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The Effects of Age at Drinking Onset and Stressful Life Events on Alcohol Use in Adulthood: A Replication and Extension Using a Population-Based Twin Sample

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Abstract

Background—A study by Dawson and colleagues (*Alcohol Clin Exp Res* 2007; 31:69) using data from National Epidemiologic Survey on Alcohol and Related Condition found earlier drinking onset age, and higher levels of past-year stressful life events (SLE) were associated with higher past-year alcohol consumption. The aims of our study were as follows: (i) to attempt to replicate this interaction; (ii) to extend it by examining sex and event dependence as potential moderators of the effect; and (iii) to estimate the roles of genetic and environmental factors in mediating the overlap of early drinking onset and SLE in their relations with alcohol consumption.

Methods—Data were from 1,382 female and 2,218 male drinkers interviewed as part of the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders. Regression models were used to evaluate the main and interactive effects of early drinking onset and moderate or severe past-year SLE on past-year drinking density (PYDD), a weighted quantity-frequency measure of alcohol consumption. Analyses adjusted for demographic covariates and were stratified by sex and whether SLE were independent or dependent on the person's actions, as rated by interviewers. Structural twin models were used to estimate the degree to which early drinking onset, SLE, and their interaction accounted for additive genetic, common environmental and individual-specific variance in PYDD.

Results—We replicated the prior finding of a main effect of higher alcohol consumption among individuals reporting earlier drinking onset. Age at drinking onset accounted for about 5% of the variation in PYDD, and this association was mostly attributable to overlapping genetic influences. Evidence for an interaction between onset age and SLE was generally weak, possibly because of lower power and other methodological differences from Dawson and colleagues' study. However, there was some evidence consistent with an interaction of higher PYDD among early drinking men who experienced independent SLE and early drinking women with dependent SLE.

Conclusions—We confirmed prior findings of an association between early age at drinking onset with higher past-year drinking among young- and middle-aged adults and found limited evidence supporting a replication for higher stress-related drinking among early-onset drinkers. The association is consistent with early onset and stress-related drinking being attributable to overlapping genetic liability. Among early drinkers, our results suggest sex differences in consumption with regard to event dependence.

Keywords

Age at First Drink; Stressful Life Events; Early-Onset Drinking; Twins; Sex Differences

Substantial evidence supports the association of early age at drinking onset with negative alcohol-related outcomes. Based on data from the National Longitudinal Alcohol Epidemiologic Survey (NLAES), Grant and Dawson (1997) established that individuals who reported an early age of drinking onset were much more likely to meet criteria for lifetime alcohol abuse and dependence. In the NLAES, more than 40% of individuals who initiated drinking at or before age 14 had alcohol dependence, compared with fewer than 17% of individuals who began drinking at age 18 or older. The association between early drinking onset and elevated rates of negative alcohol outcomes was subsequently replicated in a number of cross-sectional (Prescott and Kendler, 1999; Reboussin et al., 2006; York et al., 2004) and prospective studies (e.g., Buchmann et al., 2009; Pedersen and Skrandal, 1998; Pitkänen et al., 2005). Findings from experimental animal studies also support the relation of early drinking onset with increased drinking behaviors in adulthood (Maldonado-Devincci et al., 2010; Yoshimoto et al., 2002), suggesting that early alcohol exposure may trigger biological changes that subsequently increase one's susceptibility to problematic drinking.

A large volume of research has been conducted to identify the mechanisms underlying the association of age at drinking onset with subsequent drinking behaviors. Early drinking onset has been found to independently predict greater drinking in early- and mid-adulthood, even after adjusting for multiple familial and behavioral antecedents in childhood and adolescence (Buchmann et al., 2009; Grant, 1998; Hingson et al., 2000, 2001, 2003; Pitkänen et al., 2008). Early drinking onset is part of a cluster of factors associated with problematic alcohol use, including externalizing tendencies (e.g., aggressiveness, low prosociality), physiological signs of poor inhibition, early use of other substances (McGue et al., 2001a), adverse childhood experiences (Dube et al., 2006), early parental onset of substance use, parenting style marked by low involvement and physical punishment, poor relationship between parents (McGue et al., 2001b; Pitkänen et al., 2008), and the presence of alcoholic adult siblings (Kuperman et al., 2005). Prospective studies have identified a number of predictors of early drinking onset, including aggressiveness, oppositionality, hyperactivity/impulsivity, inattentiveness, conduct problems, low self-control in early adolescence, prior drug use, family history of alcohol use disorders, blue-collar family background (unique to girls), and being male (McGue et al., 2001a; Pitkänen et al., 2005; Trim et al., 2010). High levels of regular and pathological alcohol consumption, substance use disorders (e.g., Dawson et al., 2008; McGue et al., 2001a,b; Pitkänen et al., 2005, 2008), and other risky behaviors (e.g., fights, unprotected sex, drinking-related injuries; Hingson et al., 2000, 2001, 2003) are among the negative outcomes linked to adolescents who exhibit delinquent, norm-violating behaviors (e.g., early drinking, illicit drug use) and have attitudinal (e.g., low academic orientation) and environmental risk factors (e.g., low parental control) according to the problem behavior theory (Jessor, 1991; Jessor and Jessor, 1977).

Some early studies of the onset age–alcohol use disorder association (Grant and Dawson, 1997; Prescott and Kendler, 1999) employed retrospective reporting of onset age and symptomatology, raising the possibility that some of the association was because of response biases. Recall of onset ages is subject to “telescoping bias,” whereby older individuals recollect events as occurring closer in time (i.e., at later onset) than younger individuals (Johnson and Schultz, 2005). If the recall of alcohol-related problems is also subject to an interval-dependent reporting bias, it is possible that the early-onset problem-drinking association is spuriously inflated (i.e., by younger individuals reporting both more symptoms and earlier onset ages). However, this concern is reduced by the replication of the

association between early onset and problematic use in several studies using prospective longitudinal designs (e.g., Buchmann et al., 2009; Pedersen and Skrandal, 1998; Pitkänen et al., 2005).

Stress and Alcohol Consumption

Stressful life events (SLE) are considered a proximal trigger of drinking. The “self-medication” or tension reduction hypothesis posits that drinking behavior is reinforced through its tension- and fear-reduction properties (Cappell and Herman, 1972; Greenley and Oei, 1999; Sayette, 1999). Other theories that have sought to explain the stress/alcohol connection include the *stressor-vulnerability model* by Cooper and colleagues (1992) and the *stress response dampening model* by Sher and Levenson (1982). Empirical findings generally support a positive association between stress and increased alcohol use, but the aggregate effect is small and seems to be modified by other factors. This large literature is reviewed by Pohorecky (1991) and Veenstra and colleagues (2006).

