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Telescoped Trajectories from Alcohol Initiation to Disorder in Children of Alcoholic Parents

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Abstract

The current study tested whether and why COAs show telescoped (adolescent) drinking initiation-to-disorder trajectories as compared to non-COAs. Using longitudinal data from a community-based sample, survival analyses confirmed that COAs progress more quickly from initial adolescent alcohol use to the onset of disorder than do matched controls. Similar risks for telescoping were evident in COAs whose parents were actively symptomatic versus those whose parents were previously diagnosed. Stronger telescoping effects were observed for COAs whose parents showed comorbidity for either depression or antisocial personality disorder. Both greater externalizing symptoms and more frequent, heavier drinking patterns at initiation failed to explain COAs' risk for telescoping, although externalizing symptoms were a unique predictor of telescoping. This risk for telescoping was also evident for drug disorders. These findings characterize a risky course of drinking in COAs and raise important questions concerning the underlying mechanisms and consequences of telescoping in COAs.

Keywords

Alcohol; Children of Alcoholics; Telescoping

Used to describe the shorter drinking histories of alcoholic women as compared to alcoholic men, the term telescoping has generally referred to an accelerated trajectory from drinking onset to treatment seeking (Zilberman, Taveres, & el-Guebaly, 2003). The telescoped drinking histories of alcoholic women are significant not only in suggesting epidemiological and perhaps phenotypic differences within alcohol disorders but also in conveying greater risk than the more prolonged pre-alcoholic drinking histories of men for such negative outcomes as alcoholic liver disease and alcohol-related brain atrophy (Lisansky, 1958; Mann et al., 2005). Telescoping effects in women thus have strong clinical, phenomenological and etiological implications.

For these reasons, telescoping has received increasing attention in studies of alcohol-related problems but also of other substance disorders and gambling addictions (Haas & Peters, 2000; Hernandez-Avila, Rounsaville & Kranzler, 2004; Tavares, Zilberman, Beites & Gentil, 2001). Despite the apparent significance of telescoping effects, they have been little studied in other at-risk populations. One group that may show particular risk for an accelerated trajectory from drinking initiation to alcoholism is children of alcoholics (COAs). COAs show higher

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rates of alcohol abuse and dependence as well as an earlier age of onset for drinking as compared to their peers (Chassin, Pitts, DeLucia & Todd, 1999; Sher, Walitzer, Wood & Brent, 1991; Wong et al., 2006). Moreover, a growing body of research indicates that COAs have a greater biological risk for drinking as evidenced in studies showing unique patterns of cued psychophysiological responding to intoxication and greater sensitivity to the reinforcing properties of alcohol (Pihl, Peterson & Finn, 1990; Schuckit & Smith, 2000). In concert with environmental factors that may hasten alcohol-related problems after drinking onset, these biological and genetic vulnerabilities may create a greater risk for telescoping in COAs than in children of non-alcoholic parents. In the current study, we tested whether COAs evidence a telescoped trajectory from drinking initiation to alcohol disorder, examined potential mechanisms of risk, and considered potential gender differences and heterogeneity within this at-risk group. Given the heightened risk for disorder associated with the early onset of drinking behavior (Grant & Dawson, 1997), we focused our analyses on those who initiate alcohol use in adolescence.

Evidence for telescoping

The term telescoping was first used by Lisanksy (1958) to describe the rapid acceleration of alcohol-related problems in women following drinking onset. Since that time, most studies have used treatment samples to retrospectively estimate the ages of onset for both drinking initiation and alcohol disorders (Zilberman et al., 2003). For example, Mann et al. (2005) showed that alcoholic women had a shorter history of dependence than alcoholic men in their treatment sample; nonetheless, alcoholic women and men showed equivalent brain atrophy in comparison to non-alcoholic women and men.

However, retrospective reports of age of onset of discrete behaviors (like drinking onset) have shown limited validity. For example, previous studies find that individuals report increasing ages of onset as they age, apart from cohort or period effects, for initiating drinking, marijuana use, smoking and daily smoking (Johnson & Schulz, 2005; Johnson, Gerstein & Rasinski, 1998). Although some studies indicate fair to high reliability of reported onset of psychiatric symptoms in diagnostic interviews (e.g., Wittchen et al., 1989), Johnson and Schultz argue that most studies examine a fairly short time window (1-2 years). Moreover, they showed gender and ethnic differences in the bias underlying reported age of onset, resulting in the potential for distorted group comparisons that may either create or obscure true telescoping effects of interest.

Additional problems with the telescoping literature come from a heavy reliance on treatment samples. First, studies of treatment samples are subject to well-established biases, particularly due to their reliance on a select and typically more severely disturbed group of treatment seekers than is representative of the population (Berkson, 1950). Although Lewis, Bucholz, Spitznagel and Shayka (1996) demonstrated a limited effect of telescoping (i.e., faster emergence of alcohol-related disorders within the first two years of problem drinking) in community-sampled women than men, the extent to which findings from treatment samples are generalizable deserves further study. Second, telescoping effects typically concern the time from initiating drinking until treatment entry for alcohol disorders. Thus, for many studies treatment seeking is used as a proxy for alcohol-related problems. However, Hesselbrock et al. (1984) noted that women show shorter lags between first evidencing alcohol problems and first seeking treatment, suggesting that gender differences in telescoping may simply be due to gender differences in treatment seeking behavior.

Together, these issues undermine current efforts to estimate telescoping effects. Moreover, given that such studies have primarily focused on gender differences, little is known about telescoping in other sub-populations. To address these concerns, we tested whether COAs

showed telescoped trajectories from drinking initiation in adolescence to onset of alcohol disorder using data from a longitudinal, community-based study of adolescent COAs and matched controls.

COAs' risk for telescoping

Current epidemiological patterns of alcohol use among COAs are consistent with a telescoping effect. COAs show higher rates and faster acceleration of alcohol use starting in adolescence and continuing into adulthood (Chassin, Curran, Hussong & Colder, 1996; Sher, 1991); they report earlier ages of drinking onset in longitudinal studies that reduce risk for retrospective biases (King & Chassin, in press; Wong et al., 2006); and they report higher rates of alcohol abuse and dependence in young adulthood (Chassin et al., 1999). Although this evidence is consistent with greater telescoping in COAs, this hypothesis has not been directly tested, leaving unanswered the question of whether COAs simply have an early entry into what are similar trajectories of drinking, as compared with non-COAs, or whether they indeed show alcohol-related symptoms earlier in their drinking histories.

Among the factors accounting for COAs' potential risk for telescoping are genetic and biological influences underlying responses to alcohol that promote addiction (termed the enhanced reinforcement model by Sher, 1991). As described by Newlin and Thomson (1999), COAs show greater sensitivity to the rewarding or arousing effects of alcohol over time whereas non-COAs show chronic tolerance to these same effects. Moreover, Schuckit and Smith (2000) found that COAs have a lower physiological response to alcohol, decreasing opportunities for learning controlled drinking. These experiences may then promote pro-drinking cognitions and positive alcohol-related expectancies in COAs, increasing their alcohol seeking behavior and opportunities for developing drinking problems. Given fewer contextual barriers to heavy drinking (e.g., greater likelihood of associating with drinking friends and family members), COAs may have little to slow their progression from drinking onset to alcohol dependence. As such, COAs may move from initiation of drinking to the development of alcohol dependence through a more intensive use of alcohol supported by their genetic and environmental vulnerabilities.

A second mechanism potentially accounting for a telescoping effect in COAs is a propensity toward behavioral undercontrol more generally (termed the deviancy proneness model by Sher, 1991). Evident as a disinhibited temperament and externalizing symptoms in COAs as early as 3-5 years of age (Puttler, Zucker, Fitzgerald & Bingham, 1998; Wong et al., 2006), early engagement in deviant behaviors and continued externalizing symptoms into early adolescence are significant predictors of later alcohol disorders (Zucker, 2006). In addition to conveying a general risk for deviancy, such as the early and heavy use of alcohol, greater externalizing symptoms increase risk for affiliating with substance using peers and thus accumulating additional models of use, access to alcohol, and encouragement of drinking. As such, externalizing symptoms may also mediate the relation between parent alcoholism and telescoped onset-to-disorder trajectories.

