et al. 1993). The AUDIT is a screening instrument for problematic alcohol use, containing ten questions measuring alcohol use, alcohol dependence symptoms and alcohol-related harms to the drinker. The full AUDIT screening has proved to perform the best in detecting risky drinking when compared to shorter forms, i.e. AUDIT-C, AUDIT-3, and AUDIT-QF (Kaarne et al. 2010). Each item is given a score from 0–4 points and the total AUDIT score ranges from 0 (abstinence and no problems) to 40. The AUDIT score was used as the primary measurement in the analysis, but because AUDIT is especially in clinical settings often used in a dichotomised form using cut-off scores, we also show the associations with age of onset for AUDIT scores of 8+ (indicative of hazardous and harmful alcohol use) and scores of 16+ (which indicates a need for counseling and continued monitoring due to a high risk of alcohol problems).

Age at first drink (AFD) and age at first intoxication (AFI) were measured with the following questions: ‘How old were you when you first consumed some alcoholic beverage?’ and ‘How old were you when you were drunk for the first time?’. The length of time in years (delay) between first drink and first intoxication was calculated for people who reported both, by subtracting AFD from AFI.

For assessing recall bias, we used data from 1984 to 2016. For this part of the analysis, only AFD was available from the earlier waves of FDHS. We identified eleven 8-year birth cohorts, starting from the 1913–1920 cohort and ending in the 1993–2000 cohort. We calculated the average AFD for each cohort in each of the five waves in which the cohort was included. For example, the cohort 1945–52 entered FDHS in 1984 when they were 32–39 year olds, and in the next wave they were 40–47 year olds, and so on.

Relative AFD and AFI, i.e. age of onset variables that take into account that the definition of ‘early’ changes by cohort, were constructed by centring the variable to the median value of the respondent’s birth cohort, with the birth cohorts defined as above. AFI and delay were available in the 2016 FDHS for the subset who had ever been intoxicated.

**Statistical analyses**

For the analysis, AFD, AFI, Delay, and relative AFD and AFI were divided into four categories in order to account for the non-linear relationship between the onset indicators and the
AUDIT score. First the onset indicators were divided into four quartiles with as equal sizes as possible. To ensure that this categorisation meaningfully captured the non-linearity in the association, the cut-off points were compared to a LOESS-regression curve between each individual onset indicator and the AUDIT-score (Supplementary Appendix 1). The association between the onset indicators and the dichotomised AUDIT scores of 8+ and 16+ were modeled with logistic regression. When the AUDIT score itself was modeled, it was considered a count variable and because the score was found to be overdispersed, i.e. the variance was much higher than the mean, negative binomial regression was used to model the associations between AFD, AFI and delay with the AUDIT score. All models were adjusted for sex and age divided into three categories: 15–29, 30–59, and 60–79 years of age. The cut-off points for age were assessed similarly to onset indicators using LOESS-regression (Supplementary Appendix 1). The modelling performance was assessed using Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC). Both AIC and BIC can be written in the form $[-2L + A_n, p]$ where $L$ is the log-likelihood, $p$ is the number of parameters in the model, and $A_n$ is 2 for AIC and In(n) for BIC (Dziak et al. 2020); hence in large data sets ($n > 7$) the penalty weighting $A_n$ associated with the number of parameters (variables) is larger for BIC than for AIC and gets larger as the $n$ grows. In model selection, BIC emphasises the smallest adequate model and avoids Type I error (an overfit model) whereas AIC emphasises good prediction and avoids Type II error (an underfit model) (Dziak et al. 2020). Statistical analyses were performed using SAS EG version 7.15 (SAS Institute Inc, Cary, NC, USA), except for LOESS-regression, for which R Statistical Software version 3.6.0 (R Foundation for Statistical Computing, Vienna, Austria) was used.