Relation of Age at Drinking Onset and Stress-Related Drinking

Early age at drinking onset and SLE have both been established as antecedents of increased alcohol consumption, but few papers have described their joint effects. Research has emphasized coping strategies and resources as key determinants of stress response (e.g., Veenstra et al., 2006, 2007), and drinking to cope may be a salient coping strategy among early initiators (Buchmann et al., 2010). Studies also support a greater role of genetic influence on alcohol use disorders and transition time from drinking onset to age at alcohol dependence diagnosis among early initiators compared with late-onset drinkers (Liu et al., 2004; McGue et al., 1992), suggesting higher genetic risk for alcohol use disorders in early drinkers. Early initiators may find the physiological effects of alcohol particularly reinforcing and therefore might be prone to use drinking as a coping response to stressors. Additionally, given the correlation of early drinking onset with familial history of alcoholism and parental alcohol use (Dawson et al., 2008), early initiators may have adopted drinking as a coping strategy through imitation and easy access to alcohol. Consistent with the problem behavior theory, early initiators are more likely to engage in antisocial and risky behaviors and tend to drink more frequently and encounter more negative consequences of deviant behaviors than late initiators.

There is also some evidence from experimental animal studies that early alcohol exposure could have a direct effect on later tendencies to use alcohol in response to stress. Male and female rodents administered alcohol during adolescence had greater stress-reactive drinking than those whose first access to alcohol was in adulthood (Füllgrabe et al., 2007; Siegmund et al., 2005). Dawson and colleagues (2007) replicated this interaction between early drinking onset and stress on alcohol consumption among humans using data from the National Epidemiologic Survey on Alcohol and Related Condition (NESARC). Individuals who reported they began drinking by age 14 and experienced past-year life stressors had higher alcohol consumption compared with early drinkers without past-year life stressors and individuals who began drinking later.

The primary goal of our study was to attempt to replicate Dawson and colleagues' (2007) finding of drinking onset as a moderator of the association between SLE and alcohol consumption. We also extended their analyses in 3 ways. First, the abundant examples of sex differences in stress-related drinking behaviors (e.g., Brennan et al., 1999; Perreira and Sloan, 2001; c.f. Veenstra et al., 2007) prompted us to examine the role of sex in this association. Second, we directly investigated whether the association of drinking onset with stress-related drinking would vary depending on the degree to which an individual's behavior contributed to the occurrence of SLE. Third, because we studied a twin sample, we

were able to employ biometric analyses to explore the roles of genetic and environmental factors in mediating the overlap of drinking onset and SLE in their relations with alcohol consumption.

MATERIALS AND METHODS

Sample

The sample for this study was derived from the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders (VATSPSUD), a longitudinal study of genetic and environmental risk factors for common adult psychiatric disorders (see Kendler and Prescott, 2006). Participants were identified through the Virginia Twin Registry (now part of the Mid-Atlantic Twin Registry) by matching birth certificate information of multiple births to contact information in state records.

VATSPSUD includes data collected from Caucasian twin pairs born between 1934 and 1974. Details on ascertainment and data collection procedures have been described previously (Prescott and Kendler, 1999; Prescott et al., 2004) and are not elaborated here. The current study is based on data obtained at the most recent wave of interviews, which is wave 4 in the female–female sample (FF4) and wave 2 in the male–male and male–female sample (MF2). We used data from the FF4 and MF2 waves because they provide detailed SLE data on the largest number of individuals. The structure of the FF4 and MF2 interviews for assessing SLE was slightly different (see Measures), so the SLE prevalence from the two may not be directly comparable. We thus conducted all analyses separately by sex and excluded female twins in MF pairs, because we could not validly equate the SLE means and variances of the 2 female groups as would be required if we included MF pairs in the twin analyses.

Given our interest in studying past-year alcohol use, we excluded from the current analyses individuals who reported that they abstained from alcohol use in the year prior to the interview (FF4 past-year abstainers: $N = 785$, 36.2%; MF2 past-year abstainers: $N = 708$, 24.4%). Individual-level analyses were based on 1,382 female twins in FF4 and 2,218 male twins in MF2. FF4 twin-pair analyses were based on data from 480 complete twin pairs (294 monozygotic [MZ] pairs and 186 dizygotic [DZ] pairs) and 417 twins (258 MZ and 159 DZ) without cotwin participation. One twin pair of unknown zygosity and 3 individuals from triplets were excluded from FF4 twin-level analyses. MF2 twin-pair analyses were based on 763 complete pairs (465 MZ pairs and 298 DZ pairs) and 686 twins without cotwin participation (459 MZ and 227 DZ); 6 individuals from triplets were excluded. Zygosity classification is estimated to be >95% accurate (Kendler and Prescott, 2006). Demographic characteristics are summarized in Table 1.

Measures

Drinking Onset (ONS14, ONS17)—Drinking onset was assessed with the question, “How old were you the first time you had a drink?” A drink was defined as a full drink (12 oz beer, 5 oz wine, or 1.5 oz spirits). To allow for comparison with Dawson and colleagues (2007), drinking onset was categorized into 3 groups: young (age ≤ 14), middle (age 15 to 17), and older onset (age ≥ 18). To allow a nonlinear effect of onset age, we used 2 dummy variables in the analyses: ONS17 indicated onset by age 17 (1 = onset age ≤ 17 , 0 = onset age > 17), ONS14 indicated onset by age 14 (1 = onset age ≤ 14 , 0 = onset age > 14). Using these codes allowed us to first estimate the effect of drinking by age 17 and then estimate whether there was a significant additional effect associated with drinking by age 14.

Past-Year Drinking Density—Drinking density was a weighted quantity/frequency measure representing the average number of drinks consumed per week in the past year. Participants reported the number of drinks consumed on (i) any single day when the largest number of drinks were consumed and (ii) a typical day when they drank in the past year. Interviewers grouped the responses into *quantity categories*: “1 to 3,” “4 to 6,” “7 to 9,” “10 to 12,” and “13 or more” drinks. Next, participants reported the number of days they consumed a drink in a typical month in the past year. Using a provided list of *frequency categories* ranging from never to every day, participants indicated the frequency categories that they consumed their maximum number of drinks and for using alcohol at 1 *quantity category* lower than their past-year maximum quantity. The latter served as an intermediate measure between their maximum and typical drinking days.

Using this information, we calculated the number of drinks consumed on days when a participant had (i) the largest number of drinks, (ii) an intermediate number of drinks, and (iii) a typical number of drinks. For calculating frequency values, we used the following equivalents based on taking the midpoint of the range and converting it into days per month: “every day” = $(7 \times 52)/12$, “5 to 6 times/wk” = $(5.5 \times 52)/12$, “3 to 4 times/wk” = $(3.5 \times 52)/12$, “1 to 2 times/wk” = $(1.5 \times 52)/12$, “2 to 3 times/month” = 2.5, “1 time/month” = 1, “7 to 11 times/y” = $9/12$, “3 to 6 times/y” = $4.5/12$, “2 times/y” = $2/12$, “once/y” = $1/12$ and “never” = 0. For calculating quantity values, we used the typical and maximum reported by the participant. The intermediate was the midpoint of the quantity category one lower than the maximum (e.g., 5 if their maximum drinks were 7). Individuals who never drank more than their “typical” amount were assigned frequencies of 0 for the other categories. Weighting these 3 quantity estimates by their frequencies produced an estimate of the average number of drinks consumed per month, which we term past-year drinking density (PYDD). This variable provides a more accurate estimate than a simple quantity $\times$ frequency measure. PYDD was subsequently converted to a weekly estimate, truncated at 168 (i.e., 24 drinks/d), and then log-transformed to adjust for skewness arising from a small number of individuals who reported consuming extremely large quantities of alcohol.

