Published in final edited form as:

Drug Alcohol Depend. 2017 July 01; 176: 148–153. doi:10.1016/j.drugalcdep.2017.03.015.

Age of First Drunkenness and Risks for All-cause Mortality: A 27-year Follow-up from the Epidemiologic Catchment Area Study

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Abstract

Background—Early-onset drunkenness is associated with an increased risk of developing an alcohol use disorder (AUD), which predicts excess mortality risk. Here, we estimated mortality risk for drinkers with and without early drunkenness.

Methods—For 14,848 adult participants interviewed about drinking, drunken episodes, and AUD in 1981–83 for the Epidemiologic Catchment Area in New Haven (Connecticut), Baltimore (Maryland), St. Louis (Missouri), and Durham (North Carolina), we linked National Death Index records through 2007.

Results—Cox regression modeling estimates showed excess mortality for drinkers with age of first drunkenness earlier than 15 years old (hazard ratio, HR: 1.47, 95% CI: 1.25, 1.72) and when first drunkenness occurred at or after age 15 (HR: 1.20, 95% CI: 1.11, 1.29), as compared with adults who had never been drunk. Consistent results were observed, irrespective of AUD history. That is, early drunkenness signaled excess mortality risk even in absence of AUD.

Conclusions—In a large community sample from four cities in the US, early age of onset of drunkenness predicts mortality risk. We discuss experiments to investigate the possible causal significance of this predictive association.

Keywords

drunkenness; alcohol use disorder; mortality; epidemiologic catchment area program

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Human Participant Protection: The study was approved by the institutional review boards of the University of Florida and the Johns Hopkins Bloomberg School of Public Health. Michigan State University and Duke University Medical Center participations were judged exempt.

1. Introduction

Harmful drinking of alcoholic beverages (hereinafter, alcohol) is one of the most prevalent behavioral health problems in the world. Estimated to contribute to over 2.7 million deaths annually (Whiteford et al., 2013), the alcohol-mortality prediction often has a J-shape, with lower death rates for lighter drinking subgroups, relative to non-drinkers, and with excess mortality risk along a gradient toward heavier drinking and among subgroups with alcohol use disorders (AUD; (Mattisson et al., 2011; Ojesjö et al., 1998; Vaillant, 2003). In recent meta-analytic work that strengthens earlier summaries (Harris and Barraclough, 1998), a total of 81 observational studies with 221,683 observed deaths among 853,722 people suggested that the mortality rate among AUD cases might be an estimated three times greater than the non-AUD mortality rate (Roerecke and Rehm, 2013). Multiple background characteristic and associated covariates are thought to play a role in elevated AUD-related mortality rates. Especially prominent are acute consequences of AUD-related impairments and injury deaths (Vinson et al., 2003), and chronic health effects of drinking, such as cardiovascular diseases and cancers (French, 1971; Rehm et al., 2003; Rehm et al., 2009), as well as more occult pathways such as drinking-associated increased absorption of heavy metals (Hu et al., 2014; Newton et al., 1992).

Early onset of drinking and drunkenness also may be implicated since they are associated with AUD and alcohol-related health behaviors such as smoking, fighting, unplanned and unprotected sex, and low academic performance (Dawson et al., 2008; Grant et al., 2001; Hingson et al., 2003; Hingson et al., 2006; Kuntsche et al., 2013; Thomas et al., 2000). While most literature in the field focused on early onset of drinking, emerging evidence suggested that the association between early age of alcohol initiation and AUD does not reflect a causal relationship (Ystrom et al., 2014). Kuntsche et al. (2015) suggested to shift the focus of alcohol research away from infrequent, low-quantity drinking towards more detrimental drinking patterns.