Results

Age at first drink, age at first intoxication and delay between them

Table 1 shows the mean AUDIT score, percentage of respondents at elevated risk (AUDIT 8+) and high-risk level (AUDIT 16+) across AFD, AFI and delay quartiles of AFD, AFI, and delay in the 2016 survey. For onset indicators AFD and AFI, the mean AUDIT score and the percentage of respondents drinking at elevated risk (8+) or high-risk level (16+) is the highest among those with the lowest age of onset and lowest among those with the highest age of onset. The association is systematic across quartiles, although there is a pronounced difference between those whose age of onset is lower than median (q1 and q2) and those for whom it is higher (q3 and q4). In addition, for AUDIT 16+, the p-value for AFI was lower than for AFD (0.001 vs. 0.042). For the delay measure, over half of the respondents (53.0 per cent) had AFD and AFI at the same age. Here, the central finding was that in the group with the longest time between age of first drink and age of first intoxication (q4), less risky drinking was reported.

### Modeling the association between onset and AUDIT

The results in Table 1 show that there is a statistically significant association between all three onset indicators and AUDIT scores when the indicators are modeled individually and adjusted for sex and age. However, because the onset variables are highly correlated ($r = 0.74$ between AFD and AFI, $p < .0001$), the associations could be due to one true relationship and others merely reflecting that one. Therefore, the analysis continued by assessing which of the indicators would outperform the others if introduced in a model simultaneously (i.e. which has a stronger association than other variables when those others are controlled for; Table 2). This is informative even if care should be exercised in interpreting the resulting estimates due to the multicollinearity. All three indicators cannot be in the same model, as they are linearly dependent so that any one of the indicators can be derived from the other two.

When AFD, AFI and delay were modeled in pairs, all three indicators in models 1–3 were found to be statistically significant (adjusted for sex and age). This means that the association cannot be reduced to only one of the indicators. However, when AFD and AFI were in the same model (model

| Quartile | N   | Mean audit score (SD) | AUDIT 8+ (%) | AUDIT 16+ (%) |
|----------|-----|-----------------------|--------------|--------------|
| AFD (p-value*) |     |                       |              |              |
| 1 lowest  | 561 | 6.21 (4.61)           | < .0001      | < .0001      | 0.42         |
| 2         | 739 | 5.71 (4.61)           | 31           | 5            |
| 3         | 463 | 3.75 (3.88)           | 15           | 2            |
| 4 highest  | 244 | 2.59 (2.97)           | 10           | 1            |
| All (with AFD) | 2007 | 4.99 (4.47) | 23           | 3            |
| AFI (p-value*) |     |                       |              |              |
| 1 lowest  | 333 | 6.77 (5.04)           | 36           | 7            |
| 2         | 625 | 6.16 (4.41)           | 32           | 4            |
| 3         | 535 | 4.95 (4.38)           | 21           | 3            |
| 4 highest  | 363 | 3.14 (3.22)           | 9            | 1            |
| All (with AFI) | 1856 | 5.30 (4.50) | 25           | 4            |
| Delay (p-value*) |     |                       |              |              |
| 1 & 2 ** shortest | 978  | 5.52 (4.67)           | 30           | 5            |
| 3         | 550 | 5.69 (4.54)           | 29           | 4            |
| 4 longest  | 327 | 3.98 (3.58)           | 14           | 1            |
| All (with AFD and AFI) | 1856 | 5.30 (4.5) | 25           | 4            |

*Adjusted for age group and sex. **Quartiles 1 and 2 are combined (53 percent reported the same age for AFD and AFI). *Excluding respondents with no reported AFI.
while the estimates for the delay variable indicated that intoxication continued to be associated with the lowest risk, model 3 with AFI and Delay. In this model, a late onset of drinking. When the three models were compared using AIC criterion are constructed (BIC introduces a greater penalty than AIC). On the basis of models 3 and 7, as could be expected on the basis of how the two criteria are constructed (BIC introduces a greater penalty weight for each added variable than AIC). On the basis of AIC’s tendency to emphasize models with good estimation power, and because the delay variable was significant in models 3 and 7, these models containing AFI and delay were considered to be most supported by the data (see methods for details). Both AIC and BIC showed that when delay was included in the model, the model with absolute AFI performed slightly better than the model with relative AFI (model 3 in Table 2 vs. model 7 in Table 3), and overall, the combination of absolute AFI and delay (Model 3, Table 2, adjusted for sex and age) proved to be the best model in terms of the ratio of strength of association to subsequent mean AUDIT score.