Stressful Life Events—Participants were asked about the occurrence of 15 categories of SLE during the 12 months prior to the interview. These included assault, robbery, legal problems, major financial problems, serious housing problems, job loss, serious difficulties at work, serious illness or injury, serious marital problems, divorce/separation, loss of confidant, interpersonal conflict, serious personal crisis experienced by someone in the network, serious illness of someone in the network, and death of an individual in the network. Network questions were repeated for multiple individuals (cotwin, parent, children, spouse/partner, siblings, other relatives, and close friends). Owing to modifications in interview length and structure between FF4 (a telephone interview) and MF2 (a longer, in-person interview), the actual number of SLE questions asked in the 2 samples was different: 42 for FF4 and 70 for MF2. Responses to MF2 questions reflecting the same event were collapsed to be comparable with the corresponding item in FF4 (e.g., “separated in last 12 months” and “decide to separate for 2 weeks or more from your wife/husband/partner” in MF2 were treated as equivalent to “marital separation” in FF4).

Long-Term Contextual Threat—During the interviews, interviewers rated each SLE’s impact based on the long-term contextual threat (LTCT) concept developed by Brown (1989) using a 4-point rating: minor, low-moderate, high-moderate, and severe. “Long-term” threat was defined as lasting 10 to 14 days or longer after the event, and only such events received a high-moderate or severe rating. “Contextual” was defined as what most people would feel in response to the event given one’s unique history and circumstances. Thus, LTCT ratings were designed to be objective measures of stress and not to reflect participants’ reactions or symptoms that developed after event occurrence. They were found

to have moderate interrater reliability ($r_s = +0.69$) and test–retest reliability ($r_s = +0.60$) over a mean period of 4 weeks (see Kendler et al., 1998). In the current analyses, we included only SLE with high-moderate or severe LTCT ratings (e.g., death of a parent) to focus on severe events that would likely have a longer-term impact on drinking than events with minor or low-moderate LTCT ratings (e.g., problems getting along with a neighbor, serious personal crisis of a sibling). It should be noted, however, that the LTCT rating for each SLE varied among participants based on the impact of the event on each participant given his/her unique circumstances.

Given the small proportion of the sample who reported having 4 or more past-year SLE that were rated moderate or severe (3.6% in women, 3.0% in men), we recoded SLE to have a maximum value of 3.

Event Dependence Versus Independence—Following Brown’s (1989) method, interviewers also rated whether each SLE was, “clearly independent,” “probably independent,” “probably dependent,” or “clearly dependent” on the participant’s behavior. Interrater and test–retest reliabilities for these ratings were $r_s = 0.89$ and $r_s = 0.77$, respectively. For some analyses, we grouped these into independent and dependent categories. Independent SLE (iSLE) and dependent SLE (dSLE) were coded as dichotomous variables to indicate whether a participant had experienced at least 1 such event in the past year. Further description on LTCT and event dependence ratings are available elsewhere and not elaborated here (e.g., Kendler and Prescott, 2006; Kendler et al., 1998, 1999).

Demographic Covariates—Age at interview, current marital status (coded as married or not), years of education (truncated at 20), and household income (truncated at \$200,000) were included as covariates in the analyses.

Statistical Analyses

Individual-Level Analyses—Multiple regression analyses were used to predict PYDD from drinking onset and SLE. We performed 3 sets of regression analyses separately for men and women: (i) tot-SLE (i.e., all SLE regardless of dependence ratings), (ii) iSLE, and (iii) dSLE. A baseline model estimated the effects of demographic variables (age at interview, current marital status, years of education and household income), and subsequent models retained those that were statistically significant at $p \leq 0.05$ for either sex. Next, we tested the main effects of drinking onset and past-year SLE. Finally, we entered the interactions of the drinking onset variables (ONS17 and ONS14) with SLE. Generally, we use change in explained variance (R^2) between nested models to index the significance of the added term(s). Given that we were testing a priori hypotheses regarding onset $\times$ SLE interaction, we also refer to p -values of the interaction terms in evaluating their significance. Generalized estimating equations adjusted for nonindependence of data from twin pairs and were used to produce correct standard errors for regression coefficients. Analyses were performed with SAS v9.2 (SAS Institute Inc., 2008).

Twin Model Estimation—Structural twin models were used to estimate the extent to which SLE, drinking onset, and their interaction accounted for genetic and/or environmental variance in PYDD. We estimated 3 sources of variance: *additive genetic* (A) (i.e., variation because of allelic effects that combine additively), *common environment* (C) (i.e., variance attributable to experiences shared by twins), and *individual-specific environment* (E) (i.e., variance unique to 1 twin in a pair, including measurement error) (see Prescott et al., 2004). MZ pairs share 100% of additive genetic variance, whereas DZ pairs share 50%. Higher similarity for twins in MZ pairs than DZ pairs suggests a genetic contribution to the phenotype, and equal similarity implies common environmental contributions. To the degree

that twins in a pair are not similar, this implicates individual-specific environmental factors and measurement error as contributing to the outcome. We estimated twin models by maximum likelihood using the program Mplus v5.2 (Muthén and Muthén, 2007) based on raw data from individuals from complete and incomplete twin pairs.

To incorporate the impact of demographic factors, the outcome variable used in the twin models is the residual of PYDD after partialing the effects of demographic covariates based on the baseline multiple regression model for each sex (see Fig. 1). Parallel to the individual-level analyses, model building proceeded by adding the main effects of drinking onset and SLE, followed by their interaction term. As in multiple regression, the resulting regression coefficients (in Table 4) refer to the change in PYDD (in standard deviation units) for each standard deviation change in the covariate. We evaluated nested models by comparing the difference in variance of PYDD attributable to genetic and environmental sources in the baseline (i.e., demographics-only) model upon addition of each covariate. For instance, a reduction in genetic variance of PYDD after adding the SLE term would suggest that the influence of SLE on PYDD is mediated by genetic effects. We report results for the 3 types of SLE, analyzed separately in men and women.

RESULTS

Descriptive Analyses

Descriptive statistics are presented by sex and drinking onset in Table 1. Individuals who reported drinking onset by age 14 were on average younger and less likely to be married at the time of the interview than those in the middle (onset ages 15 to 16) and older onset (age ≥ 17) groups. Men who started drinking at age 14 or younger had lower income and less education than men in other onset groups. Across both sexes, early drinking onset was associated with higher PYDD. The young onset group reported more dSLE than the middle and older onset groups, but the 3 groups did not differ in the number of iSLE.