For the present investigation, we wondered whether early age of first drunkenness (e.g., drinking enough to get drunk at <15 years) might modulate AUD-associated excess mortality risk, given observed links from early drunkenness and later AUD, and from AUD and excess mortality (Spiegler, 1993). Accordingly, we hypothesize that mortality rates will be greater for the subgroup with early-onset drunkenness, as compared with others, and we investigated whether this predictive relationship varied by AUD status. For new empirical estimates, we constructed a prospective cohort research design, and yoked drinking characteristics of participants in the United States National Institute of Mental Health Epidemiologic Catchment Area (US, NIMH, ECA) study, 1981–83, with the US death records through 2007. In addition, an exploratory mediation analysis was conducted to estimate possible effects of AUD in a causal pathway thought to link age of first drunkenness with occurrence of death. Of methodological note are two pertinent facts: (1) the ECA assessments assessed age of first drunkenness but not age of first drink, and (2) we have aggregated ‘never drinkers’ with the subgroup of drinkers who never got drunk. We note that detrimental drinking patterns such as drunkenness might be more appropriate predictors of death and other alcohol-related health consequences (Dawson et al., 2008; Kuntsche et al., 2015), but we return to this issue of age of first drink versus age of the first

drunken episode in our discussion section, as well as the aggregation of ‘never drinkers’ with ‘never drunk’ individuals.

2. Methods

2.1 Study population

The ECA study was designed in the late 1970s as a multi-site National Institute of Mental Health collaboration with five participating university-based research teams (Yale University, Johns Hopkins University, Washington University, Duke University, and University of California at Los Angeles), with probability sample surveys of nearby community residents initiated site-by-site in the early 1980s (Eaton WW, 1985). The California site did not retain identifiers required for NDI linkages, but it was possible to construct a prospective cohort study of 14,848 adult household residents at the other four sites, all of whom had baseline interview records on age at 1st drunkenness; 592 were excluded due to missing data on this alcohol variable.

2.2 Mortality assessment

The vital status of participants as of December 2007 was extracted from three data sources as described elsewhere (Eaton et al., 2013): 1) the NDI database that includes vital statistics data from the 50 states, 2) the Social Security Death Index, and 3) information obtained during the process of re-engaging subjects for follow-up surveys. Among the 14,848 participants in our analysis sample, 6,497 had died by the end of 2007. As in prior ECA research on death rates (Eaton et al., 2013), person-years of follow-up were derived with the date of baseline assessment as the origin on the time axis, and age in years served as the time scale (units: year) for survival analyses.

2.3 Assessment of Early-Onset Drunkenness

Drinking history and age of first drunken episode were assessed midway through the ECA baseline interview in the early 1980s, using standardized items with the NIMH Diagnostic Interview Schedule module used to assess alcohol use disorders (DIS; (Robins et al., 1981). The main item was “How old were you the first time you ever drank enough to get drunk?” Participants responding “Don’t know” (n=273) were asked: “Do you think it was before or after you were 15?” On this basis, we set age 15 as the cutoff point for drunkenness, and sorted participants into one of three groups: (1) never had a drunken episode, (2) got drunk for the first time before age 15, and (3) got drunk for the first time at or after age 15.

2.4 Covariates

Covariates under study included study site as well as demographic and socioeconomic characteristics assessed during the baseline DIS assessment, which included age at interview, sex, education (recoded as <high school, high school graduate or equivalent, some college, or college graduate), marital status (recoded as married, widowed, divorced/separated, or never married). Due to small numbers of other minority subgroups, we created a dummy-coded covariate for self-designated ‘Black’ versus ‘Non-black’ subgroups. Occupational status at baseline is from the Nam-Powers occupational status index,

categorized into quartiles. The index was calculated using income, occupation, and prestige ratings (Nam and Terrie, 1988).

A sensitivity analysis considered potential errors of inference due to uncontrolled manifestations of childhood conduct problems (CCP) with onset before age 15 years, which also might influence mortality rates and explain away any observed drunkenness-mortality association. Early CCP manifestations were assessed in a DIS module, including truancy, school suspension or expulsion, arrest, running away from home, lying, stealing, vandalism, poor grades, trouble at school, and starting fights, with an exploratory factor analysis (EFA) used to derive a rough index for this potentially confounding influence on excess mortality. A CCP item on early alcohol or other drug use was not included within the EFA in order to reduce complexity of interpretation of estimates from this sensitivity analysis.

