Published in final edited form as:
Psychol Addict Behav. 2014 June ; 28(2): 322–335. doi:10.1037/a0035488.

Alcohol use disorder in women: Risks and consequences of an adolescent onset and persistent course

Katherine T. Foster, B.A.[1,2] and **Brian M. Hicks, Ph.D.**[1]

[1]Department of Psychiatry, University of Michigan

[2]Department of Psychology, University of Michigan

William G. Iacono, Ph.D. and **Matthew McGue, Ph.D.**
Department of Psychology, University of Minnesota

Abstract

Women are more vulnerable to the deleterious effects of both acute and protracted alcohol use than men, but women's lower levels of alcohol consumption and alcohol use disorder (AUD) have resulted in a paucity of investigations on the development of alcohol problems in women. In particular, it is not clear to what extent the cascading effects of key etiological factors that contribute to an especially severe course of AUD in men also underlie the development of AUD in women. To fill this gap, we examined the adolescent risk factors and adult consequences associated with an adolescent onset and persistent course of AUD in a community sample of women (n=636) from ages 17 to 29. Women with AUD exhibited greater psychopathology and psychosocial impairment than those without, with an adolescent onset and persistent course indicative of the greatest severity. Notably, high levels of impairment across all women with AUD reduced the utility of onset and course to differentiate profiles of risk and impairment. In contrast to previous work in men, even women whose AUD symptoms desisted continued to exhibit impairment, suggesting that an adolescent onset of AUD is associated with enduring consequences for women's health and functioning, even after ostensible “recovery.”

Keywords

Alcohol Use Disorder; Adolescent Risk Factors; Adult Outcomes; Women

Alcohol use disorders (AUD; DSM alcohol abuse and dependence) constitute major public health and safety problems. Among the general United States population, AUD is more prevalent among men (abuse, 24.6%; dependence, 17.4%) relative to women (abuse, 11.5%; dependence, 8.0%) (Keyes et al., 2008). Over the last 70 years, however, the male to female ratio has decreased from 7:1 to 2:1 for abuse and from 5:1 to 2:1 for dependence (Keyes et al., 2008). For both men and women, drinking alcoholic beverages typically begins in adolescence, escalates through the mid-20s, and declines to moderate levels in adulthood (Chassin et al., 2004; Chen & Kandel, 1995; Johnstone et al., 1996). Trends in AUD

Correspondence: Katherine T. Foster, *Addiction Research Center*, Rachel Upjohn Building, 4250 Plymouth Road, Ann Arbor, MI 48109, Phone: 734.998.7454, ktfoster@umich.edu.

prevalence follow a similar developmental pattern for both genders as well, with disordered drinking emerging in late adolescence, peaking in the mid-20s, and declining thereafter. In men, the risks and consequences associated with AUD vary as a function of the onset and course of alcohol problems (Leggio et al., 2009). Specifically, deviation from a normative developmental pattern of alcohol use—especially an adolescent onset and persistence of AUD past young adulthood—has been linked with more severe alcohol dependence and greater polysubstance use, antisocial behavior, and psychosocial impairment in men (Hicks et al., 2010). The patterns of risk and consequences associated with the onset and course of AUD among women, however, remain less clear.

AUD in Women: A more severe disorder than in men?