Despite the potential relevance of these two mediating mechanisms for explaining a general pattern of risk for telescoping in COAs, previous studies also indicate heterogeneity in COAs' risk for alcohol-related problems (Zucker, 2006). We examined four sources of heterogeneity in particular. First, we tested whether the recency of parent alcoholism impacted evidence of telescoping in COAs. If environmental risk factors for drinking involvement are an essential component to evidencing telescoped onset-to-disorder trajectories in adolescence, then the trajectories of children whose parents are actively abusing alcohol should show greater telescoping as compared to those of children whose parents are not actively abusing alcohol. More recent parent alcoholism may convey greater risk for telescoping due to increased

environmental risk (such as greater parental modeling, impaired parenting, and easier access to alcohol) and/or genetic influences (such as denoted by a more chronic form of parent alcoholism). This hypothesis is consistent with findings that the magnitude of risk for elevated alcohol and drug use in early to middle adolescence varies with the recency of parent alcoholism, such that the strongest risk is found for those with current alcoholic parents and more moderate risk for those with recovered alcoholic parents (Chassin, Rogosch & Barrera, 1991).

Second, we tested whether comorbid disorders in alcoholic parents heightened risk for telescoping among COAs. Specifically, both antisocial personality disorder and depression are highly comorbid with alcohol disorders and previous studies indicate that these comorbidities may define unique subtypes of alcoholism (Zucker, 2006). Although previous studies suggest that antisocial alcoholism is more heritable, more common in men and related to an early onset of problem drinking, depression has also been associated with telescoped onset-to-disorder patterns and depressive alcoholism is more common in women. Children of parents with antisocial and depressive alcoholism have shown greater risk for negative outcomes in comparison to children of parents with alcoholism only, with no differences evident between children of parents with alcoholism only and controls in some instances (Hussong, Flora, Curran, Chassin & Zucker, in press; Hussong, Wirth et al., 2006; Puttler et al., 1998; Wong, Zucker, Puttler, & Fitzgerald, 1999).

Two other factors that may impact the telescoping effect in COAs are gender and age of drinking initiation. Given methodological limitations in studies of telescoping effects for women versus men (reviewed earlier), some skepticism may be raised about the generalizability of this effect for studies that rely on community-based samples, longitudinal assessments to reduce retrospective recall, and dating of disorder onset based on symptom appearance rather than treatment entry. Nonetheless, studies of gender differences in risk for alcohol-related outcomes related to parental alcoholism also indicate that females may be more vulnerable than their male counterparts (Russell, Cooper, & Frone, 1990; Sher et al., 1991). Thus, given observed trends in telescoping and in COAs' risk for alcohol-related outcomes, we tentatively posited that female COAs would show greater risk for telescoped trajectories from drinking-initiation to disorder-onset than male COAs.

Age of drinking initiation may also play a moderating role in this risk. Grant and Dawson (1997) reported 8% and 14% decreases in risk for alcohol abuse and dependence, respectively, for each year in which drinking initiation was delayed through adolescence. Although other researchers have replicated the association between early onset of drinking and increased risk of alcohol disorder (Pitkanen, Lyyra & Pulkkinen, 2005), many of these studies serve to qualify this finding. Notably, this risk appears to be stronger for alcohol dependence (Grant, Stinson & Harford, 2001; Prescott & Kendler, 1999), particularly when comorbid with drug dependence (King & Chassin, in press), and at longer intervals between initiating drinking and disorder onset (Grant et al., 2001). However, the age of initiating drinking was based on retrospective report in most studies (with the notable exception of King & Chassin, in press). We provide more temporally proximal reports of drinking initiation and we focus on the relation of drinking onset in early adolescence, when most individuals begin drinking and when drinking is most predictive of later disorder (before age 16; Grant & Dawson, 1997, Pitkanen et al., 2005), and alcohol disorders in early adulthood, when the highest rates of alcohol abuse and dependence are observed and normative declines in drinking begin to emerge (Windle & Davies, 1999). Although Grant (1998) did not find that parental alcoholism moderated the relation between age of initiation and whether or not an alcohol disorder was observed, the extent to which age of drinking initiation shows differential effects on the timing of alcohol disorder among COAs versus children of non-alcoholic parents is unknown.

The current study

In the current study, we tested whether COAs show telescoped drinking onset-to-disorder trajectories as compared to non-COAs. Moreover, we tested for heterogeneity in COAs' risk for telescoping as a function of recency of parent alcoholism, evidence of comorbid disorders in the alcoholic parents, child gender, and age of onset of drinking. In addition, we also examined whether the early heavier drinking patterns and greater externalizing symptoms of COAs may in part explain any risk they evidence for telescoping. Finally, to examine whether COAs' risk for telescoping is specific to alcohol disorders (as suggested by the mediating mechanism of early heavy drinking patterns and a unique physiological response to alcohol in COAs) versus more broadly relevant for disinhibitory disorders (as suggested by the mediating mechanism indicated by greater externalizing symptoms and a potential genetic vulnerability to behavioral undercontrol), we also tested whether COAs showed a telescoped trajectory from drinking onset to drug disorder. To test these hypotheses, we examined a community-based sample of COAs and matched controls who participated in a longitudinal study beginning in early adolescence. Because increasing evidence indicates greater risk for alcohol disorders and related negative consequences with an early onset of drinking, we focused our analyses on telescoping effects following from an adolescent onset of alcohol involvement.

Method

Sample and procedures

A community sample of 454 families completed three annual interviews when the target child was an adolescent (at ages 11-16, 12-17 and 13-18 across waves) and two follow-up interviews when the target was a young adult (approximately 5 and 10 years after wave 3 at median ages 20 and 25, respectively). Alcoholic parents were identified through court records, HMO wellness questionnaires, and telephone surveys. Inclusion criteria for COA families were: living with a biological child aged 11-15, non-Hispanic Caucasian or Hispanic ethnicity, English speaking, and a biological and custodial parent who met DSM-III lifetime criteria for alcohol abuse or dependence. Control families were matched to these COA families on the basis of ethnicity, family structure, SES and the adolescent's age and sex. Attrition biases were minimal, with 97% subject retention over the first three waves of assessment and 90 and 91% retention of the initial wave 1 participants at each of the young adult follow-ups (for details, see Chassin, Barrera, Bech, & Kossak-Fuller, 1992; Chassin et al., 1999).

During the first four waves, adolescents and their parents (mothers and fathers, when available) completed computer-based interviews administered to them separately in their homes or at the university. At wave five, adolescents continued to complete computer-based interviews. On each occasion, participants received up to $70 compensation. Many steps were taken to protect confidentiality and increase honest reporting (see Chassin et al., 1991).

For the current study, only participants who reported initiating alcohol use during the first three, adolescent interviews were retained for primary analysis. Additionally, those who were classified as meeting criteria for alcohol abuse or dependence were required to provide valid data on the age of symptom onset (8 participants did not). The resulting analysis sample consisted of 214 participants, with 131 COAs and 83 controls (51% male, 30% Hispanic and 70% non-Hispanic Caucasian, with 25% of parents having a high school education or less, 45% having some vocational, technical or college training, and 30% having graduated from college). Across the five waves of assessment, adolescents were on average 13.1, 14.1, 15.1, 20.2 and 23.5 years old. As expected given their selection on initiating alcohol use in adolescence, participants in the analysis sample were slightly older at interview ($t(452)=5.35$, $p<.001$) and more likely to be COAs ($\chi^2(1,N=454)=8.06$, $p<.01$), but little difference was detected in ethnicity ($\chi^2(1,N=453)=0.23$, $p=.63$), gender ($\chi^2(1,N=454)=0.29$, $p=.59$), or the

educational status (t(452)=0.41, p=.68) of the parents relative to the overall sample. Retention rates for this subsample at waves four and five were 96 and 100%, respectively.

Measures

Demographic variables included participant gender (0 =female; 1 = male), age of participants at first assessment, ethnicity (0=non-Hispanic Caucasian, 1=Hispanic American) and parent education (maximum of either parent's educational status assessed through parental report on a 6-point scale ranging from (0) less than 12 years or not a high school graduate to (5) graduate or professional school training).

Parent Diagnoses—Families were classified as alcoholic if either parent met DSM-III lifetime criteria for diagnoses of alcohol abuse or dependence. When possible, parents were directly interviewed using a computerized version of the Diagnostic Interview Schedule III (DIS; Robins, Helzer, Croughan & Ratcliff, 1981) to assess diagnostic status. In cases where a biological parent was not directly interviewed (19% of fathers and 8% of mothers in the full sample), the reporting parent was used as the informant using the FH-RDC (Andreasen, Endicott, Spitzer & Winokur, 1977) to assess alcoholism. Previous studies have found a high degree of test-retest reliability using this method to diagnose alcoholism (98.8% agreement, Kappa=.95; Zimmerman, Coryell, Pfohl & Stangl, 1988) and excellent specificity (92%) and sensitivity (90%) for wives reporting on their husbands' substance abuse disorders (Kosten, Anton & Rounsaville, 1992). In the current sample, 61% of families had at least one alcoholic parent.