Relation of Onset Age and SLE to Past-Year Drinking

Regression results for women and men are shown in Table 2a,b, respectively. We first estimated the association of demographic covariates with PYDD. Higher PYDD was associated with not being married in both sexes, higher income in women and lower education in men. We thus used the fit of a model consisting of marital status, income, and education as the baseline against which subsequent models were compared (Models F1 and M1). When simultaneously adjusting for the aforementioned demographic covariates, age at interview was not a significant predictor of PYDD and thus was not included in subsequent models.

In the next steps, we tested the main and interactive effects of totSLE and drinking onset on PYDD. TotSLE was a significant predictor of PYDD in men ($b = 0.08$, $SE = 0.03$, $R^2 = 0.56\%$), but not in women ($b = 0.01$, $SE = 0.02$, $R^2 = 0.04\%$) (Models F2 and M2). In contrast, drinking onset was a significant predictor of PYDD in both sexes. Among women, ONS17 accounted for 4.4% of variance in PYDD ($b = 0.28$, $SE = 0.04$; Model F3a); and ONS14 accounted for an additional 1.3% ($b = 0.25$, $SE = 0.07$; Model F3b). In men, ONS17 accounted for nearly 3.0% of variance in PYDD ($b = 0.42$, $SE = 0.05$; Model M3a); and ONS14 accounted for another 1.0% ($b = 0.25$, $SE = 0.06$; Model M3b). The interaction terms of totSLE with drinking onset ages were not significant (women: SLE $\times$ ONS17: $b = 0.05$, $SE = 0.05$; SLE $\times$ ONS14: $b = 0.07$, $SE = 0.08$; men: SLE $\times$ ONS17: $b = 0.001$, $SE = 0.07$; SLE $\times$ ONS14: $b = 0.08$, $SE = 0.06$; Model 5) and provided little additional explained variance in PYDD (Model 5 vs. 4: $\Delta R^2 = 0.21\%$ in women, 0.04% in men). Figure 2A,B shows the expected values of PYDD for women and men by onset age group based on the

estimates from Models F5 and M5, respectively. The evidence of interactions is weak but is in a pattern consistent with that reported by Dawson and colleagues (2007): the earliest onset drinkers with most SLE reported the greatest PYDD.

Next, we tested whether type of SLE (i.e., dependent or independent) moderated the association between drinking onset and PYDD (Table 3). For dSLE in women, the full model explained 11.6% of the total variation in PYDD, with 5.1% of the variation attributable to demographic variables, 5.7% to drinking onset, and 0.4% to dSLE. The onset $\times$ dSLE interactions accounted for 0.8%, due primarily to the dSLE $\times$ ONS17 term, which was statistically significant ($b = 0.31$, $SE = 0.11$). As shown in Fig. 3A, estimates based on the full model indicate that relative to women in the same onset group with no dSLE, those reporting at least 1 dSLE were estimated to consume 0.9 additional drinks per week if they began drinking by age 14 (1.8 drinks/wk for 0 dSLE vs. 2.7 drinks/wk for 1+ dSLE for onset ≤ 14), and 0.4 additional drinks if they began drinking by age 17 (1.3 drinks/wk for 0 dSLE vs. 1.7 drinks/wk for 1+ dSLE), whereas those who began drinking after age 17 drank slightly less (0.3 drinks) if they reported any dSLE (1.0 drink/week for no dSLE vs. 0.7 drinks/week for 1+ dSLE).

For dSLE in men (Table 3 and Fig. 3B), the full model explained 9.5% of the variation in PYDD, with most due to demographic characteristics and onset age. Less than 0.5% of the variance was accounted for by the main effect of dSLE and the onset $\times$ dSLE interactions. None of the onset $\times$ dSLE interaction terms was significant for men.

For iSLE in women (Table 3 and Fig. 4A), the full model explained 10.8% of the total variation in PYDD, with virtually all of this attributable to demographic characteristics and a significant main effect of onset. Less than 0.1% of the variance was accounted for by the main effect of iSLE and the onset $\times$ iSLE interactions, which were not significant.

In men, a somewhat different pattern of association with iSLE was observed. The full model accounted for 9.2% of the total variation in PYDD, with 5% attributable to demographic variables, 4% to the significant main effect of drinking onset, 0.02% to iSLE, and 0.2% to the independent effect of the onset $\times$ iSLE interactions. Despite the small R^2 , the interaction of ONS14 $\times$ iSLE was statistically significant ($b = 0.27$, $SE = 0.13$). As shown in Fig. 4B, compared to men who initiated drinking after age 14 and did not experience any past-year iSLE, those with a past-year iSLE and ONS14 consumed 1.2 additional drinks per week (4.3 drinks/wk for 0 iSLE vs. 5.5 drinks/wk for 1+ iSLE for onset ≤ 14). In the other onset groups, there was little effect of iSLE.

We also examined the bivariate association between current age and drinking onset age for evidence consistent with a telescoping bias. We investigated the potential for such a bias to influence our analyses related to drinking onset and SLE by testing whether current age moderated the SLE $\times$ onset age interaction in the 2 cases where we had some evidence for interactions (dSLE in women and iSLE in men). We added two 3-way interaction terms (age $\times$ SLE $\times$ ONS17 and age $\times$ SLE $\times$ ONS14) and 3 additional 2-way interaction terms (age $\times$ SLE, age $\times$ ONS14, and age $\times$ ONS17) to the models in Table 3. For dSLE in women, there was little evidence of any telescoping bias. The increase in R^2 associated with the 5 additional interaction terms was $<1.2\%$, and all the 3-way interaction terms had $p > 0.55$. For iSLE in men, the age $\times$ ONS14 $\times$ dSLE term was statistically significant ($p = 0.054$), but the regression weight was in the direction *opposite* to what would be predicted for a telescoping effect: *older* (rather than younger) age at interview combined with drinking onset by age 14 and having 1 or more past-year iSLE was associated with heavier PYDD. At any rate, the effect accounted for relatively little explained variance in PYDD ($\Delta R^2 < 0.5\%$).

for 5 additional terms). We thus conclude that there is little evidence that telescoping bias influenced our analyses investigating interactions between onset and SLE.

Sources of Variation in PYDD, SLE, and Onset

We conducted 3 sets of twin-pair analyses to estimate the sources of genetic and environmental overlap between onset age, PYDD (after partialing the effects of demographic covariates), and each type of SLE: totSLE, dSLE, and iSLE. For all 3 types of SLE, eliminating shared environmental influences resulted in minimal loss of fit relative to the full model ($\Delta\chi^2 < 0.1$, $\Delta df = 1$ for both men and women). Therefore, a reduced model of genetic and individual environmental influences (AE model) was used as the baseline for comparing subsequent nested models.

Consistent with findings from the regression analyses (Table 3), the most parsimonious model for women included interactions between drinking onset and dSLE; and for men included interactions between drinking onset and iSLE (full series of models not shown, but available upon request).