2.5 Statistical analysis

Descriptive statistics such as person-years since baseline interview and the age-standardized mortality rate (per 1,000 person-years) were derived and are presented for the three drunkenness subgroups. The direct age-standardized mortality rate was calculated using the 2000 US standard population (Klein and Schoenborn, 2001). Kaplan-Meier life table analyses were conducted to estimate variations in survival curves for individuals with different ages of first drunkenness. Cox proportional hazards models were used to assess the relationship between age of first drunkenness and all-cause mortality. The modeling included three stages of covariate adjustment, including: 1) a non-adjusted model (with no covariates); 2) a minimally-adjusted model that controlled for age at baseline, sex, minority status, and site; and 3) a fully-adjusted model that additionally controlled for the other baseline characteristics. We also produced model-based estimates for mortality associated with early-onset drunkenness, stratified by AUD status.

Our exploratory mediation analysis was performed using the unified approach proposed by Lange et al. (Lange et al., 2012), which is based on the counterfactual framework. This approach allows decomposition of the total effect of early onset drunkenness on the overall mortality into a natural direct effect ($A \rightarrow O$ in Supplemental Figure 1) and a natural indirect effect through the mediator AUD ($A \rightarrow M \rightarrow O$ in Supplemental Figure 1). The unbiased estimates for the direct and indirect effects were obtained from weighted Cox regression on A , A^* (where A represents direct effects and A^* represents indirect effects) and confounders (C) using a duplicated dataset. The duplicated dataset was constructed by replicating the original dataset three times such that A^* takes three different possible values

of A through three replications. The weights were then determined by $W = \frac{P(M|A^*, C)}{P(M|A, C)}$, where $P(\cdot)$ was the predicted probability derived from a logistic regression of the mediator (M) on the exposure (A) and confounders (C). The hazard ratios for A and A^* from the weighted Cox model can then be used as an unbiased estimate of the natural direct and indirect effects, respectively. The total effect is the product of the natural direct and indirect effects, and the proportion mediated by M can be calculated by dividing the natural indirect effect using the total effect. The standard errors and confidence intervals were generated by

using 1,000 bootstrap replications in R. All statistical analyses were conducted using SAS 9.4 (Cary, NC) and R 3.1.3 (R Foundation for Statistical Computing, Vienna, Austria).

Sensitivity analyses were conducted to test the robustness of our results. Firstly, we used the EFA index to adjust for early-onset childhood conduct problems that might function as confounding variables (Niemelä et al., 2006). Secondly, a non-nested mixed-effects Cox model with varying intercept by site and birth year (<1900–1900–1919–1920–1939, or 1940) were conducted using the “coxme” package in R (Therneau, 2012), in order to account for variations between different sites and birth years as well as varying legal minimum drinking age regimens across these sites (Cheng and Anthony, 2016; Hanson and David, 2014),

3. Results

Table 1 is a display of characteristics of the 14,848 adult study participants at baseline interview by age at first drunkenness. In aggregate, 60 percent were female and 27.1% self-designated as ‘Black’. A total of 9,089 adults (61.2%) said they had been drunk, with most of them first getting drunk at or after age 15 (86.6%). Irrespective of age at the 1st drunken episode, most who had gotten drunk were between 18 and 44 years of age at the time of the baseline interview in the early 1980s. Among those with early-onset drunkenness, just over one-third qualified as AUD cases (37.4%), while only 0.5% of those who had never been drunk qualified as AUD cases. Males, as well as the subgroup of ‘never married’, were over-represented among individuals with early-onset drunkenness (Table 1).

Table 2 shows the person-years as calculated since baseline interview, as well as the estimated age-standardized mortality rate (per 1,000 person-years) during the 27-year follow-up period, stratified by age of first drunkenness (<15 years, 15 years, or never). For the subgroup with early onset of drunkenness, the estimated age-standardized mortality rate was 25.8 per 1,000 person years. For the subgroup with later onset of drunkenness, the estimate was 22.5 per 1,000 person years, and those who had never been drunk, it was 18.9 per 1,000 person years. Stratification by covariates disclosed a consistent pattern of larger age-standardized mortality rate estimates in the subgroup with an early onset of drunkenness, with one exception being the subgroup of AUD cases who had never been drunk.