Due to an underrepresentation of women in AUD research (Nolen-Hoeksema, 2004), less is known about the risk factors for and consequences of AUD specific to women. To some extent, AUD+ women have received less attention because women exhibit lower levels of exposure to risk factors for AUD. In particular, women have higher rates of abstinence (Cotto et al., 2010), more desistence from alcohol use (Wilsnack et al., 2009), and lower levels of alcohol consumption (Olenick & Chalmers 1991; Ross, 1989; Wilsnack et al. 2009). Despite the lower frequency of heavy drinking and alcohol problems among women, more severe biopsychosocial consequences of both short term alcohol consumption (Grant et al., 2012; Stockwell et al., 2002) and protracted heavy alcohol use (Acker, 1986; Brown & Tapert, 2004; Hommer et al., 1996; Mann et al., 1992; Niaura et al., 1987; Nixon, 1994) are evident in women than men. Gender differences in the negative consequences of drinking have routinely been conceptualized as protective factors reducing rates of AUD in women (Nolen-Hoeksema & Hilt, 2006). The women who do develop AUD, however, may do so along a unique etiological pathway, and tend to exhibit a more severe symptom profile relative to their male counterparts. For example, women progress faster from the onset of drinking to dependence and treatment than men (i.e., telescoping; Diehl, et al., 2007; Hernandez-Avila et al., 2004; Piazza et al., 1989; Randall et al., 1999; Schuckit et al., 1995), and AUD+ women exhibit higher rates of comorbid externalizing and internalizing disorders compared with AUD+ men (Dawson et al., 2010), suggesting a higher risk loading and greater impairment in functioning. These findings are consistent with the notion that AUD may represent a more severe form of psychopathology in women relative to men.

Leveraging Knowledge of AUD in Men

Studies focused on AUD in women can leverage what is known about men with AUD to examine the similarities and differences in the development of AUD in women. AUD has a relatively late onset and so is typically conceptualized as the endpoint in a long history of exposure to multiple, interacting risk factors. Of these risk factors, behavioral disinhibition appears a key etiological pathway for severe AUD in men (Iacono et al., 2008). Behavioral disinhibition refers to a number of correlated behaviors, personality traits, and psychiatric disorders which reflect an inability to restrict socially undesirable behavior (Caspi et al., 1996; Iacono et al., 2008; Krueger et al., 2007; Slutske et al., 2002). A number of longitudinal studies have confirmed that disinhibited children are more likely to develop adolescent externalizing problems such as alcohol and illicit substance misuse and antisocial

behavior (Caspi et al., 1995, 1996; Masse & Tremblay 1997; Wong et al. 2006). As such behavioral disinhibition is a non-specific risk factor that affects multiple domains. For example, disinhibited children often strain the family system and exhibit significant dysfunction in school and social contexts (Granic & Patterson, 2006). Subsequent peer rejection and academic difficulties have been linked with shame and depressed mood (Patterson & Capaldi, 1990; Tangney, Miller, Flicker, & Barlow, 1996) that further strains the parent-child relationship (Patterson, 1982; Patterson et al., 1992). Disinhibited children then tend to join deviant peer networks and begin exhibiting the delinquent behavior modeled by these peers including early initiation of alcohol and drug use (Dishion, Patterson, Stoolmiller, & Skinner, 1991; Snyder, Dishion, & Patterson, 1986). Continued use of alcohol and other substances is then incentivized through biological reward systems and the social environment, increasing the likelihood of early onset substance use disorders. While behavioral disinhibition is highly heritable (Kendler et al., 2003; Krueger et al., 2002; Young et al., 2000), the interaction of these risks implicates a number of biological and contextual risk factors that accumulate over the course developmental that lead to disordered alcohol use in adulthood (Blazei et al., 2006; Hicks et al., 2010).

Studies focused on developmental trajectories of AUD (i.e., onset and course) have also been helpful in differentiating risk profiles for AUD including a type associated with behavioral disinhibition (Babor et al., 1992; Clark et al., 1998; Cloninger, 1987; Zucker et al., 1994). AUD that onsets in adolescence and persists into the late 20's is associated with especially poor outcomes (Clark, 2004; Hicks et al., 2010), including comorbid substance use and abuse, antisocial behavior, and various indicators of psychosocial impairment in adolescence (Hicks et al., 2010). Using a community sample of male twins, Hicks et al. (2010) found that measures of behavioral disinhibition in adolescence were the most consistent predictors of a persistent course AUD and associated polysubstance abuse, psychiatric problems, and poor psychosocial adjustment out to age 29. The combination of an adolescent onset and persistent course of AUD was associated with the greatest risk profile at age 17 and the worst outcomes at age 29. Desistence of symptoms by age 29, regardless of onset, largely—though not entirely—mitigated the negative consequences of AUD. Notably, men who had an adult onset and desisted by age 29 experienced few long-term negative consequences of their AUD, and seemed to represent a developmentally-limited, remitting type (Sher & Gotham, 1999; Zucker et al., 1994). These findings illustrate that the onset and course of AUD can differentiate the patterns of risk and consequences associated with the disorder, and further clarify the role of behavioral disinhibition as a developmental pathway to AUD.