To code for recency of parent alcoholism, we identified whether alcoholic parents met criteria for alcohol abuse or dependence in the assessment year prior to that in which the adolescent reported initiating alcohol use. For waves 2 and 3, diagnostic interviews were not available, though parents reported the frequency of their alcohol use as well as their experience of alcohol-related consequences and dependence symptoms (using items from Mayfield, McLeod, & Hall, 1974 and Sher, 1991). We selected self-report items reflecting DSM-IV criteria for alcohol abuse and dependence (APA, 1994). With respect to alcohol abuse, two items each assessed physically hazardous drinking (e.g., alcohol use caused you to have an accident or injury) and legal problems (e.g., alcohol use caused you to get arrested for drunk driving), five items assessed failed role obligations (e.g., alcohol use caused you to lose a job or get kicked out of school), and six items assessed interpersonal problems (e.g., alcohol use caused you to get complaints from your family or friends). With respect to alcohol dependence, one to two items each concerned the development of tolerance (e.g., found that you needed larger amounts of alcohol to get an effect), withdrawal, using more than intended, failing to cut down, excessive time spent in obtaining alcohol, giving up important activities and using despite physical symptoms, with item wording closely following diagnostic criteria. Parents endorsing at least weekly drinking in the past year and meeting criteria for abuse (one of the four indicators) or dependence (any three of seven indicators), were diagnosed as alcoholic within the past year. Parents were classified as controls (i.e., parents never reporting an alcohol disorder, n=83) or reporting active alcoholism (i.e., parents reporting an alcohol disorder recently, n=45) or inactive alcoholism (i.e., parents reporting a lifetime but not a recent alcohol disorder, n=86).

Lifetime affective disorder (major depression or dysthymia) and antisocial personality (ASP) were also obtained with the DIS interview (Version 3, Robins et al., 1981). Of 142 alcoholic parents, 15 and 11% evidenced an affective disorder and ASPD, respectively. (Fifteen were missing comorbidity data and thus were omitted from subsequent analyses of parent comorbidity.) We used these diagnoses to define three groups: (a) comorbid alcoholic families (16% of families) who had an alcoholic parent also evidencing either a affective or ASP disorder (regardless of the diagnostic status of the other parent), (b) non-comorbid alcoholic

families (43%) with at least one alcoholic parent but without an alcoholic parent who evidenced an affective or ASP disorder (regardless of diagnostic status of a non-alcoholic parent, if present) and (c) non-alcoholic families (42%) with neither parent evidencing an alcohol disorder (regardless of the presence of affective or ASP disorders).[1]

Drinking frequency—At all waves, participants reported the frequency of their use of beer/wine and hard liquor as well as the frequency of drunkenness and drinking five or more drinks at a single occasion. These four items all assessed use within the past year with an 8-point response scale ranging from (0) never to (7) daily. The mean of the four items formed the initial drinking frequency variable within wave. We created an index of initial drinking based on the wave (one to three) that most closely followed the reported age of onset for drinking (M=.60, SD=.92, α=.88). We also created an indicator of late drinking frequency that indexed drinking behavior at the wave of assessment (one through five) immediately following onset of alcohol disorder for those who met criteria.

Externalizing symptoms—Adolescents reported on externalizing symptoms in waves one to three using an adapted form of the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1978). A mean of 22 items served as the within wave externalizing scale, with items selected that loaded on the externalizing factor for both boys and girls aged 12-16. The original CBCL response scale was expanded to a five-point Likert scale to increase variability in responses. For the current study, the externalizing scale for each adolescent was based on the wave of measurement at or just before the reported age of onset for drinking or on the wave one report if initiation was before assessments began.

Age of drinking initiation—To reduce problems with long-term retrospective bias, repeated assessments of age of onset during early to middle adolescence were coded to capture the age of initiating alcohol use. Although these reports involve retrospective reporting, they are based on short-term retrospection (given that these reporters are adolescents) and on multiple reports (to reduce bias). We assessed age of onset at the first three waves (although items differed from wave 1 to waves 2 and 3), and we used the average of these reports to index age of drinking initiation. In wave 1, participants reported the calendar year when they first initiated alcohol use ("more than just a few sips") and we derived age of onset based on this report as well as the year of the interview and adolescents' ages at wave 1. At waves 2 and 3, a two-part question asked adolescents to report their age in years (rather than by calendar year) when they initiated alcohol use or, if they could not recall the exact age, to report their age of initiation using the following response scale: (a) before age 10, (b) 10 or 11, (c) 12 or 13, (d) 14 or 15, or (e) 16 or over. For participants who only used this response scale to report age of initiation, responses of "before age 10" and "16 or over" were coded as missing because exact ages could not be recovered. [2] Other categories were recoded at the mid-point (i.e., 10.5 for the response "10 or 11"). Correlations among reported age of onset across waves were moderate in magnitude (r=.31 to .50), similar to other studies (Labouvie, Bates & Pandina, 1997).[3]

Onset of Alcohol and Drug Disorders—At waves 4 and 5, interviewers administered the computerized version of the DIS (C-DIS-III-R, Robins et al., 1981) to participants to assess abuse and dependence of alcohol as well as eleven additional classes of substances (i.e.,

[1]Note that 9 and 4% of 124 non-alcoholic spouses of alcoholic parents and 4 and <1% of 166 parents in non-alcoholic families also evidenced an affective disorder and ASPD, but all individuals were categorized similarly as 'non-alcoholic' prior to defining families as noted above.

[2]Outlying early ages (i.e., at or before age 6) for either alcohol initiation or disorder onset were recoded as missing because they were thought to represent unreliable reports. This occurred for six observations, leaving two cases which were omitted due to insufficient data to calculate ages of onset. Only one observation and thus one case was omitted due to unreliable reports of age of disorder onset. Open-ended ages of onset in the ordinal response scale for waves two and three were recoded to missing for eight observations, leading to the omission of three cases across waves.

cannabis, amphetamine, sedative/hypnotic/anxiolytic, prescription drug, cocaine, heroin, opioid, PCP, hallucinogen, inhalant, and NOS). If participants met abuse or dependence criteria for a given substance, the age at which they first experienced diagnostic symptoms was assessed. The mean reported age of onset of abuse or dependence symptoms over waves 4 and 5 indexed alcohol disorder onset for the current study. For drug disorders, the youngest age of onset for abuse or dependence of the eight substances was defined within waves 4 and 5 and an average of these ages of onset across the two waves served as the index of age of onset for drug disorders in subsequent analyses. Agreement in reported age of onset across waves was modest (r=.29, N=131.[4]

Analysis Plan

To test our hypotheses regarding telescoping, we used survival analysis. For our dependent variable, we estimated the probability (or hazard) of onset for alcohol disorders as a function of the number of years that had passed since the initiation of alcohol use. To conduct these analyses, we first had to construct a data set indexing years from age of drinking initiation to disorder onset. Thus, the time variable for the survival analyses was the number of years the adolescent had been drinking (rounded to the nearest whole year), beginning with the age of onset and ending with the participant's age at the last wave of data collection for which data were available on alcohol disorder. As such, time zero for all participants in the analysis sample was set to the year in which he or she initiated alcohol use. We then used these data to construct life tables indexing the hazards of disorder from the point of drinking initiation and then to conduct inferential tests of predictors of these hazards that directly examined our hypotheses.

Results

Life Tables for disorder hazard rates

Among 214 participants, we observed a total of 2180 life-years between the time of drinking initiation and either the onset of alcohol disorder or censoring. Table 1 presents the life table describing this sample and shows that 34% of controls and 67% of COAs reported the onset of an alcohol disorder during the course of the study. Moreover, most participants were diagnosed within the first ten years of observation when very little censoring occurred. Given this, the censoring at the later points had no apparent impact on the final survival rate estimates for controls and COAs.

Telescoping trajectories in COAs

To test our primary hypotheses, we estimated discrete-time hazard models that used the logit link function.[5] Within this framework, we identified telescoping effects based on different

[3]Due to the relatively low correspondence in reports of drinking age of onset across waves and to the concern with potential bias due to increasing ages of onset with greater retrospective lags, we also conducted our primary analyses (model 3, Table 2) coding age of onset for drinking based on the first wave in which drinking onset was reported. Little change in our primary findings emerged concerning COAs' risk for telescoping. However, the interaction between age of drinking onset and gender was only marginally significant such that the effect for girls was non-significant whereas that for boys remained significant (i.e., in which later ages of onset were associated with a greater hazard of alcohol disorder.) We retained our indicator of drinking age of onset averaged across waves to report our primary findings because of the greater reliability that multiple items provide for this index and because we found only limited evidence of an increasing age trend due to retrospective bias in our reports over time.