Table 4a,b shows parameter estimates from twin analyses of the PYDD residual score based on the AE model and the models of PYDD regressed on SLE and onset age. In the baseline PYDD model for women (Table 4a), genetic and individual-specific environmental influences accounted for 47 and 53%, respectively of variation in PYDD. For men (Table 4b), genetic and individual-specific environmental factors accounted for 43 and 57%, respectively of variation in PYDD.

Comparing the values of the genetic and specific environmental estimates between the baseline model and the values from the other models in Table 4 provides an indication of the genetic and environmental contributions to PYDD attributable to onset, the different types of SLE and their interactions. For women, incorporating the main effects of drinking onset, SLE, and their interactions resulted in a decrease in the genetic variance estimate, from 47 to ~40%, consistent with most of the effect of these variables on PYDD being accounted for through genetic effects. Similarly for men, the genetic variance estimate of PYDD decreased from 43 to ~38% upon including the main effects of drinking onset, SLE, and their interactions in the model.

DISCUSSION

The primary goal of our study was to replicate the finding reported by Dawson and colleagues (2007) of a stronger association between number of past-year stressors and alcohol intake among individuals who initiated drinking at age 14 or younger compared with those who began drinking at age 18 or later. Although the pattern of our findings is consistent with the interaction reported by Dawson and colleagues (2007), the main effect of stressful life events (regardless of dependence) and the interaction of onset age with life events explained relatively little of the variation in past-year drinking. For both sexes, having 1 or more life events accounted for $\leq 0.5\%$, and the life event by onset age interaction accounted for $\leq 1\%$ of the variation in drinking. In contrast, age at drinking onset was a more powerful predictor of variation in past-year drinking in both sexes, accounting for 3 to 6% of the total variance, similar in magnitude to the combined effects of sociodemographic variables.

A number of methodological differences between our measures and analytic approaches may partially explain the differences in results. Dawson and colleagues (2007) reported the interaction finding in terms of the effect on average daily drinking associated with each additional stressful event. After adjusting for demographic characteristics, the per-stressor

effect on daily drinking was 16% higher among those with young drinking onset (ages ≤ 14), and 7% among those with middle drinking onset (ages 15 to 17) compared with those with older drinking onset. We used a different measure of drinking outcome that reflected typical and heavy drinking days and only analyzed stressful events of moderate or severe intensity, so our effects sizes are not directly comparable. We found that after adjusting for demographic covariates, the effect on past-year drinking associated with each SLE (up to 3) among individuals with young onset was 138% (1.2 drinks/wk) higher for women and 99% (2.7 drinks/wk) higher for men, compared to those with older onset. Among those with middle onset, PYDD was found to be 55% (0.5 drinks/wk) higher among women and 70% (1.3 drinks/wk) higher among men for each additional event (up to 3), when compared to those with older onset. Given the distribution of our SLE variables, we coded totSLE as 0, 1, 2, 3+ and used binary coding (0 vs. 1+) for iSLE and dSLE. This difference from Dawson and colleagues' (2007) coding of SLE (0, 1, 2, 3 to 5, 6+) is likely to have contributed to differences in our findings. By restricting our analyses to SLE with at least high-moderate long-term threat, our sample had fewer SLE (0.7 totSLE vs. 2 in the NESARC study). Our intent was to exclude minor events that would not be expected to influence drinking as measured over the course of a year, but this may also have reduced our power to detect some interactions in subsets of the data.

Our analyses adjusted for sociodemographic variables, but not for other correlates of early onset. Dawson and colleagues (2007) reported stress-related differences in ethanol intake for each onset group after adjusting for sociodemographic characteristics, and also after adjusting for family history of alcoholism, personal history of alcohol dependence and other psychopathology, as well as the interaction of stressors with covariates. As mentioned earlier, the risk of early drinking onset on subsequent consumption is often thought to be transmitted via a constellation of individual, familial, and genetic risk factors (e.g., Jessor and Jessor, 1977; McGue et al., 2001a,b; Pitkänen et al., 2008). Consistent with this hypothesis, adjusting for these variables in the NESARC study decreased the effect size associated with drinking onset age, but the stress-onset interaction effect remained. The interaction observed in the NESARC sample appeared to be largely based on high levels of past-year drinking among individuals in the youngest onset group who reported 6 or more past-year stressors. Individuals in this subgroup reported drinking on average 3.5 oz of alcohol per day (i.e., ~6 drinks), much more than early-onset drinkers with 2 or fewer past-year stressors (0.8 to 0.9 oz), and than those with 3 to 5 stressors (1.8 oz; Dawson et al., 2007, Table 2). Although we had a relatively large sample, we did not have sufficient power to detect effects of this kind. For example, fewer than 4% of our participants reported having 3 or more SLEs that exceeded our severity threshold for inclusion in analyses.

Association of Age at Drinking Onset and Stress-Related Drinking: Differential Findings by Sex and Event Dependence

A noteworthy finding in Dawson and colleagues' (2007) paper is that when analyses were restricted to a set of 4 SLE deemed unlikely to be consequences of drinking ("death of a family member or close friend, illness of a family member or close friend, change in living situation, and change in work hours or responsibilities"; p. 71), the effect size for the interaction between SLE and drinking onset was similar to that obtained in the analyses for the full set of 12 events. This suggests that the impact of the independent events was similar to that of the other SLEs. We tested this explicitly using measures of event dependence coded from interviewer ratings rather by inferring independence based on type of event. If early drinkers have greater consumption when faced with stress and behave in ways that lead to more SLE, we would expect to see a significant onset $\times$ SLE interaction for both dependent and independent events.

We found a significant early onset $\times$ iSLE interaction among men. However, the effect was relatively weak and, for men with drinking onset before age 15, translated into a difference of about 1 drink per week associated with an independent event. Conversely, our results indicated that the interaction between SLE and drinking onset on past-year drinking among women was largely associated with dependent events. Although our design did not permit us to directly test for gender differences in effect sizes, it appeared that women's drinking was more closely tied to the occurrence of dependent events than was observed for men; this was particularly true among women with early drinking onset. A previous study of female twins from the same sample found that after adjusting for event severity, the onset of depression was more closely tied to dependent than independent SLE (Kendler et al., 1999). Other studies examining sex differences in sensitivity to the depressogenic effects of SLE found that women were more likely than men to become depressed over events related to their immediate social network (e.g., problem getting along, termination of romantic relationships; Kendler et al., 2001; Kessler and McLeod, 1984). It has been suggested that women's greater investments in social relationships make them more emotionally vulnerable to the impact of network events, which are more likely to be considered dependent events. To the extent that participants perceive events as dependent on their behavior, it is possible that women's stronger internalizing tendencies lead them to take responsibility for the occurrence of negative events within their control, which in turn triggers negative affective consequences (e.g., guilt, depression) and drinking to alleviate negative mood. Supporting the notion that women are more likely to cope with negative mood by drinking, Prescott and colleagues (2004) found that higher scores on a measure of drinking alcohol to manage negative affect were more strongly associated with risk of having a DSM-IV lifetime diagnosis of alcohol abuse or dependence among women than men.