Extending the Developmental Risk Accumulation Model to Women with AUD

It remains unclear, however, whether onset and course can differentiate profiles of risk and consequences of AUD in women, and if the behavioral disinhibition pathway validated in men generalizes to women. The lower prevalence rate in women and their greater sensitivity to alcohol's effects suggests that women may require a higher loading of risk factors to express AUD. For instance, women on average are less disinhibited than men. Therefore,

women who develop AUD as a result of the accumulated risk associated with behavioral disinhibition represent a more deviant group relative to same gender non-AUD individuals than men with AUD. Consequently, these women are also likely to experience more problems in functioning, either due to AUD itself or to the accumulated risk associated with AUD. Further, this risk accumulation may also operate in gender-specific ways. For example, disordered eating patterns (Rosval et al., 2006; Vitousek et al., 1994; Westen & Harnden-Fischer, 2001) and reproductive health risk behavior (Kahn et al., 2002) have also been linked with behavioral disinhibition in women. The accumulation of risk associated with AUD, then may produce lasting impairment that extends beyond ostensible "recovery" from AUD (i.e., desistence from symptoms). That is, due to this high loading of risk, remission of AUD symptoms alone may be insufficient to attain normative functioning. As a consequence, if desistence of symptoms fails to lead to full recovery of function, onset and course of AUD may not differentiate risk profiles as effectively as they have in men.

Similar to our prior study in men (Hicks et al., 2010), we examined the differential associations between an adolescent onset and persistent course of AUD in women with adolescent risk factors and adult outcomes using a large community sample followed from age 17 to 29. We organized our analyses around the framework of a behavioral disinhibition pathway to AUD with its associated person-level and contextual risk factors. Risk factors were grouped under the broad categories of substance use and mental health problems, psychosocial functioning, and environmental risk and protective factors. We also examined links with adult psychosocial outcomes and indices of the successful transition to adult roles. Whenever possible, we used multiple measures to assess different constructs, focusing on the patterns of effects across multiple domains of functioning rather than relying on any single measure or statistically significant hypothesis test.

We hypothesized that AUD in women would be associated with a high accumulation of risk exposure, that is, higher mean-levels of risk exposure and functional impairment relative to non-AUD controls. We also hypothesized that an adolescent onset and persistent course of AUD would be associated with the greatest severity. Further, we expected that the high-level of risk accumulation needed for women to express AUD would reduce the utility of onset and course to differentiate risk and outcome profiles. Specifically, AUD among women would be associated with enduring risk and consequences, even after AUD symptoms desisted.

Method

Sample

Participants were 674 female twins taking part in the Minnesota Twin and Family Study (MTFS; for extensive details on study design see Iacono et al., 1999, 2006), a longitudinal, community study designed to examine the etiology of substance use disorders. Participants began the study at age 17 and then completed follow-up assessments every 3-5 years. Minnesota public birth records were used to recruit all twin pairs born between the years of 1975 and 1979. Over 90% of families were located, and more than 80% of located families completed an in person assessment at the university laboratory. Nearly all participants reported European American ancestry (96%; Iacono et al., 1999) and did not differ from

non-participating families on parental occupation, education, and history of mental health treatment.

Assessment

At the age 17 intake assessment, multiple informants (participants, parents, and teachers) were utilized to conduct a relatively comprehensive assessment of psychiatric, psychosocial, and environmental functioning. Follow-up assessments were then conducted at the target ages of 20 (n = 630; 93.5%), 24 (n = 634; 94.1%) and 29 (n = 636; 94.4%). Portions of the assessment battery are briefly described below, with extensive detail of identical methods provided in prior publications (Hicks et al., 2009, 2010). A large number of measures were used to assess risk factors and domains of functioning, focusing on patterns of effects (rather difference on any single measure) to evaluate the validity and reliability of potential differences across AUD groups that differed in onset and course.