[4]The low agreement for age of onset for drug disorders is consistent with previous studies showing low agreement in absolute reported age of onset for illicit drug use in a high-risk sample (37% report the same age of onset; Parra, O'Neill, & Sher, 2003). Higher agreement rates are evident with a shorter (two year) lag (67%, 54%, 43% reporting ages of onset for use marijuana, cocaine, and crack within one year; Johnson & Mott, 2001), particularly with repeated short lags (ICCs=.59-.73 in Parra et al., Johnson & Mott), and for reported age of onset for drug disorders in an inpatient, drug treatment sample (ICC=.51, .93, and .95 for cannabis, cocaine, and opiate disorders). Thus, it is difficult to determine the comparability of our reports of age of onset for drug disorders with the literature given substantial study differences, although these self-reports are notably poor across most indicators of non-clinical samples in the literature.

[5]Use of the logit link imposes as assumption of proportional odds for disorder at any given point in time. This assumption was found to be reasonable for the data.

rates of survival following drinking initiation for COAs versus non-COAs. Given sparseness at some of the time points, we used a parametric function to model the logit of the hazard rates over time, as recommended by Singer and Willett (2003). Specifically, the logit of the hazard was modeled as a quadratic function of years since drinking onset and COA status. Plots of the observed hazard and survival rates against the fitted hazard and survival rates, shown in Figure 1, indicated that the quadratic model fit the data quite well.[6] In this baseline model, we show a significant telescoping effect of COA status (b=1.01, $\chi^2(1)=20.08$, p<.001; OR=2.74) with the estimated odds ratio indicating that the odds of disorder for COAs was 2.7 times higher than the odds of disorder for non-COAs at each point in time (conditional on not already having been diagnosed). The telescoping effect is clarified by examining the fitted survival functions in Figure 1 which show that 25% of COAs showed onset of disorder within four years of initiating drinking whereas 25% of controls did so within seven years of initiating drinking. (See baseline model in Table 2).

This telescoping effect for COAs was maintained once we controlled for child gender, age at first interview, ethnicity and parental education. (See model 1 in Table 2). In addition, boys (b=.69, $\chi^2(1)=11.01$, p<.001; OR=1.98) also showed telescoping effects or a greater odds of disorder onset following initiating drinking. No effect for ethnicity or child age was found, nor did child gender interact with parent alcoholism in predicting the hazards for onset of disorder (and thus this interaction is not reported in Table 2).

Because COAs do report a younger age of initiating drinking, we also controlled for age of drinking onset in these analyses (centered at the median value of 12.5 years of age), but once again found that the telescoping effect for COAs was maintained (b=1.13, $\chi^2(1)=23.18$, p<.001; OR=3.08; see model 2, Table 2). Moreover, age of drinking onset showed a positive association with the hazard for onset of disorder such that an older age of onset marginally predicted an accelerated trajectory to alcohol disorder (b=.13, $\chi^2(1)=3.12$, p=.08; OR=1.14). No interaction between parent alcoholism and the age of drinking onset was found (and thus is not reported in Table 2).

Because findings for gender and age of drinking initiation were contrary to some previous reports, we further probed our findings through post-hoc analyses. First, model diagnostics suggested the need for two interactions in the model, one between age of onset and gender (b=.34, $\chi^2(1)=7.44$, p<.01; OR=1.41), and the other between age of onset and period, or years of drinking (b=−.17, $\chi^2(1)=37.34$, p<.001; OR=.85). Plots of these interactions (see Figure 2) indicate that later initiation increases risk for telescoping in boys. For girls, the pattern changes over time such that, like boys, later onset girls show somewhat greater telescoping within the first 3-5 years following drinking onset than do early onset girls. However, eight years post-drinking onset, early onset girls appear to show higher rates of a new alcohol disorder than their later onset female peers, who show little onset of new disorder (i.e., change in their survival rates) from this point forward. Thus, the cross-over in survival curves for girls beginning eight years post drinking onset appears driven by an overall greater risk for alcohol disorder in early versus later onset girls (shown as a higher peak hazard in Figure 2) that is manifested as a more prolonged period of risk. This prolonged period of risk may in part be driven by the developmental press of emerging adulthood. As shown in Figure 3, which shifts the time axis to years of age rather than years from initiation, the highest hazards for first evidencing diagnostic symptoms for alcohol disorders occurs around age 18 regardless of age of drinking initiation. Figure 3 also shows that girls who initiate early (i.e., at age 10) have higher hazard rates than girls who initiate later (i.e., at age 14), whereas boys show the opposite pattern.

[6]The addition of a cubic term to the model did not significantly improve model fit, nor did the inclusion of interaction terms between the time trends and COA status (a test the proportional odds assumption).

To further probe these findings, we repeated the analyses based on the hazards of first experiencing a symptom of alcohol dependence rather than alcohol abuse or dependence. No significant changes in the findings emerged. Second, we split the analyzed sample into three groups based on age of drinking initiation representing the youngest 25%, the middle 50% and the oldest 25%. A fourth group was defined by the excluded participants that did not onset within the first three waves of assessment. Simple survival rates were calculated as the proportion of individuals not meeting diagnostic criteria by the fifth wave of assessment. These results were consistent with our previous findings. In order of increasing age of onset, survival rates were .44, .62, .69 and .77 for the four age groups for girls and were .41, .33, .15 and .65, respectively, for boys. The nonlinear pattern for boys indicates that delaying onset is associated with lower survival within the age of onset range represented in our hazard models. In contrast, delaying onset until late adolescence or beyond reverses this trend, producing the highest survival rate for boys.

Early drinking patterns and externalizing behavior as potential mediators

To test whether greater rates of early drinking and externalizing behavior among alcohol initiating COAs as compared to their peers may partially account for the telescoping of alcohol initiation-to-disorder trajectories in this at-risk group, we included the frequency of alcohol use and externalizing symptoms at the closest wave to drinking initiation as potential mediators in separate analyses. Drawing on work by MacKinnon and Dwyer (1993), we estimated additional models building on model 3 in which we included each of these potential mediators in turn. To determine whether the effect of parent alcoholism diminishes once controlling for each mediator, we first rescaled regression coefficients from models 3 and 3a (which included drinking frequency) and from models 3 to 3b (which included externalizing symptoms) to put the coefficients in a comparable scale (following Winship & Mare, 1983, 1984). To determine whether the COA coefficient diminished with the inclusion of the mediator, we used bootstrapping techniques to create confidence intervals for the difference between the two coefficients. In model 3a, the effect of drinking frequency at initiation did not attain statistical significance and the bootstrapped confidence interval for the difference in the COA effect included zero. Thus there was no support for the hypothesis that this variable was a mediator of the COA telescoping effect. In contrast, the effect of externalizing behavior in model 3b was statistically significant, indicating that adolescents rated higher in externalizing had higher hazard rates for alcohol abuse and dependence. However, the confidence interval for the associated reduction in the COA effect again included zero, providing no support for the hypothesis that this effect of externalizing behavior mediates COA telescoping.[7]

Telescoping in drinking versus consequences

At least two plausible explanations may account for these telescoping effects in COAs. First, COAs may show an accelerated progression from drinking onset to first experiencing diagnostic symptoms of alcohol disorders because they have a greater propensity to experience consequences and dependency symptoms at the same level of drinking as non-COAs. Second, COAs may show this accelerated progression because they have more sharply accelerating consumption trajectories so that they experience symptoms at typical levels of consumption but it is their consumption that is telescoped. Although the nature of our data did not permit a

[7]In post-hoc analyses, we also tested the potential mediators of children's' internalizing symptoms, peers' substance involvement and parenting (i.e., parental consistency and monitoring). The potential roles of internalizing symptoms and parenting were suggested by findings from Stice, Barrera and Chassin (1998) showing that internalizing symptoms exacerbated and parental support buffered the risk for greater subsequent alcohol-related consequences as predicted by alcohol consumption. Internalizing symptoms may impact how adolescents respond to alcohol (i.e., with greater despondence or withdrawal) and the pattern of their involvement, leading to more socially notable consequences. Parental support, on the other hand, may create a familial environment more tolerant of minor transgressions involving alcohol use and thus reduce conflict and other potential negative consequences related to drinking. However, no support for these mediators was found in the current study.

test of consumption trajectories as mediators of telescoping, we were able test whether our data are consistent with this possibility by comparing the drinking frequency of COAs and non-COAs at the wave of assessment immediately following their age of onset of abuse/dependence. Though COAs show a higher drinking frequency at the point of drinking initiation ($t(212) = 3.13$, $p < .01$), they did not differ from non-COAs in drinking frequency at the point of disorder ($t(110) = -1.00$, $p = .30$). Thus, despite their accelerated drinking trajectories, COAs report similar levels of drinking as controls at the point of disorder.