Kaplan–Meier survival curves for the three drunkenness subgroups are shown in Figure 1. Survivorship was most favorable for the ‘never drunk’ subgroup, and was least favorable for the subgroup with early-onset drunkenness.

Table 3 presents hazard ratio estimates from the Cox proportional hazards models (HR). In the model with no covariates, an early age of first drunkenness predicted greater overall mortality rates (HR = 1.92; 95% CI = 1.72, 2.13), and in addition, there was excess mortality in the subgroup with onset of drunkenness at or after age 15 years, as compared with the ‘never drunk’ subgroup: (HR = 1.57; 95% CI = 1.49, 1.65). Weakened but generally consistent estimated effects were observed in the model with covariate control for age at baseline, sex, self-designation as Black, and site (e.g., HR for age of first drunkenness <15

years = 1.49; 95% CI = 1.33, 1.67). Estimates did not differ appreciably with more complete covariate-adjustments (with terms for these additional covariates: education, marital status, and occupational status). Supplemental Table 1 shows the effect estimates for all covariates.

In order to address possible modulation by AUD status, we re-estimated the hazard ratios for persons with and without AUD at baseline via models that included all of the above-listed covariates. This additional modeling showed that even among individuals without AUD, an early age of first drunkenness predicted greater overall mortality rates (HR = 1.46; 95% CI = 1.27, 1.69), and in addition, there was excess mortality in the subgroup with onset of drunkenness at or after age 15 years, as compared with the ‘never drunk’ subgroup: (HR = 1.21; 95% CI = 1.13, 1.29).

Exploratory Analyses

Our exploratory analyses addressed estimation of the possibility that AUD might function as an intermediate variable (M) on a causal pathway linking early-onset drunkenness (A) with the later mortality outcome (O), under an assumption that AUD post-dated the 1st drunken episode. Supplemental Table 2 displays estimates for the direct effect ($A \rightarrow O$ in Supplemental Figure 1) and indirect effect ($A \rightarrow M \rightarrow O$ in Supplemental Figure 1), and provides an estimated proportion of effects mediated by AUD (assuming the model specifications are correct). For those with earlier age of first drunkenness (<15 years), the estimated total HR of 1.47 (95% CI = 1.25, 1.72) was decomposed into a direct HR estimate of 1.36 (95% CI = 1.14, 1.60) and an indirect HR of 1.09 (95% CI = 1.05, 1.13), with an estimated 21% (95% CI = 11%, 42%) of the total effects mediated through AUD. For comparative purposes, these effect estimates were derived for persons with later age of first drunkenness (≥ 15 years): Estimated total HR = 1.20 (95% CI = 1.11, 1.29); Estimated direct HR = 1.16 (95% CI = 1.07, 1.26); Estimated indirect HR = 1.04 (95% CI = 1.02, 1.05); Estimated proportion mediated = 0.20 (95% CI = 0.09, 0.42).

Sensitivity Analyses

Our sensitivity analyses addressed potential confounding by level of childhood conduct problems, as well as site and birth year variations (via non-nested mixed effect Cox models). Supplemental Figure 2 shows the EFA results used to derive the CCP index (e.g., scree plot). Supplemental Table 3 offers a comparison of results from the main analyses and these sensitivity analyses to address CCP. The overall conclusion is one of no appreciable variation in estimates when statistical adjustment is made for CCP, and when variations attributable to site and to birth year are taken into account.

4. Discussion

In this study, we found that drunkenness, earlier than mid-adolescence, or later than mid-adolescence, is a predictor of excess mortality, even among drinkers who did not qualify as alcohol use disorder cases at baseline assessment. The estimated effect of early-onset drunkenness (before age 15 years) was numerically larger than the estimated effect of later-onset drunkenness, but the 95% confidence intervals for these estimates showed considerable overlap. Our exploratory mediation analyses designed to decompose the

estimated total drunkenness-mortality association into estimates of direct and indirect effects (mediated by AUD) suggest a somewhat larger indirect effect via intermediate AUD when the pathway from early-onset drunkenness is being studied and somewhat smaller indirect effects when the pathway from later-onset drunkenness is being studied. Sensitivity analyses did not contradict these general conclusions.