AUD Diagnosis

Trained staff administered the Substance Abuse Module (SAM; Robins & Cottler, 1987) of the Composite International Diagnostic Interview (Robins et al., 1988) to determine lifetime AUD status at age17. Subsequent evaluations assessed the presence of AUD symptoms since the last visit. Consistent with the previous study of male twin participants of the MTFS (Hicks et al., 2010), AUD was defined as 2 or more DSM-III-R symptoms (rather than 3) to ensure detection of early onset cases and to increase statistical power. Prior MTFS studies have also demonstrated the validity of this approach (Elkins et al., 2004; Elkins et al., 2006; Elkins, McGue, & Iacono 2007; McGue & Iacono, 2005), and item response theory analyses have demonstrated that symptoms of alcohol abuse and dependence are best conceptualized as unidimensional trait without loss of sensitivity or specificity for detection of alcohol problems (Borges et al., 2010; Krueger et al., 2004). AUD+ participants were grouped based on onset (adolescent vs. young adult) and course (persistent vs. desistent). Adolescent onset (AO) required AUD by age 17, while young adult onset (YAO) required AUD by age 20 or 24. Persisting course (P) required AUD at age 29 and at a previous assessment. Desisting course (D) required the absence of any symptoms at age 29 after having a diagnosis at a previous assessment. Participants were then assigned to one of four groups: adolescent onset, desisting course (AO-D; n = 33); adolescent onset, persisting course (AO-P; n = 14); young adult onset, desisting course (YAO-D; n = 43); young adult onset, persisting course (YAO-P; n = 27). Each AUD group was also compared to a control group of participants who never met criteria for an AUD (n = 449) between ages 17 and 29. This procedure classified 90% of women that completed the age 29 assessment. The remaining participants were excluded from analysis due to either an especially late onset (at age 29) or because it was difficult to determine the course of their AUD (e.g., 1 symptom of AUD at age 29 as well as an AUD diagnosis at a previous assessment).

Substance Use, Antisocial Behavior/Disinhibition, and Mental Health Problems

Alcohol, nicotine, and drug use patterns and DSM-III-R symptoms of abuse and dependence were assessed using the SAM. Alcohol use measures included age at first drink without parental permission, past year alcohol consumption quantity (standard drinks per occasion), and frequency (ranging from abstinence to multiple drinks per day), total number of

intoxications (log transformed to correct for positive skew), and the maximum number of drinks consumed in 24 hours.

Nicotine use included dependence symptoms, along with quantity (cigarettes per day) and frequency (days per month) of smoking. Symptoms of illicit drug abuse and dependence were assessed for marijuana, amphetamine, cannabis, cocaine, hallucinogen, inhalant, opioid, PCP, and sedatives; the highest number of symptoms reported for any drug class was used for each participant's drug dependence variable. Participants also rated their access to substances and reported their marijuana use (log transformed) and the number of different drug classes tried (0-4+ age 17 and 0-5+ at age 29).

Adult and child antisocial behavior were assessed using a structured interview similar to the antisocial personality disorder module of the SCID-II. Symptom measures were augmented with scores on the Delinquent Behavior Inventory (DBI; $\alpha = 0.96$; Taylor et al., 2000), the behavioral disinhibition scale ($\alpha = 0.67$; Taylor et al., 2000), and a composite of early adolescent problem behavior (sum of alcohol, nicotine, and illicit drug use and police contact, and initiation of sexual intercourse before age 15; range 0 to 5). Symptoms of major depression and eating disorders were assessed using the Structured Clinical Interview for DSM-III-R. Additional measures of mental health included history of mental health problems (i.e., prior suicide attempts and treatment or hospitalization) and a teacher report of internalizing distress (Cronbach's $\alpha = 0.85$).