**Recall bias: do cohorts’ reported age of onset change over time?**

Figure 1 shows average reported ages at first drink in different birth cohorts as they progress from one age group to another in the five waves of FDH-surveys carried out from 1984–2016, with each line describing responses of one cohort in different years. For example, the first data point for the 1953–1960 cohort is for the year 1984, when the cohort was 24–31 years old, and the last point is for the year 2016, when the cohort was 56–63 years old. Overall, the AFD decreased with birth cohorts until the cohort 1961–1968 (i.e. the lines for later cohorts in this range tend to be at a lower level than the lines for earlier cohorts). After this cohort, the record-low ages at onset were also maintained for the two cohorts born between 1969 and 1984. However, for the two most recent cohorts born between 1985 and 2000, the average AFD began to increase, which reflects the reduced rates of drinking and postponing of drinking onset among the under-aged in the 21st century.

Next, we investigated whether the mean AUDIT score is better predicted by the absolute age of onset (as in previous tables) or relative age (i.e. age of onset centred on the median value of the respondent’s birth cohort). The results shown in Table 3 are given only for AFI (and not AFD), because it was found above that AFI was more strongly associated with AUDIT scores than AFD.

When modelled individually (and adjusted for sex and age), AFI, when defined both in absolute and relative terms, was statistically significantly associated with the mean AUDIT score (models 4 and 5). When modeled individually, both model criteria indicated that the relatively defined AFI performed marginally better compared to the absolute AFI, but when they were entered in the same model (model 6), neither was statistically significant. Absolute AFI was combined with the delay in model 3 (shown in Table 2), and relative AFI with delay in model 7. BIC was found to be at its lowest for the absolute AFI, when defined both in absolute and relative terms (models 4 and 5), while AIC was its lowest for the models with delay added (models 3 and 7), as could be expected on the basis of how the two criterion are constructed (BIC introduces a greater penalty weight for each added variable than AIC). On the basis of AIC’s tendency to emphasise models with good estimation power, and because the delay variable was significant in models 3 and 7, these models containing AFI and delay were considered to be most supported by the data (see methods for details).

### Table 2. Comparison of models containing AFD, AFI and Delay: estimated incidence rate ratios for the mean AUDIT score and model comparison criteria. Finnish Drinking Habits Survey 2016.

| Model | AFD + AFI | 95% Confidence Limits | Model | AFD + delay | 95% Confidence Limits | Model | AFI + delay | 95% Confidence Limits |
|-------|-----------|-----------------------|-------|------------|-----------------------|-------|-------------|-----------------------|
| AFD   |           |
| p-value | .034 | <.0001 | 1 lowest | 0.97 (0.77, 1.21) | 1.50 (1.28, 1.76) | 2 highest | 1 |
|       | 1 lowest | 0.97 (0.77, 1.21) | 1.50 (1.28, 1.76) | 2 | 1.06 (0.87, 1.30) | 1.42 (1.22, 1.66) | 3 | 0.89 (0.74, 1.07) | 1.06 (0.91, 1.24) | 4 highest | 1 |
| AFI   | <.0001 | <.0001 | 1 lowest | 1.72 (1.41, 2.09) | 1.72 (1.49, 1.99) | 2 | 1.48 (1.25, 1.75) | 1.57 (1.38, 1.79) | 3 | 1.36 (1.17, 1.57) | 1.32 (1.17, 1.50) | 4 highest | 1 |
| DELAY |        | 0.100 | 1 & 2** shortest | 1.28 (1.15, 1.43) | 1.04 (0.92, 1.16) | 3 | 1.31 (1.17, 1.47) | 1.16 (1.03, 1.31) | 4 longest | 1 |
| AIC   | 9844.16 | 9848.36 | 9841.47 | 9844.16 | 9848.36 | 9841.47 | 9844.16 | 9848.36 | 9841.47 |
| BIC   | 9904.95 | 9903.62 | 9896.73 | 9904.95 | 9903.62 | 9896.73 | 9904.95 | 9903.62 | 9896.73 |