This study's findings on the drunkenness-mortality association are novel, but tend to be consistent with prior evidence on other outcomes. For example, age of first drunkenness has been linked to an array of risky behaviors and drug problems, including AUD (Hingson et al., 2003; Kuntsche et al., 2013), and AUD is known to be associated with excess mortality (Roerecke and Rehm, 2013). It is of interest that our primary estimates for the earlier-onset drunkenness subgroup did not differ appreciably from our primary estimates for the later-onset drunkenness subgroups. It is possible that the age 15 years cutoff point is not sufficiently discriminating. An earlier age of first drunkenness might provide greater discrimination, but exploration of these variations will require larger sample sizes. We also note that the aggregation of 'never drinkers' with the 'never drunk' subgroup might have been sub-optimal, but the baseline DIS interview data did not permit disaggregation.

In our primary analyses, stratification by AUD showed drunkenness-mortality predictions for persons without AUD, but not for AUD cases. It occurred to us that AUD, as a composite diagnostic variable, might be a source of variation that functions to absorb any variation attributable to early drunkenness. Nonetheless, our exploratory analysis of mediational pathways suggests incompleteness in this respect, and there remained a tangible estimated drunkenness-mortality prediction even when the indirect AUD pathway was taken into account. This topic can be pursued in a future line of investigation in which early-onset AUD is decomposed into its individual constituent parts (e.g., item-sets that tap individual diagnostic criteria of AUD), with early-onset drunkenness as an inter-correlated (but not identical) manifestation of alcohol problems that develop before late adolescence. A recent example of this type of investigation, and its utility, can be found in Cheng et al. (2016) who applied this type of composite indicator approach in order to investigate which specific antioxidants or antioxidant combinations might be accounting for reduced C-reactive protein levels. The same can be done for the individual constituent parts of early-onset alcohol problems in order to clarify the predictive relationships discovered in this initial investigation.

A note about age at first drunken episode versus age at first drink is in order, given a growing body of literature on age at first drink and the potential complications of early-onset first drink (DeWit et al., 2000; Grant et al., 2001; Hingson et al., 2006; Thomas et al., 2000). Age at first drunkenness was studied by us for several reasons. First, since the DIS used in the ECA program assessed age of first drunkenness but not age of first drink, we were not able to evaluate the possibility of differential effects between age of first drink versus age of first drunkenness. Second, as Dawson and colleagues have suggested (Dawson et al., 2008), the effects of early drinking might be present only for drinkers who at consumed alcohol to the point of intoxication and becoming drunk, but not among those who consume no more than a sip (or even a full drink over the course of a meal) and for whom there is no palpable sensation of intoxication. It is possible that using small quantities of alcohol at an early age,

without intoxication, is an inert experience with respect to later risk of dying, unless the alcohol consumption involves intoxication and drunkenness. More recently, Kuntsche et al. (2015) have argued that alcohol research might usefully shift towards more detrimental drinking patterns, in complement with or instead of a focus on ‘any drinking’.

Counterbalanced with potential weaknesses along the lines just discussed, our study had several strengths. First, the ECA data provided us with an unusual opportunity to estimate variation in mortality rates as might be predicted by age of first drunkenness as measured in community probability samples of adults during the course of a 27-year follow-up interval. Second, statistical adjustments for multiple covariates were made part of the estimation approach, and the result is a more complete representation of the potential hypothesized effects of early-onset (and later-onset) drunkenness. Third, the exploratory analyses help to strengthen the body of evidence, including statistical adjustment for childhood conduct problems, and modeling of site-related variations, as well as the use of mediation analyses to estimate possible direct effects of age of first drunkenness on mortality as well as indirect effect pathways with mediation via AUD.