Twin data provided the opportunity to investigate these mechanisms by examining the sources of common variance among drinking onset, SLE, and past year alcohol consumption. Our estimates of the contribution of genetic and environmental influences on alcohol consumption are consistent with those reported in other twin studies (see review by Dick et al., 2010). The degree of overlap between age at drinking onset and PYDD was modest and primarily attributable to genetic influences contributing to both measures. This reflects that among MZ pairs, past-year drinking of twin A is about as strongly related to the drinking onset age of twin B as it is to twin A's own onset age. In our prior study of the shared variance between onset age and lifetime alcohol abuse/dependence, the overlap was much greater (women: 38%, men: 23%; Prescott and Kendler, 1999) than between onset age and PYDD in the current study (women: 2.8 to 4.1%, men: 2.9 to 3.1%). However, this is not surprising because PYDD is only a "snapshot" of drinking during a single year. The association between SLE and alcohol consumption was significant only among women, which was because of a very small degree of overlap between dSLE and PYDD that was attributable mostly to genetic factors. For both sexes, the overlap among SLE, onset, and alcohol consumption was small and largely attributable to genetic factors contributing to all 3 measures.

These results are consistent with a common liability model whereby overlapping genetic factors influence onset, SLE exposure, and alcohol consumption. Genetic overlap could arise because age at drinking onset, SLE, and past-year alcohol consumption are all influenced by another genetically influenced variable such as mood-related drinking motives. For example, if mood-related drinking motives are (i) genetically influenced, (ii) associated with early drinking onset, and (iii) increase risk for drinking in response to SLE, this would create shared genetic variation for early onset, SLE, and past-year drinking. Another explanation could be that genetic factors underlying early drinking onset influence risk for experiencing SLE. For instance, individuals who are genetically predisposed for sensation-seeking may both initiate drinking at an early age and place themselves at risk for

experiencing various SLE (e.g., physical injury, legal problems). Thus, it is possible that increased rates of stress-related drinking observed among early drinkers are explained by their greater liability to engage in behaviors which potentially create life stress.

While research suggests that the association between stress and drinking behaviors is moderated by the specific nature of the stressor (e.g., Veenstra et al., 2006), our goal was to follow up on Dawson and colleagues' (2007) finding that stress-reactive drinking was unique to events potentially caused by drinking. Therefore, we did not separately examine the stress-alcohol association by specific event type. Our use of a validated measure of *event dependence* was particularly apt for the purpose of this study. In light of these findings, future research may investigate sex, drinking motives (e.g., Young-Wolff et al., 2009), coping style (e.g., Cooper et al., 1992), psychological distress (e.g., Mulia et al., 2008), event type, and their interactions as mediators or moderators of the association among drinking onset, SLE and drinking behavior.

Inclusion of a measure of event dependence in our study afforded better traction on understanding the causal relations between alcohol consumption and life event occurrence. We found that events rated as independent of respondents' behavior were associated with greater alcohol consumption among early drinking men. This makes it unlikely that the causal direction was for heavier drinking to create greater stress exposure. Rather, our findings are consistent with the notion that early drinking men are more likely to drink in response to independent life events compared with men with later drinking onset. However, we are unable to address the direction of causality between dependent life events and past-year drinking because our study design did not provide closely timed data on event occurrence and consumption. It is possible that early initiators have drinking patterns in adulthood that more often contribute to occurrence of dependent events. There is some support for this in our sample, as evidenced by the higher levels of dependent events among early initiators (see Table 1).

LIMITATIONS

Several limitations of this study should be noted. First, our method of data collection relied on self report, thus recall bias may affect data accuracy. Drinking behavior and SLE occurrence were based on retrospective report for a 1-year period prior to the interview and for an even longer duration for drinking onset. Although questions were asked in different sections of the interview to avoid priming participants of potential links between drinking and SLE, it is possible that biased recall could spuriously account for part of the association. Age at drinking onset might be confounded by "telescoping" bias, when older individuals recall events as occurring closer in time than they actually occurred, as we observed that late-onset drinkers tended to be older at the time of the interviews than early- and middle-onset drinkers (see Table 1). However, we did not find evidence for (linear) age effects on past-year drinking, nor that current age altered the association between onset age and past-year drinking patterns, as would have suggested systematic recall bias. Second, our sample comprises Caucasian twins born in Virginia. Our findings may not generalize to individuals of other ethnic or cultural backgrounds. Third, as discussed earlier, we are limited in our ability to make conclusions about the direction of causality between SLE and drinking. Data on more closely timed assessments of SLE and drinking are needed to address this question more fully.

SUMMARY

The results of this study replicate prior findings of higher alcohol consumption among individuals reporting earlier drinking onset. This association was modest and mediated by

shared genetic influences contributing to both early drinking onset and adult alcohol consumption. Past-year stress-related drinking was higher among early drinking men who experienced independent stressful events and early drinking women who experienced dependent stressful events. These results await further replication and should be considered in the context of known risk factors correlated with early onset, such as familial history of problematic substance use and externalizing tendencies.

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Fig. 1.

Model used for estimation of genetic and environmental sources of variation in past-year drinking density, partialing age at drinking onset, stressful life events, and their interaction. Rectangles represent manifest variables, circles represent latent variables, triangle represents PYDD intercept. SLE, stressful life events; Onset14, drinking onset ≤ 14 ; Onset17, drinking onset ≤ 17 ; PYDD, past-year drinking density; Genetic, variation in PYDD because of additive genetic effects; Common Env, variation in PYDD because of experiences shared by twins in a pair; Indiv Env, variation in PYDD unique to 1 twin in a pair. The effects of demographic covariates (marital status, years of education, and family income) have been partialled from PYDD prior to model-fitting. Model was fit separately to data from male and female twin pairs.

Fig. 2. Model prediction of past-year drinking density (PYDD) by number of stressful life events in the past year and age of drinking onset in **(A)** females and **(B)** males. Expected values of PYDD are based on group means on demographic characteristics and estimates from Models F5 and M5 in Table 2. Only stressful life events of at least high-moderate severity were included.

Fig. 3. Model prediction of past-year drinking density (PYDD) by number of dependent stressful life events in the past year and age of drinking onset in **(A)** females and **(B)** males. Expected values of PYDD are based on group means on demographic characteristics and estimates from Table 3. Only stressful life events of at least high-moderate severity were included.

Fig. 4.

Model prediction of past-year drinking density (PYDD) by number of independent stressful life events in the past year and age of drinking onset in **(A)** females and **(B)** males. Expected values of PYDD are based on group means on demographic characteristics and estimates from Table 3. Only stressful life events of at least high-moderate severity were included.