**Adjusted for age group and sex. **Quartiles 1 and 2 are combined (53 percent reported the same age for AFD and AFI).
As a sensitivity analysis, the same systematic increase in average AFD was also found when excluding respondents whose age at first drink was greater than 25. This means that the telescoping effect is not an artefact created by respondents who have started drinking late in their life.

**Discussion**

This study set forth to explore the associations between retrospective reporting of the age of onset of alcohol drinking and of alcohol intoxication and risky drinking later in life. When the strength of AFD, AFI and delay as predictors of mean AUDIT score was assessed, all three were found to be individually associated with risky drinking, but in general, AFI was found to be the best predictor. Secondly, we compared absolutely and relatively defined onset indicators and found no additional benefit in the relative definition. Thirdly, the recall bias was found to be systematic across five waves of FDH-survey in 1984–2016 so that reported AFD increased with age among nine different birth cohorts, essentially attesting to a telescoping effect.

The finding concerning the association of AFD to risky drinking followed the findings from numerous previous
studies, where early AFD has been associated with heavier drinking and adverse outcomes later in life (Grant & Dawson, 1997; Guttmannova et al. 2011; Kuntsche et al. 2016). However, when the effect of AFI was accounted for, AFD no longer captured the excess risk from early onset of drinking. This means that AFI is more robust in capturing the association between early onset of drinking and subsequent risky drinking, and AFD would appear to be merely a proxy reflecting this relationship. It therefore seems that at least in a country like Finland that belongs to a group of countries where alcohol intoxication has an established place in the drinking culture (Sieroslawski et al. 2016) and where it is common for adolescents to have their first drink and get intoxicated for the first time on the same occasion, it is rather the timing of first intoxication than of first instance of drinking that matters for later risk of alcohol-related harm. In addition, the delay variable indicated that with a given age of intoxication onset, a long delay – i.e. an earlier onset of drinking without intoxication – was associated with a lower risk than a somewhat shorter delay.

This result adds to the growing evidence emphasising the need for alcohol research to shift focus from age at first drink to age at first intoxication (Kuntsche et al. 2016; Newton-Howes et al. 2019). Some studies on the theme of drinking with parents have pointed out similar implications compared to our results on delay: it has been found that infrequent (fewer than 3 times within 12 months) drinking with parents may not increase the likelihood of risky drinking for adolescents (Pape & Bye, 2017), while drinking unsupervised is especially risky (Gilligan et al. 2012). It is also established that alcohol-specific parenting reduces the risk of excessive use of alcohol by adolescents (Pape & Bye, 2017; Yap et al. 2017). Based on these results, it could be assumed that our results about a ‘protective’ effect of earlier age of drinking once the age of intoxication is adjusted for could be due to involvement of the family of the respondent. The result could also reflect cultural differences between families where the young have been exposed to alcoholic drinks without getting intoxicated and families where no such exposure has happened or where intoxication has soon followed.