Heterogeneity in COAs' telescoping effect

We performed two tests of heterogeneity in COAs' risk for telescoping in alcohol onset-to-disorder trajectories. First, we tested whether the recency of parent alcoholism impacted COAs' risk for telescoping. Children whose mother or father reported symptoms consistent with alcohol abuse or dependence within the previous year in any of the three adolescent interviews were considered children of 'active alcoholic' parents, whereas all other COAs were considered children of 'inactive alcoholic' parents. These two groups were contrasted with children of never alcoholic parents, and both groups of COAs showed a greater hazard for disorder onset as compared to controls ($b = 1.01$, $\chi^2(1) = 12.36$, $p < .001$ for active and $b = 1.21$, $\chi^2(1) = 22.64$, $p < .0001$ for inactive, respectively; see model 1, Table 3). A planned contrast showed that children of active and inactive alcoholic parents did not differ significantly from one another ($\chi^2(1) = 0.59$, $p = .44$).

Second, we tested whether children whose parents reported both an alcohol disorder and a comorbid disorder (of either depression or antisociality) were at greater risk for telescoping as compared to children whose parents reported only an alcohol disorder or no psychopathology. Although children in both of the alcoholic parent groups showed greater telescoping as compared to controls ($b = 1.69$, $\chi^2(1) = 27.77$, $p < .001$ for comorbid and $b = 1.09$, $\chi^2(1) = 18.20$, $p < .001$ for non-comorbid, respectively; see model 2, Table 3), most importantly, those whose parents had a comorbid form of alcoholism showed a greater effect than those with only alcoholic parents ($\chi^2(1) = 4.42$, $p < .05$).

Telescoping in trajectories to drug disorders—To test whether the telescoping effect in COAs is specific to alcohol disorders versus more generally applicable to substance disorders, we repeated key analyses (models 1 and 3, in Table 2) testing whether COAs accelerate more quickly from drinking onset to drug disorder as compared to non-COAs. (We retained our focus on alcohol use at the point of initiation because drinking is typically a gateway event to other drug use and because of the small sample that onset drug use in waves one to three in this study, n=121). Given the low occurrence of drug disorders, particularly in non-COAs (n=10 of 84) as compared to COAs (n=38 of 135), we interpret these results with caution and present them as an initial examination of this question of specificity.

We estimated discrete-time hazard models that used the logit link function, where the logit of the hazard model was again best characterized by a quadratic function of years since drinking onset and COA status. (Observed hazard and survival rates against the fitted hazard and survival rates are shown in Figure 4). In the baseline model, we showed a significant telescoping effect of COA status on drug disorders ($b = 0.95$, $\chi^2(1) = 7.03$, $p < .01$; OR=2.59). This risk is similar in magnitude to that for alcohol disorders, though the overall odds for evidencing a drug disorder are significantly lower in both COAs and controls than the odds for evidencing an alcohol disorder. Similar to results of our analyses of alcohol disorders, this effect of COA status was maintained once we controlled for child gender, age at first interview, ethnicity and parental education ($b = 0.98$, $\chi^2(1) = 7.25$, $p < .01$; OR=2.65). No significant effect of gender or interaction between gender and age of drinking onset was found, although higher risk for having a drug

disorder was associated with a younger age of drinking onset during adolescence (b=−0.12, $\chi^2(1)=10.20$, p<.01; OR=.88).

Discussion

The current study shows that COAs, compared to their peers, have a telescoped pattern of alcohol involvement, escalating more quickly from initiation (in adolescence) to disorder. COAs' risk for telescoping was robust after controlling for age of drinking initiation and did not differ by gender or as a function of whether or not alcoholic parents were actively symptomatic. COAs whose alcoholic parents showed comorbidity for either depression or antisocial personality disorder, however, did evidence stronger telescoping effects than those whose alcoholic parents did not show comorbidity. Externalizing symptoms and early drinking patterns failed to explain COAs' risk for telescoped drinking onset-to-disorder trajectories, although externalizing symptoms were a unique predictor of telescoping in their own right. Moreover, COAs' risk for telescoping included a faster escalation from drinking initiation to the onset of drug disorder. Collectively, these findings depict a broad, significant impact of parent alcoholism on the course of drinking following initiation in adolescence, with an average of about four years separating the time to disorder for the first quartile of COAs progressing to the point of alcoholism versus seven years for their counterparts.

The current findings further clarify and integrate the pattern of risk for drinking outcomes consistently reported for COAs. Previous studies have characterized static outcomes to define this risk, showing younger ages of initiating drinking, greater rates of drinking beginning in early adolescence onward, and higher rates of alcohol disorders emerging with late adolescence (Chassin et al., 1991; Wong et al., 2006; Sher, 1991). They have also shown differences in developmental trajectories of drinking involvement, finding faster acceleration in drinking with development and more intense use over time in COAs than in children of non-alcoholic parents (Chassin et al., 1996; Chassin, Pitts & Proust, 2002). The current findings take into account these differences in the point of drinking initiation and the intensity of early drinking patterns to show that COAs progress more quickly from an adolescent initiation of alcohol use to the point of alcohol disorder. As such, we focus on COAs' risk for negative alcohol-related outcomes over time defined by drinking history rather than by developmental status or age alone.

These findings are striking in that they show telescoping effects in COAs to be independent of when or how (i.e., the level of alcohol use) drinking is initiated. These telescoping effects in COAs may result from a greater propensity to experience consequences and dependency symptoms at the same level of drinking as non-COAs and/or from accelerated consumption trajectories, so that they experience symptoms at typical levels of consumption but it is their consumption that is telescoped. Although we were unable to directly compare these two possibilities in the current study, the lack of differences between COAs and their peers in levels of drinking at the point of disorder along with previous evidence of more rapid escalations in consumption in COAs provide some support for accelerated consumption as underlying COAs' telescoping (Chassin et al., 1996; Chassin et al., 2002). However, we are cautious in drawing this conclusion for three reasons. First, our analyses were limited by an imprecise marker of drinking levels at the point of alcohol disorder and thus future work is needed to replicate this finding with methods better suited to make this distinction. Second, differences in early patterns of alcohol involvement (assessed close to the point of initiation) between COAs and non-COAs did not explain COAs' risk for telescoping. Although not a direct test of this hypothesis, this finding is consistent with the conclusion that different patterns of alcohol involvement do not alone account for telescoping. Third, evidence for accelerated drinking trajectories does not rule out the possibility that telescoping in COAs' drinking onset-to-disorder trajectories are also due to other factors, including different physiological risk for experiencing alcohol-related

consequences and dependency symptoms even at similar levels of consumption as non-COAs (e.g., stress-response dampening and lower physiological responsiveness to alcohol, Schuckit & Smith, 2000; Sher, 1991). Thus, the relative role of consumption and factors such as physiological response to alcohol as related to telescoping in COAs remains unclear and deserves further study.

Nonetheless, our results indicate that telescoping in COAs is not due to greater externalizing symptoms. Given that externalizing symptoms are a significant predictor of alcohol involvement and mediate COAs' risk for drinking in early adolescence (Zucker, 2006), they are clearly a marker of risky drinking involvement. Indeed, adolescents with greater externalizing symptoms prior to initiating drinking in adolescence also showed a telescoping effect in their drinking onset-to-disorder trajectories. Like COAs, young adults with greater externalizing symptoms may show greater responsivity to the psychopharmacological effects of alcohol (Hoaken, Campbell, Stewart, & Pihl, 2003; Sher & Walitzer, 1986). Thus, these findings are consistent with evidence that externalizing symptoms are themselves a significant marker for risky alcohol involvement, but they also indicate that other mechanisms are responsible for the telescoping effect in COAs.

In addition to our tests of mechanisms, we examined heterogeneity in COAs' risk for telescoping. COAs whose parents were active versus inactive alcoholics did not differ in telescoping, suggesting that direct exposure to problem drinking by the parent during the child's adolescent years (and related mechanisms such as modeling, greater access, and impaired parenting when intoxicated) is not a central factor impacting telescoping. However, COAs whose alcoholic parent evidenced comorbid disorders (of either depression or antisocial personality disorder) showed greater telescoping effects than COAs whose alcoholic parent did not evidence comorbidity. Previous research has also shown differential risk for internalizing symptoms, externalizing symptoms and social functioning deficits in children whose parents show such comorbid forms of alcoholism (Hussong et al., in press; Hussong et al., 2006; Hussong, Zucker, Wong, Fitzgerald, & Puttler, 2005; Puttler et al., 1998). Together, these studies may suggest meaningful phenotypic differences that emerge over development in alcoholism as a function of comorbid psychopathology (Zucker, 2006). Alternatively, comorbid forms of alcoholism may reflect greater impairment or a more chronic course of alcoholism, each of which may increase COAs' risk for telescoping trajectories via genetic transmission.