Of course, this study’s estimates, including estimates from our exploratory mediation analyses, must be interpreted with some caution. AUD case status measured at baseline can be regarded as an incompletely measured time-varying characteristic in that some ECA participants might have become AUD cases after baseline and before death, and they might have become drunk for the first time after baseline. In this study’s analyses, these post-baseline AUD cases are included in the subgroup of individuals with no AUD, and those with post-baseline drunkenness are counted in the ‘never drunk’ subgroup at baseline. Lopez-Quintero and colleagues (2015) have mentioned this type of scenario as a ‘Len Bias’ bias that must be faced in prospective cohort studies of alcohol and other drug-related mortality unless there is continuous time surveillance and monitoring of the alcohol and other drug exposures throughout the follow-up interval. Because continuous time surveillance and monitoring of alcohol and other drug exposures may never be achieved in prospective studies of long duration, we judge this caution as one that will apply generally in studies of drunkenness and AUD-associated mortality. Any future exceptions will be quite noteworthy. In addition, recall bias may exist for age of onset. However, it is likely to be a non-differential bias, thus leading the estimates towards null.

We also should clarify that this study’s estimates of direct and indirect effects with respect to mortality rates for subgroups with age of first drunkenness <15 years versus ≥15 years might be biased toward the null, given that early age of first drunkenness is associated with greater incidence of AUD (Hingson et al., 2003; Kuntsche et al., 2013). We draw attention to our finding that evidence of a somewhat stronger indirect effect can be seen among those with early-onset drunkenness as compared to those with late-onset drunkenness.

Finally, we note that the causal significance of the observed prediction from early-onset drunkenness (or later-onset drunkenness) to mortality risk now remains uncertain, even though our modeling has included statistical adjustments for multiple suspected confounding variables. In order to secure definitive evidence on the suspected cause-effect relationship that might link drunkenness with excess mortality, intervention experiments might be

required. In this context, it will not be possible to assign drunkenness at random, but it might be possible to concoct an intervention that discourages drunkenness and promotes alternative drinking patterns. Initial phases of the experiment might focus upon near-term drunkenness consequences (e.g., occurrence of risky behaviors such as unsafe sex or driving under the influence), with appropriate analyses when we can ‘encourage’ but not mandate desired health behaviors (Mock et al., 2005). Even if it is not feasible to extend this line of experimentation long enough, or with large enough samples, for investigation of death outcomes, the evidence from these intervention experiments can be yoked with replications of this prospective cohort study of mortality in order to create chains of inference about cause-effect relationships that might link early-onset drunkenness with this ultimately absorbing public health consequence of vital importance.

5. Conclusion

In this prospective cohort study of a probability sample of adult community respondents recruited from four US cities in the early 1980s, a baseline history of lifetime drunkenness predicted excess mortality, with marginally higher rates for subgroups of drinkers whose first episode of being drunk occurred before age 15. In addition, exploratory mediational analyses are suggestive of a possibility that an indirect mediational pathway, via AUD, might be substantively more important for the earlier-onset drunkenness subgroup than for the later-onset drunkenness subgroup. Intervention experiments might be used to disentangle these hypothesized direct and indirect effects, as well as the causal significance of what can be seen as a predictive association that links becoming drunk earlier in life with later mortality risk.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding for this study was provided by the National Institute on Drug Abuse (grants R01 DA026652, T32 DA021129, T32 DA035167, and K01 DA015799)

List of abbreviations

AUD	Alcohol use disorder
CCP	Childhood conduct problems
CI s	Confidence intervals
ECA	Epidemiologic Catchment Area
EFA	Exploratory factor analysis
HR s	Hazard ratios
NDI	National Death Index

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Highlights

- Getting drunk predicts excess mortality risk, relative to individuals with no drunkenness episode.
- Those who first got drunk before age 15 had an even higher mortality risk.
- Long-term intervention experiments may be required to examine whether early drunkenness is a cause of excess mortality risk.

Figure 1.

Kaplan-Meier survival curve estimates for onset of drunkenness. Data from household survey samples at four Epidemiologic Catchment Area Study sites, with baseline assessments in 1981-1983 and mortality follow-up through 2007 ($n = 14,848$).