Table 1

Descriptive Statistics of Past-Year Drinkers by Sex and Age at Drinking Onset

		Age ≤14	15 to 17	≥18	All	Min	Max
<i>Demographic variables</i>							
<i>N</i>	M	619	1,070	518	2,218	–	–
	F	233	558	591	1,382	–	–
<i>Age at interview</i>							
M		33.6 (7.8) ^{a,b}	36.4 (9.0) ^c	38.9 (10.2)	36.2 (9.2)	20.4	58.2
F		32.0 (6.4) ^{a,b}	34.5 (7.3) ^c	38.1 (8.8)	35.6 (8.1)	21.4	61.5
<i>Married at interview (%)</i>							
M		52.7 ^{a,b}	61.0	60.0	58.3	–	–
F		53.2 ^{a,b}	64.0	68.0	63.9	–	–
<i>Family income (in \$1K)</i>							
M		49.2 (33.5) ^{a,b}	55.5 (34.2)	56.1 (36.8)	53.9 (34.8)	0	200
F		48.4 (33.0)	53.0 (33.6)	53.7 (33.3)	52.5 (33.4)	3	200
<i>Education (years)</i>							
M		13.5 (2.6) ^b	13.6 (2.6)	14.0 (2.8)	13.7 (2.6)	2	20
F		14.8 (2.2) ^b	14.6 (2.1)	14.3 (2.3)	14.5 (2.2)	3	20
<i>Drinking variables</i>							
<i>Drinking onset (years)</i>							
M		12.2 (2.3)	15.9 (0.8)	19.4 (2.4)	15.7 (3.1)	3	42
F		12.8 (1.8)	16.1 (0.8)	19.9 (3.0)	17.2 (3.4)	4	42
<i>Past-year drinks/wk</i>							
M		15.5 (25.2) ^{a,b}	9.2 (14.5) ^c	6.5 (13.9)	10.3 (18.3)	<1	168
F		5.0 (8.9) ^{a,b}	3.0 (5.7) ^c	1.8 (3.6)	2.8 (5.8)	<1	70
<i>Past-year stressful life events</i>							
<i>totSLE of LTCT≥3</i>							
M		0.7 (1.2) ^{a,b}	0.6 (1.1)	0.5 (1.0)	0.6 (1.1)	0	8
F		0.9 (1.3) ^a	0.7 (1.0)	0.8 (1.2)	0.8 (1.2)	0	9
<i>Any independent SLE</i>							
M		2.3 (1.9)	2.1 (2.0)	2.3 (2.0)	2.2 (2.0)	0	11
F		2.3 (1.8)	2.1 (1.7)	2.3 (2.0)	2.2 (1.8)	0	12
<i>Having ≥1 independent SLE of LTCT≥3 (%)</i>							
M		28.3	23.4	23.2	24.7	–	–
F		34.8	28.9	35.0	32.5	–	–
<i>Any dependent SLE</i>							
M		1.5 (1.4) ^{a,b}	1.2 (1.4)	1.1 (1.2)	1.3 (1.4)	0	10
F		1.2 (1.6) ^{a,b}	0.8 (1.2)	0.7 (1.1)	0.8 (1.2)	0	9
<i>Having ≥1 dependent</i>							
M		20.7 ^b	19.2	14.9	18.6	–	–

		Age ≤14	15 to 17	≥18	All	Min	Max
SLE of LTCT≥3 (%)	F	26.6	20.1	20.1	21.2	–	–

M, male; F, female; SLE, stressful life events; LTCT ≥3, long-term contextual threat severity was high-moderate or severe; totSLE, SLE combined across independent and dependent events.
Entries are means (SD) unless otherwise noted.

Significant pairwise differences by drinking onset at $\alpha \leq 0.05$ are indicated by:

- ^a Onset ≤14 versus 15 to 17;
- ^b Onset ≤14 versus ≥18;
- ^c Onset 15 to 17 versus ≥18.

Results from Stepwise Phenotypic Regression Models Predicting Weekly Alcohol Consumption Age at Drinking Onset and Total Stressful Life Events for (a) Females and (b) Males

Table 2

	F1	F2	F3a	F3b	F4	F5a	F5b
(a) Females (N = 1,350)							
Intercept	1.09 (0.17)	1.07 (0.17)	0.97 (0.17)	0.98 (0.17)	0.97 (0.16)	0.99 (0.16)	0.99 (0.16)
Marital status (current; 1 = Y, 0 = N)	-0.36 (0.05)	-0.36 (0.05)	-0.35 (0.05)	-0.35 (0.05)	-0.34 (0.05)	-0.34 (0.05)	-0.34 (0.05)
Family income (in \$10K)	0.03 (0.01)	0.03 (0.01)	0.03 (0.01)	0.03 (0.01)	0.03 (0.01)	0.03 (0.01)	0.03 (0.01)
Education (in years)	-0.01 (0.01)	-0.01 (0.01)	-0.01 (0.01)	-0.01 (0.01)	-0.01 (0.01)	-0.01 (0.01)	-0.01 (0.01)
Each additional totSLE (up to 3)	-	0.01 (0.02)	-	-	0.01 (0.02)	-0.03 (0.03)	-0.03 (0.03)
Onset ≤17	-	-	0.28 (0.04)	0.22 (0.04)	0.22 (0.04)	0.17 (0.05)	0.18 (0.05)
Onset ≤14	-	-	-	0.25 (0.07)	0.25 (0.07)	0.25 (0.07)	0.20 (0.09)
totSLE × Onset ≤17	-	-	-	-	-	0.07 (0.05)	0.05 (0.05)
totSLE × Onset ≤14	-	-	-	-	-	-	0.07 (0.08)
R ² (%)	5.11	5.15	9.47	10.76	10.78	10.95	10.99
ΔR ² c.f. previous model unless noted (%)	-	0.04	4.36(c.f. F1)	1.29	0.02	0.17	0.04
(b) Males (N = 2,160)							
Intercept	2.50 (0.15)	2.43 (0.15)	2.15 (0.15)	2.13 (0.15)	2.05 (0.15)	2.06 (0.15)	2.07 (0.15)
Marital status (current; 1 = Y, 0 = N)	-0.43 (0.05)	-0.42 (0.05)	-0.44 (0.05)	-0.43 (0.05)	-0.42 (0.05)	-0.42 (0.05)	-0.42 (0.05)
Family income (in \$10K)	-0.001 (0.01)	-0.0003 (0.01)	0.001 (0.01)	0.003 (0.01)	0.003 (0.01)	0.003 (0.01)	0.003 (0.01)
Education (in years)	-0.04 (0.01)	-0.04 (0.01)	-0.04 (0.01)	-0.04 (0.01)	-0.04 (0.01)	-0.04 (0.01)	-0.04 (0.01)
Each additional totSLE (up to 3)	-	0.08 (0.03)	-	-	0.08 (0.03)	0.06 (0.05)	0.06 (0.05)
Onset ≤17	-	-	0.42 (0.05)	0.33 (0.05)	0.33 (0.05)	0.31 (0.06)	0.33 (0.06)
Onset ≤14	-	-	-	0.25 (0.06)	0.25 (0.06)	0.25 (0.06)	0.20 (0.07)
totSLE × Onset ≤17	-	-	-	-	-	0.03 (0.06)	0.001 (0.07)
totSLE × Onset ≤14	-	-	-	-	-	-	0.08 (0.06)
R ² (%)	5.02	5.58	7.97	9.01	9.46	9.47	9.50
ΔR ² c.f. previous model unless noted (%)	-	0.56	2.95(c.f. M1)	1.04	0.45	0.01	0.03

totSLE, combined independent and dependent events of at least high-moderate threat, coded as 0, 1, 2, 3+, Onset ≤17, age at onset 17 or younger; Onset ≤14, additional effect of onset at age 14 or younger. Alcohol consumption was log-transformed using the equation: $\ln(\text{drinking density}+1)$.