The risk for telescoping evidenced by COAs may also extend beyond the specific outcome of alcohol disorders to the broader class of disinhibitory disorder, as suggested by our finding that COAs show telescoping for drug disorders as well. Although externalizing symptoms failed to mediate the prediction of telescoping to alcohol disorders in COAs, externalizing symptoms were directly predictive of accelerated drinking onset-to-disorder trajectories. Thus, although other mechanisms may account for COAs' telescoping of alcohol disorders, COAs may face additional risk for telescoping of disinhibitory disorders due to their elevated rates of externalizing symptoms and behavioral undercontrol. Further exploration of non-substance based disorders, including affective disorders which index disturbance beyond the disinhibitory spectrum, will clarify the generality of COAs' risk for telescoping.

In addition to the effects of telescoping, a strong maturational trend was evident in the timing of alcohol disorder onset. Consistent with previous literature, peak rates for first evidencing an alcohol disorder across COAs and non-COAs occurred in early adulthood around age 18 (Kessler et al., 2005). The relative freedoms accompanying the transition to adulthood, such as leaving home, in conjunction with potential entry into heavy drinking environments, such as college or young adult peer groups, may at least in part account for this trend. However, a substantial proportion of those evidencing disorder at this time are expected to offset within a

few years, showing a developmentally limited pattern of alcohol abuse (Zucker, 2006). Defining the role of telescoping in those who persist versus desist in problematic alcohol involvement subsequent to early adulthood is thus an important area for future research, requiring follow-up of participants into adulthood.

Although not impacting the relation between parent alcoholism and alcohol disorder, gender differences were evident in the incidence of alcohol disorder. These effects were moderated by the age of drinking initiation, such that girls who onset drinking earlier showed greater risk for developing an alcohol disorder at every age of assessment (see Figure 3) and over a more prolonged period of risk. For boys, later drinking onset predicted greater risk for incidence of alcohol disorder (after mid-adolescence). The greater risk for alcohol disorders found for early onset drinkers in girls is consistent with previous literature (Grant & Dawson, 1997) and the more prolonged period of risk for evidencing an alcohol disorder may reflect an interaction among underlying biological propensities for alcohol responsivity. For example, Varlinskaya and Spear (2006) showed that older adolescent, female rats have a greater sensitivity to the anxiolytic benefits of alcohol as compared to males and younger adolescent females. If such physiological sensitivities to alcohol are heightened with maturation in females, then those who are already experienced drinkers may increase their drinking involvement to experience these newly enhanced mood-regulating benefits of alcohol. For boys, however, our finding of a greater incidence of alcohol disorder among those who initiate drinking later in adolescence is not consistent with this literature. Importantly, post-hoc analyses showed a non-linear pattern of survival for boys, such that boys who initiate drinking early or after-adolescence (if at all) are less likely to first evidence an alcohol disorder than boys who do so in mid- to late-adolescence. Although previous studies of drinking initiation and risk for disorder have examined a longer period of drinking onset, none have considered non-linear relations. To reconcile these findings, we need long-term assessments that can consider later ages of drinking onset and disorder, particularly as a function of gender, as well as the potential for non-linear relations between age of drinking onset and risk for alcohol disorders.

Telescoping within the first five years after drinking onset was strongest in those who began drinking later (around age 14 or later) rather than earlier in adolescence, though the pattern was more pronounced in boys than in girls. One explanation for telescoping in later-onset adolescent drinkers is that the socio-cultural context supporting alcohol use may intensify pressures for heavy drinking in mid- to late-adolescence, potentiating risk for negative drinking consequences in naïve drinkers. At this time, peer drinking becomes more normative and observable, alcohol becomes easier to access and more central to social activities, heavy drinking becomes more acceptable and encouraged (Johnston, O'Malley, Bachman, & Schulenberg, 2006), and maturational strivings to imitate adult-like behaviors, such as heavy drinking, become stronger. As a result, adolescents who begin drinking later in adolescence may escalate more quickly in their alcohol use and become vulnerable to alcohol-related consequences. As noted earlier, however, some portion of problem drinking at the late adolescent/ early adulthood is developmentally limited, and will end with the assumption of adult roles (Zucker, 2006). Thus, the complex relation between age of onset and telescoping of alcohol disorders (i.e., moderated by gender and non-linear in boys) likely reflects the combination of the late adolescent social contextual risk for heavy drinking and individual vulnerabilities. Moreover, the fact that age of onset showed simpler relations for telescoped drug diagnoses (i.e., earlier onset associated with greater telescoping) is consistent with this interpretation, given that the extent of social context pressures for illegal drug use is likely to be somewhat less prevalent and intense then for alcohol use. In addition, the lower number of drug diagnoses compared to alcohol diagnoses also reduces the statistical power to detect such complex interactions.

The important contributions of the current findings should be qualified by study limitations. Although the study design permitted less reliance on retrospective reporting than in previous investigations, greater precision in identifying the timing of drinking initiation and disorder onset are possible through more intensive designs aimed at mapping the early course of drinking and significant mile markers along the way to disorder. This is particularly important in light of the modest correspondence across reported ages of drinking and disorder onset in the current study. In addition, we focused our analysis on those who initiated drinking in early to middle adolescence and onset disorder by their mid- to late-20′s (although our survival curves indicated little new onset of alcohol disorder past age 23 in this sample). Although capturing an important risk period for initiation, the study does not permit comparisons across individuals with a much larger window of drinking initiation or conclusions about whether there are differences in telescoping for developmentally limited versus more persistent forms of alcohol disorder. Nor does the study capture the onset of alcohol disorders beyond young adulthood. Finally, the sample size may also have resulted in limited power for sub-group analyses (i.e., comparing active versus inactive alcoholic parents) and tests of mediation. Nonetheless, the longitudinal assessments of COAs from early adolescence through young adulthood examined here are unique in the field and thus offer a significant opportunity for understanding the drinking histories of at-risk adolescents.

In conclusion, we found that COAs are at significant risk for evidencing alcoholism more rapidly following the initiation of drinking than are their peers. Whether this effect of telescoping has implications for other alcohol-related consequences (such as the occurrence of alcohol-related disease) is not yet known, but current evidence that this risk extends to the outcome of drug disorders as well are consistent with a broad-based risk for telescoping effects in COAs. Such questions form an important agenda for future research. These findings underscore the importance of targeting COAs early in their drinking histories to prevent alcohol disorder.