Table 1

Selected characteristics at baseline interview by drunkenness categories. Data from household survey samples at four Epidemiologic Catchment Area Study sites, with baseline assessments in 1981–1983 and mortality follow-up through 2007 (n=14,848).

Onset of Drunkenness	<15 Years (n=1,214)	15 Years (n=7,875)	Never (n=5,759)	All Persons (n=14,848)
Selected Characteristics at Baseline Interview	Unweighted n (%)	Unweighted n (%)	Unweighted n (%)	Unweighted n (%)
Sex				
Male	820 (67.5)	3,942 (50.1)	1,131 (19.6)	5,893 (39.7)
Female	394 (32.5)	3,933 (49.9)	4,628 (80.4)	8,955 (60.3)
Minority Status				
Non-Black	847 (69.8)	5,710 (72.5)	4,260 (74.0)	10,817 (72.9)
Black	367 (30.2)	2,160 (27.4)	1,490 (26.0)	4,017 (27.1)
Age				
18–24 years	345 (28.4)	1,033 (13.1)	380 (6.6)	1,758 (11.8)
25–34 years	348 (28.7)	2,122 (26.9)	558 (9.7)	3,028 (20.4)
35–44 years	127 (10.5)	1,205 (15.3)	475 (8.2)	1,807 (12.2)
45–64 years	206 (17.0)	1,765 (22.4)	1,370 (23.8)	3,341 (22.5)
65 years	188 (15.5)	1,750 (22.2)	2,976 (51.7)	4,914 (33.1)
Education				
<High school	511 (42.1)	2,743 (34.8)	2,810 (48.8)	6,064 (40.9)
High school graduate	342 (28.2)	2,406 (30.6)	1,620 (28.1)	4,368 (29.5)
Some college	187 (15.4)	1,131 (14.4)	589 (10.2)	1,907 (12.9)
College graduate	170 (14.0)	1,584 (20.1)	727 (12.6)	2,481 (16.7)
Marital Status				
Married	512 (42.2)	4,078 (51.8)	2,516 (43.7)	7,106 (47.9)
Widowed	92 (7.6)	826 (10.5)	1,915 (33.3)	2,833 (19.1)
Divorced/separated	211 (17.4)	1,351 (17.2)	560 (9.7)	2,122 (14.3)
Never married	399 (32.9)	1,617 (20.5)	766 (13.3)	2,782 (18.7)
Nam-Powers Occupational Status Index				
Quartile 1	303 (25.0)	1,490 (18.9)	1,626(28.2)	3,419 (24.7)
Quartile 2	426 (35.1)	2,776 (35.3)	1,939(33.7)	5,141 (37.1)
Quartile 3	282 (23.2)	1,879 (23.9)	970(16.8)	3,131 (22.6)
Quartile 4	141 (11.6)	1,380 (17.5)	653(11.3)	2,174 (15.7)
Alcohol Use Disorder ^a				
Yes	453 (37.4)	1,100 (14.0)	29 (0.6)	1,582 (10.7)
No	758 (62.6)	6,760 (86.0)	5,727 (99.4)	13,245 (89.3)
Site				
New Haven, Connecticut	328 (27.0)	2,600 (33.0)	1,960 (34.0)	4,888 (32.9)
Baltimore, Maryland	337 (27.8)	1,864 (23.7)	994 (17.3)	3,195 (21.5)
St. Louis, Missouri	312 (25.7)	1,771 (22.5)	876 (15.2)	2,959 (19.9)
Durham, North Carolina	237 (19.5)	1,640 (20.8)	1,929 (33.5)	3,806 (25.6)

^aDSM-III diagnoses at baseline

Due to rounding, some of the column-wise percentages do not add to 100%.

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Table 2

Person-years and age-standardized mortality rate by drunkenness categories and selected baseline characteristics. Data from household survey samples at four Epidemiologic Catchment Area Study sites, with baseline assessments in 1981–1983 and mortality follow-up through 2007 (n=14,848).