Personality

Personality was assessed using the Multidimensional Personality Questionnaire (MPQ; Tellegen and Waller, 2008), a well-validated measure that includes 3-higher order factors: Positive Emotionality (overall well-being and tendency to enjoy social engagement), Negative Emotionality (propensity to breakdown under stress and have a suspicious and aggressive interpersonal style), and Behavioral Constraint (inclination toward planning, traditional social values, and caution).

Academic and Intellectual Functioning

Both the participant and their mother reported on academic functioning using the Academic History Questionnaire (Johnson et al., 2006). Items included cumulative grade point average (GPA), expected level of academic attainment (e.g., complete high school, complete college), positive school attitudes (e.g., “has a good attitude about school”, “enjoys attending school”; $\alpha = 0.83$), and disciplinary problems (detention, sent to principal's office, sent home, suspended, expelled; $\alpha = 0.85$). Verbal (vocabulary and information), performance (block design and picture arrangement), and full scale IQs were estimated using the Wechsler Adult Intelligence Scale–Revised (WAIS-R; Wechsler, 1981).

Parent and Family Characteristics

Socioeconomic status for the family of origin was estimated using the mean z-score for mother's and father's years of education, occupational status (Hollingshead Index) and annual income. Parental externalizing disorders were a composite (mean z-score) of symptoms of antisocial personality disorder, and alcohol, nicotine, or drug abuse/

dependence. Parent-child relationship quality was assessed using the Parental Environment Questionnaire (PEQ; Elkins et al., 1997). Parents provided independent ratings for both their own and the other parent's relationship with each child, and twins completed ratings of the quality of their relationship with each parent (scores were the mean z-score of the three informant ratings for the first principle component of the PEQ scales; Hicks et al., 2009).

Stressful Life Events

Family-level stress was assessed using 18 items (financial, legal, marital and mental health difficulties) from the Life Events Interview (LEI; reliability as estimated by agreement across twin reports was 0.89). Other stressful events included school problems (changing schools, failing a course, repetition of an academic grade, summer school, suspension or expulsion, and worry about school performance) and legal and alcohol/drug problems (police contact other than for traffic violations, jail sentence, and alcohol or drug problems). At age 29, individual-level financial problems included receiving money from a government agency, losing a job, 6+ months of unemployment, financial problems in adulthood, and a history of bankruptcy.

Peers

Self-report questionnaires (19-items at age 17; 27-items at age 29) were used to characterize a participant's peer group on dimensions of antisocial ($\alpha = 0.82$; e.g., my friends enjoy getting drunk, get into fights, can't seem to hold a job) and prosocial behaviors ($\alpha = 0.60$; e.g., my friends work hard, do volunteer work, have a regular job).

Social Outcomes

At age 29, the LEI and Social Adjustment Interviews were used to assess psychosocial functioning including educational attainment, occupational status (Hollingshead index), and annual income (U.S. dollars before taxes, capped at ≤ $250,000 to reduce positive skew), and role transitions (number of sex partners, cohabitation with a romantic partner, marriage, and parenthood).

Romantic Partner's Drinking and Attitudes

Participants who were married, cohabiting, or consistently dating the same person for 3 months or more, reported their romantic partner's past year drinking patterns (mean z-score for frequency, quantity, and proportion of intoxicating drinking episodes) and attitudes toward substance use (e.g., “my spouse/partner would be upset if he knew I was smoking”; “my spouse/partner would purchase alcohol if I asked him to”; “my spouse's/partner's friends use marijuana”) using an 11-item scale ($\alpha = 0.84$).

Statistical Analysis

A 2 × 2 ANOVA was used to compare the 4 AUD groups on Onset (adolescent vs. young adult), Course (persistent vs. desistent), and the Onset × Course interaction. Due to the number of statistical tests, only effects with p-values < 0.01 are reported as statistically significant. Interaction terms were removed from the model when p-values were > 0.10. As each twin was treated as an independent observation, linear mixed models (LMMs) in SPSS

were used to adjust p-values levels for the correlated twin observations (Peugh & Enders, 2005). Because LMMs do not have a straightforward measure of effect size, the incremental predictive power of the onset and course variables was assessed using the partial eta squared (η^2) from the ANOVAs unadjusted for the twin observations. Generalized estimating equations were used for categorical outcomes with odds ratios used for effect sizes. Additionally, dummy coding was used to compare each of the AUD onset and course groups to the control group in separate models.