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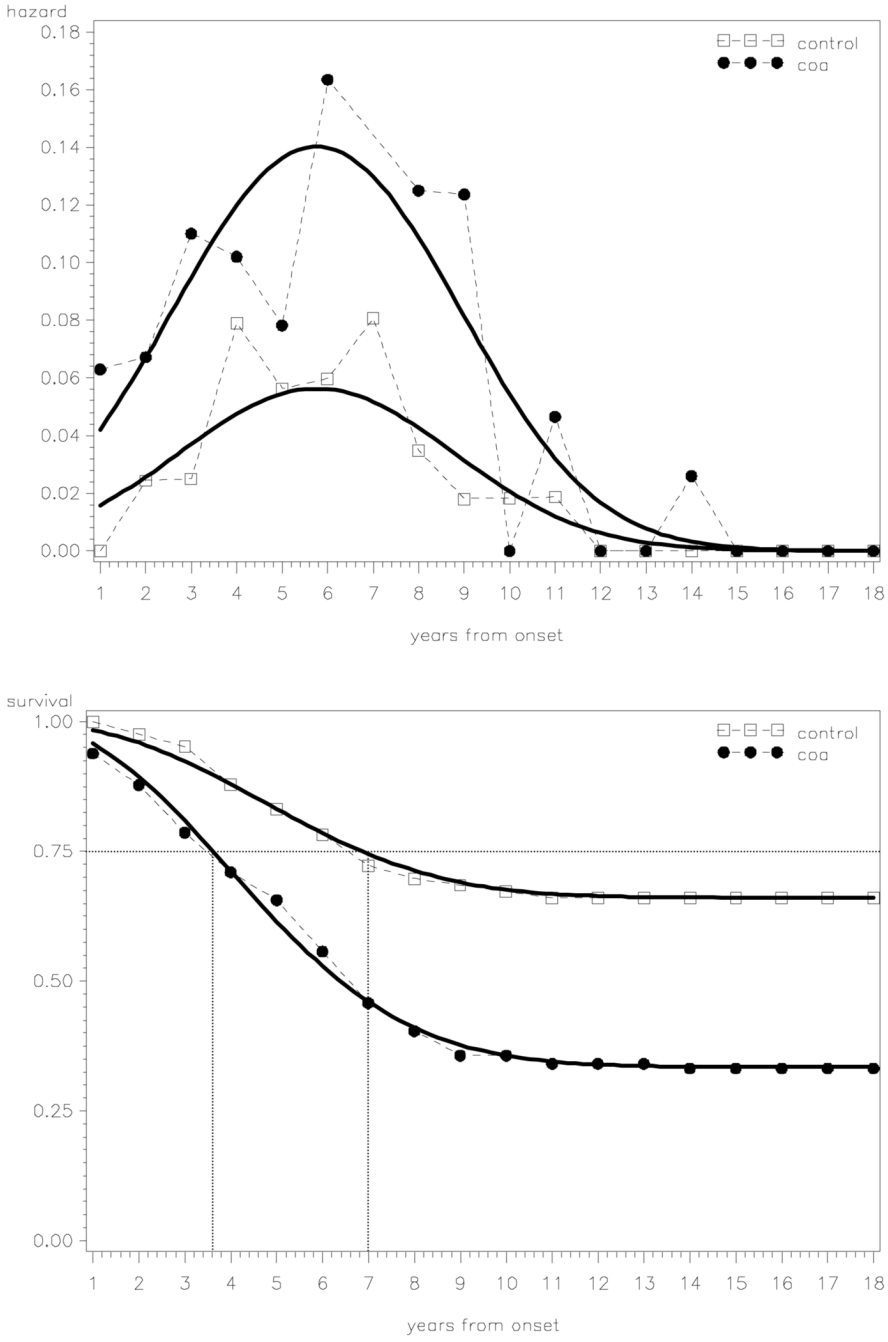

Figure 1.
Observed Hazard and Survival Rates for Alcohol Disorders against the Fitted Hazard and Survival Rates

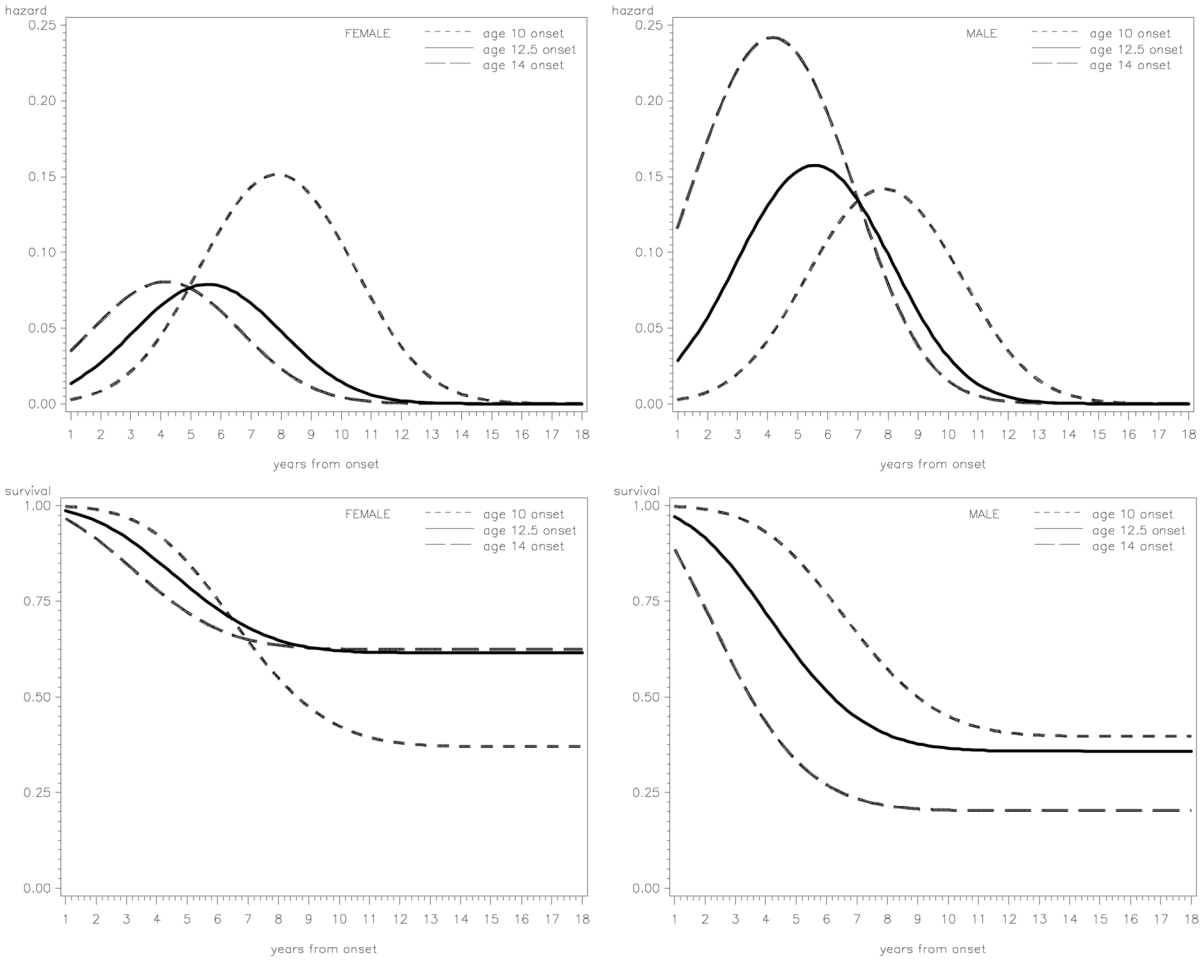

Figure 2.
Hazard and Survival Rates for Alcohol Disorders by Gender, Period, and Age of Drinking Initiation

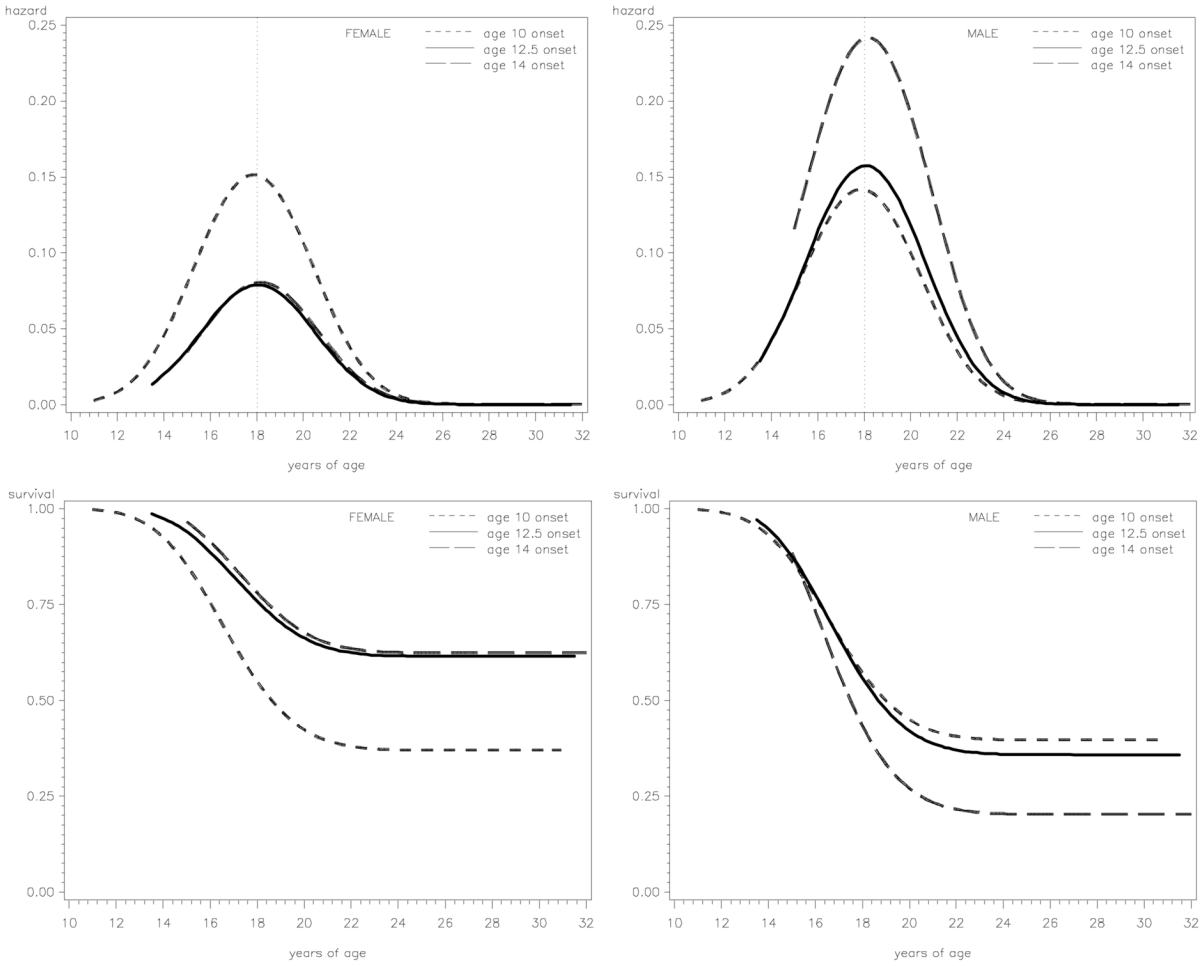

Figure 3.
Hazard Rates for Alcohol Disorders by Gender, Age and Age of Drinking Initiation