To our knowledge, the present study was the first attempt to analyse drinking onset indicators in absolute and relative terms at the same time. As the association between AFD and risky drinking was found to be weaker, the analysis was focused on AFI. The absolute AFI and relative AFI (i.e. AFI centred to cohort’s median AFI) were compared in order to shed light on whether absolute biological age of onset matters more than the age of onset in relation to one’s birth cohort. The analysis showed that there was no real benefit of defining AFI in relative terms and the absolute AFI performed slightly better in model comparisons when combined with the delay variable. This result can be seen as suggestive against the ‘marker hypothesis’, according to which people with greater general vulnerability to problematic alcohol involvement, which could be due to, e.g. personality factors or upbringing, would be expected in any given cohort to be among the first ones to take up alcohol use. However, because there wasn’t very much variation in absolute and relative AFI in our data, this question warrants revisiting in future studies.

In our comparison of 11 birth cohorts across five repeated cross-sectional studies, the between-cohort changes showed that the average reported AFD decreased from one cohort to the next one until the trend plateaued with cohorts born between the 1960s and the mid-1980s and then increased again for the two youngest cohorts born between 1985 and 2000. This finding is in line with previous studies among Finnish youth, which have indicated a decline in youth drinking and a postponing of drinking onset (Lintonen et al. 2016; Raitasalo et al. 2018).

Our within-cohort results showed that the average reported AFD increased systematically with age for each cohort and that the increase was steepest in the younger age groups. It is not really possible that for a given cohort, the true average age of onset would systematically change (especially as sensitivity analysis checked that the result was unchanged if late ages of onset were excluded). Rather, the plausible explanation is recall bias, i.e. forward telescoping, or that the definition of what was a first drink changes over time. Some previous studies have shown that also at the individual level, the reported age of drinking onset has increased with age on average (Parra, O’Neill, and Sher 2003; Sartor et al. 2011; Shillington et al. 2012). While these studies had some limitations, i.e. relatively short follow-up time or a limited amount of participants, the present study was able to replicate the same result on the aggregate level with five repeated general population surveys covering a time frame of over three decades. Because the so-called forward telescoping affects practically all self-reporting of distant substance use (Janssen, Chessa, and Murre 2006; Shillington et al. 2012), the same limitation has to be considered when using alternative indicators for early age at onset, e.g. AFI and delay.

The present study had some limitations, which should be taken account of when interpreting the results. The measures of AFD and AFI were based on a subjective definition of first drink and first intoxication, which has been shown to be inferior to objective measures, such as first time drinking one drink or 5+ drinks (Morean et al. 2018). The waves of the FDH survey were cross-sectional and birth cohorts were represented by a different set of participants in each wave, which could potentially produce some bias compared to a situation where different cohorts of children could actually be followed over time. Lastly, we did not have information at our disposal on factors that could potentially confound or mediate the relationship between age of onset and later risky drinking, e.g. other substance use, maternal/paternal alcohol use, or externalising behaviours during adolescence. However, as the aim of the study was to compare the performance of AFD and AFI when predicting an AUDIT score, not to make statistical inference concerning individual pathways to adverse outcomes, the impact of the lacking confounding or mediating information is diminished. Additionally, if the association is due to individual factors like externalising behaviours rather than early age of onset per se, then one would expect the relative age of onset to be more important than the absolute age of onset; this
hypothesis was tested and there was no support (when delay was included) or no strong support (when it was not) for the idea that the relative age of onset would have a stronger connection to the AUDIT score than the absolute age of onset.

In conclusion, the present paper adds to the growing literature of critical remarks on using AFD as a predictor of subsequent risky drinking, and the results show that, for defining early onset of alcohol use, AFI has an advantage over AFD. For either measure, when using retrospective recall of age at onset, it needs to be borne in mind that when time passes, later ages of onset are reported. In addition, our results do not lend direct support for the marker hypothesis, i.e. to an idea that the association would be due to other factors causing vulnerability to problematic alcohol use. The implication of these results is that the mere act of tasting or having one drink is not as essential as postponing the first alcohol intoxication. Nevertheless, further studies will be needed to disentangle the causal factor in the association between early age at onset and later risky drinking, while taking into account confounding effects.

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