Table 3

Results from Phenotypic Regression Models Predicting Weekly Alcohol Consumption from Age at Drinking Onset, and Dependent or Independent Stressful Life Events

	<u>Dependent SLE</u>		<u>Independent SLE</u>	
	<u>Female</u>	<u>Male</u>	<u>Female</u>	<u>Male</u>
	B (SE)	B (SE)	B (SE)	B (SE)
Intercept	0.99 (0.16)	2.08 (0.15)	1.01 (0.16)	2.11 (0.14)
Demographic variables				
Marital status (Current; 1 = Y, 0 = N)	-0.34 (0.05)	-0.41 (0.05)	-0.35 (0.05)	-0.43 (0.05)
Family income (in \$10 K)	0.03 (0.01)	0.003 (0.01)	0.03 (0.01)	0.003 (0.01)
Education (in years)	-0.01 (0.01)	-0.04 (0.01)	-0.01 (0.01)	-0.04 (0.01)
Main effects				
Having ≥ 1 SLE	-0.16 (0.07)	0.10 (0.13)	-0.05 (0.06)	0.04 (0.10)
Onset ≤ 17	0.15 (0.05)	0.31 (0.06)	0.21 (0.05)	0.36 (0.06)
Onset ≤ 14	0.22 (0.08)	0.25 (0.06)	0.23 (0.09)	0.18 (0.06)
Interaction terms				
SLE $\times$ onset ≤ 17	0.31 (0.11)	0.10 (0.16)	0.002 (0.09)	-0.11 (0.13)
SLE $\times$ onset ≤ 14	0.11 (0.15)	0.00 (0.15)	0.08 (0.13)	0.27 (0.13)
Sequential R^2 attributed to				
Demographic variables (%)	5.11	5.02	5.11	5.02
Onset (%)	5.65	3.99	5.65	3.99
SLE (%)	0.40	0.41	0.01	0.02
SLE $\times$ Onset (%)	0.82	0.03	0.05	0.17
Total (%)	11.62	9.45	10.82	9.20

$N = 1,350$ for females; $N = 2,160$ for males. SLE, stressful life events of at least high-moderate severity. Dependent and independent SLE were coded as dichotomous variables of 0 versus 1+ event. Alcohol consumption was log-transformed using the equation: $\ln(\text{drinking density}+1)$. To calculate sequential R^2 , covariates were added in a stepwise manner using the order listed. Estimates are unstandardized regression weights from the full model.

Table 4

Parameter Estimates from Best-Fitting Twin Models for Prediction of Drinking Density by Drinking Onset and Stressful Life Events in (a) Females and (b) Male

	PYDD: AE model			totSLE → PYDD			dSLE → PYDD			iSLE → PYDD		
	B	SE	Var (%)	B	SE	Var (%)	B	SE	Var (%)	B	SE	Var (%)
(a) Females												
Genetic	0.68	0.03	46.8	0.63	0.03	40.2	0.63	0.03	39.4	0.63	0.03	40.1
Specific envt	0.73	0.03	53.3	0.74	0.03	54.8	0.74	0.03	55.2	0.74	0.03	55.2
SLE	–	–	–	–0.04	0.04	0.2	–0.08	0.04	0.6	–0.03	0.04	0.1
Onset ≤17	–	–	–	0.12	0.04	1.3	0.10	0.03	1.0	0.13	0.03	1.8
Onset ≤14	–	–	–	0.09	0.04	1.6	0.10	0.03	1.8	0.11	0.03	2.3
Onset ≤17 × SLE	–	–	–	0.05	0.05	0.7	0.12	0.05	1.3	0.01	0.05	0.1
Onset ≤14 × SLE	–	–	–	0.05	0.04	1.1	0.03	0.04	0.7	0.02	0.04	0.4
R ² due to onset	–	–	–	–	–	2.9	–	–	2.8	–	–	4.1
R ² due to interaction	–	–	–	–	–	1.9	–	–	2.0	–	–	0.5
(b) Males												
Genetic	0.66	0.03	43.3	0.62	0.03	38.1	0.62	0.03	38.0	0.62	0.03	38.7
Specific envt	0.75	0.02	56.7	0.76	0.03	58.1	0.76	0.02	58.2	0.76	0.02	57.8
SLE	–	–	–	0.04	0.05	0.2	0.02	0.05	0.0	0.01	0.04	0.0
Onset ≤17	–	–	–	0.12	0.03	1.6	0.11	0.02	1.3	0.14	0.03	1.9
Onset ≤14	–	–	–	0.08	0.03	1.3	0.10	0.04	1.8	0.07	0.03	1.2
Onset ≤17 × SLE	–	–	–	0.001	0.05	0.02	0.04	0.05	0.6	–0.04	0.05	–0.3 ^d
Onset ≤14 × SLE	–	–	–	0.04	0.03	0.8	0.00	0.03	0.0	0.07	0.03	1.0
R ² due to onset	–	–	–	–	–	2.9	–	–	3.1	–	–	3.0
R ² due to interaction	–	–	–	–	–	0.8	–	–	0.6	–	–	0.6

Analyses for females based on data from 284 MZ and 186 DZ complete pairs and 417 twins from incomplete pairs. Analyses for males based on data from 465 MZ and 298 DZ complete pairs and 686 twins from incomplete pairs. AE model, genetic and specific environmental components; B, standardized betas; Var, variance proportion; totSLE, combined independent and dependent events of at least high-moderate threat; dSLE, dependent events of at least high-moderate threat; iSLE, independent events of at least high-moderate threat; DZ, dizygotic; MZ, monozygotic; PYDD, past-year drinking density. SLE, type of SLE (totSLE, dSLE, or iSLE, see column heading). Onset ≤17, age at onset 17 or younger; Onset ≤14, additional effect of onset at age 14 or younger. R² for onset calculated as the variance in PYDD attributable to Onset17 and Onset14, including their covariance with each other and with the main effect of SLE. R² for SLE × onset interaction calculated as the variance in PYDD attributable to SLE × Onset17 and SLE × Onset14, including their covariance with each other and with the main effects of SLE, Onset17, and Onset14.

^dVariance % is negative because of a negative covariance between Onset17 and SLE.