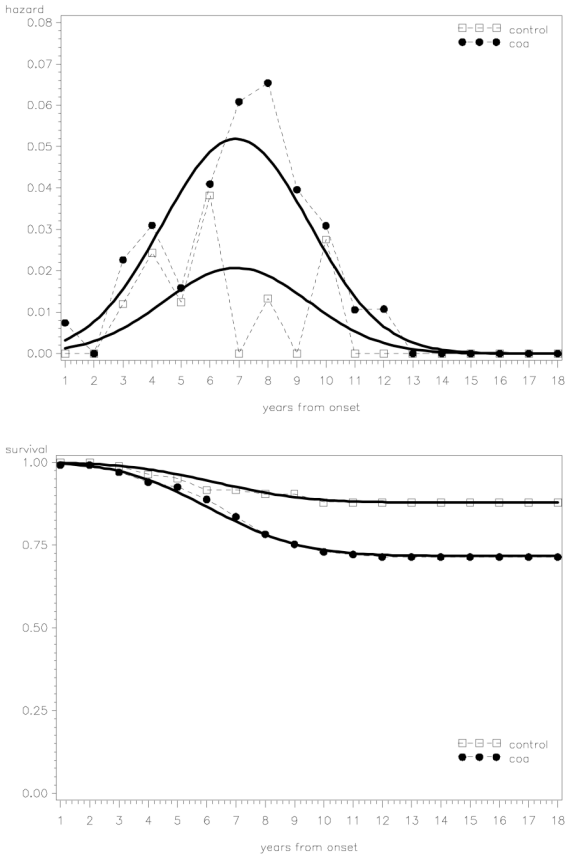

Figure 4.
Observed Hazard and Survival Rates for Drug Disorders against the Fitted Hazard and Survival Rates

Table 1

Life Tables Describing Event Occurrence by Risk Group for Alcohol Disorder

Years from Onset	Time Interval	Frequency						Proportion of			
		Drinkers at beginning of year (Risk Set)		Number who met criteria for diagnosis during year		Number censored at the end of the year		Drinkers who were diagnosed during year (Hazard Function)		All drinkers not diagnosed by end of the year (Survival Function)	
		Non-COA	COA	Non-COA	COA	Non-COA	COA	Non-COA	COA	Non-COA	COA
0	[0,1)	83	131	-	-	-	-	-	-	1.0000	1.0000
1	[1,2)	83	131	0	8	0	0	0.0000	0.0611	1.0000	0.9389
2	[2,3)	83	123	2	8	0	0	0.0241	0.0650	0.9759	0.8779
3	[3,4)	81	115	2	12	0	0	0.0247	0.1043	0.9518	0.7863
4	[4,5)	79	103	6	10	0	0	0.0759	0.0971	0.8795	0.7099
5	[5,6)	73	93	4	7	0	0	0.0548	0.0753	0.8313	0.6565
6	[6,7)	69	86	4	13	0	0	0.0580	0.1512	0.7831	0.5573
7	[7,8)	64	73	5	13	1	0	0.0781	0.1781	0.7220	0.4580
8	[8,9)	58	59	2	7	1	1	0.0345	0.1186	0.6971	0.4037
9	[9,10)	56	51	1	6	0	1	0.0179	0.1176	0.6846	0.3562
10	[10,11)	55	44	1	0	0	1	0.0182	0.0000	0.6722	0.3562
11	[11,12)	54	44	1	2	0	0	0.0185	0.0455	0.6597	0.3400
12	[12,13)	53	42	0	0	0	0	0.0000	0.0000	0.6597	0.3400
13	[13,14)	52	42	0	0	2	0	0.0000	0.0000	0.6597	0.3400
14	[14,15)	49	39	0	1	4	6	0.0000	0.0256	0.6597	0.3313
15	[15,16)	42	31	0	0	10	8	0.0000	0.0000	0.6597	0.3313
16	[16,17)	31	22	0	0	12	10	0.0000	0.0000	0.6597	0.3313
17	[17,18)	18	12	0	0	14	9	0.0000	0.0000	0.6597	0.3313
18	[18,19)	7	6	0	0	7	4	0.0000	0.0000	0.6597	0.3313
19	[19,20)	2	2	0	0	4	4	0.0000	0.0000	0.6597	0.3313

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Table 2

Odds Ratios from Hazard Models Comparing COAs to Controls on Alcohol Disorder

Predictors	Baseline Model		Model 1		Model 2		Model 3		Model 3a		Model 3b	
	OR	χ^2	OR	χ^2	OR	χ^2	OR	χ^2	OR	χ^2	OR	χ^2
Period	1.74	23.95***	1.76	25.17***	1.80		2.25			33.54***	2.30	35.01***
						26.39***		33.55***	2.25			
Period^2	0.94	28.64***	0.94	28.56***	0.94		0.91			37.70***	0.91	38.10***
						29.56***		37.84***	0.91			
COA	2.74	20.08***	3.02	22.60***	3.08		3.10			20.42***	2.96	20.55***
						23.18***		22.94***	3.01			
Gender			1.98	11.01***	2.24		2.18			12.98***	2.14	12.18***
						13.66***		12.95***	2.18			
Age at First Interview			1.11	1.96	1.04	0.16	1.06	0.42	1.03	0.15	1.06	0.42
Ethnicity			0.75	1.60	0.73		0.68	2.70		2.78	0.67	2.95
						1.81			0.67			
Parent Education			1.14	1.68	1.14	1.56	1.11	0.95	1.10	0.81	1.08	0.54
Age of Drinking Onset					1.14	3.12	1.91	14.58***	1.93	14.90***	1.95	14.89***
Age of Drinking Onset × Gender							1.41	7.44**	1.39	6.39*	1.44	8.24**
Age of Drinking Onset × Period							0.85	37.34***	0.85	35.87***	0.84	37.66***
Drinking Frequency									1.06	0.31		

Predictors	Baseline Model		Model 1		Model 2		Model 3		Model 3a		Model 3b	
	OR	χ^2	OR	χ^2	OR	χ^2	OR	χ^2	OR	χ^2	OR	χ^2
Externalizing Behavior											2.12	13.66***

Note: Period refers to the linear effect of years since onset and Period^2 refers to the quadratic effect.

* p < .05

** p < .01

*** p < .001.

Table 3

Odds Ratios from Hazard Models Comparing COA subtypes to Controls on Alcohol Disorder

Predictors	Model 1		Model 2	
	OR	χ^2	OR	χ^2
Period	2.26	33.80***	2.26	32.11***
Period^2	0.91	37.96***	0.92	35.36***
Active Parent Alcoholism vs. Control	2.75	12.36**		
Inactive Parent Alcoholism vs. Control	3.34	22.64***		
Comorbid Parent Alcoholism vs. Control			5.40	27.77***
Non-Comorbid Parent Alcoholism vs. Control			2.96	18.20***
Gender	2.22	13.39**	2.51	16.25***
Age at First Interview	1.05	0.29	1.04	0.15
Ethnicity	0.67	2.88	0.71	1.91
Parent Education	1.11	1.02	1.08	0.54
Age of Drinking Onset	1.93	14.92***	2.06	16.63***
Age of Drinking Onset × Gender	1.41	7.32**	1.43	7.11**
Age of Drinking Onset × Period	0.85	37.02***	0.84	35.91***

Note: Period refers to the linear effect of years since onset and Period^2 refers to the quadratic effect.

* $p < .05$

** $p < .01$

*** $p < .001$.