Onset of Drunkenness	<15 Years (n=1,214)		15 Years (n=7,875)		Never (n=5,759)	
	Person-years ^a	Mortality Rate ^b	Person-years ^a	Mortality Rate ^b	Person-years ^a	Mortality Rate ^b
Total	26,667	25.8	167,980	22.5	103,929	18.9
Sex						
Male	17,447	27.4	78,280	25.6	19,539	22.5
Female	9,220	23.0	89,700	18.8	84,390	18.1
Minority Status						
Nonblack	18,905	26.8	120,680	22.4	72,935	19.0
Black	7,762	24.7	47,235	21.9	30,879	16.9
Education						
<High school	9,787	27.0	49,065	26.2	44,279	21.1
High school graduate	7,999	29.7	54,966	20.9	32,900	17.9
Some college	4,565	23.1	26,042	21.7	11,987	16.7
College graduate	4,216	18.5	37,693	17.6	14,592	17.1
Marital Status						
Married	10,971	25.9	87,287	20.8	49,727	17.2
Widowed	1,342	29.6	11,619	25.1	26,344	23.6
Divorced/separated	4,632	23.7	29,737	23.1	11,502	17.8
Never married	9,722	28.2	39,281	26.1	16,330	20.2
Nam-Powers Occupational Status Index						
Quartile 1	6,350	28.3	30,393	23.4	27,944	19.9
Quartile 2	9,367	28.3	59,021	23.5	35,937	19.6
Quartile 3	6,167	24.3	39,753	22.6	17,569	18.3
Quartile 4	3,268	25.1	31,144	20.1	12,519	17.1
Alcohol Use Disorder ^c						
Yes	9,822	26.7	22,298	29.2	466	31.2
No	16,795	25.2	145,402	21.5	103,384	18.8
Site						

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Onset of Drunkenness	<15 Years (n=1,214)		15 Years (n=7,875)		Never (n=5,759)	
	Person-years ^a	Mortality Rate ^b	Person-years ^a	Mortality Rate ^b	Person-years ^a	Mortality Rate ^b
New Haven, Connecticut	7,149	28.9	52,684	21.4	30,614	18.6
Baltimore, Maryland	7,446	33.0	40,586	27.3	18,613	24.5
St. Louis, Missouri	7,177	18.3	39,703	22.5	17,275	19.7
Durham, North Carolina	4,895	23.0	35,007	18.8	37,427	15.2

^aPerson-years since baseline interview.

^bPer 1,000 person-years, age-standardized.

^cDSM-III diagnosis at baseline.

Table 3

Estimated hazard ratios for prediction from onset of drunkenness to time of death. Data from household survey samples at four Epidemiologic Catchment Area Study sites, with baseline assessments in 1981–1983 and mortality follow-up through 2007 (n=14,848).

Onset of Drunkenness	Non-adjusted Model	Minimally-adjusted Model ^a	Final-adjusted Model ^b
<i>Unstratified:</i>			
Never	Reference	Reference	Reference
Onset <15 years	1.92 (1.72, 2.13)	1.49 (1.33, 1.67)	1.47 (1.25, 1.72)
Onset 15 years	1.57 (1.49, 1.65)	1.23 (1.16, 1.31)	1.20 (1.11, 1.29)
<i>Stratified: AUD Cases</i>			
Never	Reference	Reference	Reference
Onset <15 years	1.17 (0.73, 1.90)	1.13 (0.70, 1.84)	1.21 (0.74, 2.00)
Onset 15 years	1.11 (0.70, 1.76)	1.07 (0.67, 1.70)	1.06 (0.66, 1.71)
<i>Stratified: AUD Non-Cases</i>			
Never	Reference	Reference	Reference
Onset <15 years	1.80 (1.57, 2.05)	1.46 (1.28, 1.68)	1.46 (1.27, 1.69)
Onset 15 years	1.51 (1.43, 1.59)	1.21 (1.13, 1.28)	1.21 (1.13, 1.29)

^aReference subgroup for all models = 'never drunk' as of baseline. Adjusted for age at baseline, sex, minority status, and site.

^bAdjusted for age at baseline, sex, minority status, site, as well as education at baseline, marital status at baseline, and occupational status at baseline.