Results

The tables provide the means and standard deviations for the AUD groups on each dependent variable, as well as the F-statistics and effect sizes for the main effects of an adolescent onset and persistent course from the 2 × 2 ANOVAs.

Substance Use and Mental Health Problems at Age 17

Each measure of substance use and abuse at age 17 was associated with an adolescent onset of AUD, though none were significantly associated with a persistent course (Table 1). Effect sizes for adolescent AUD were largest for number of intoxications, maximum number of drinks, alcohol frequency and quantity, and symptoms of nicotine and illicit drug dependence. Compared to the control group, AUD+ adolescents initiated alcohol use at significantly younger ages, had greater access to substances, and engaged in significantly more polysubstance use.

Adolescent AUD was associated with higher scores on all measures of antisocial behavior and behavioral disinhibition, while a persistent course had a trend-level association with higher Delinquent Behavior Inventory scores ($p = 0.048$). Compared to the control group, women with an adolescent onset had higher levels of early adolescent problem behavior, delinquent behavior, behavioral disinhibition, and symptoms of conduct disorder and adult antisocial behavior. Compared to controls, a young adult onset of AUD was associated with early adolescent problem behavior, while the YAO-P group had higher scores on the behavioral disinhibition scale and the YAO-D group exhibited more symptoms of adult antisocial behavior.

Adolescent AUD also had a trend-level association with major depression ($p = 0.012$). A significant Onset x Course interaction was detected for symptoms of bulimia nervosa, $F(1, 112.0) = 14.0$, $\eta^2 = 0.111$, $p < 0.001$, and binge eating disorder, $F(1, 90.4) = 6.9$, $\eta^2 = 0.071$, $p = 0.009$. Main effects of onset and course were observed for bulimia nervosa ($p = 0.005$ and < 0.001, respectively) with trend-level main effects of onset and course for binge eating disorder ($p = 0.047$ and 0.014, respectively), indicating that elevated eating disorder symptoms were associated with a severe form of AUD characterized by both an adolescent onset and persistent course.

Psychosocial Functioning at Age 17

AUD group comparisons for personality, academic functioning, and intellectual ability are reported in Table 2. Adolescent AUD was associated with lower positive emotionality, and there was a trend-level association ($p = 0.025$) with lower behavioral constraint scores. The

AO-D and AO-P groups had significantly lower behavioral constraint scores than the control group. The AO-D and YAO-P groups also had higher negative emotionality scores than the control group (trend-level for the AO-P group, $p = 0.017$). Adolescent AUD was associated with lower expectations of academic attainment and engagement, more disciplinary problems, and lower GPAs. There was a trend-level Onset x Course interaction for performance IQ, $F(1, 107.8)=5.3$, $\eta^2 = 0.055$, $p = 0.023$. This was a cross-over interaction such that the AO-D and YAO-P groups had lower performance IQ scores than the AO-P and YAO-D groups. The YAO-P group also had lower verbal and full scale IQ scores than the control group.

Environmental Risk and Protective Factors at Age 17

Group comparisons for parental characteristics, stressful life events, and peer affiliation are reported in Table 3. Adolescent AUD was associated with father—but not mother—externalizing disorders, an effect primarily driven by the AO-D group. There was a trend-level association between adolescent AUD and lower mother-child relationship quality ($p = 0.035$). Compared to the control group, the AO-D and AO-P groups had lower mother-child relationship quality. Also, the AO-D, AO-P, and YAO-P groups had lower father-child relationship quality than the control group.

Adolescent AUD was associated with more family-level stressful life events, as well as school, legal, and alcohol or drug problems. Finally, adolescent AUD was associated with fewer prosocial peers and more antisocial peers. Each AUD group reported more antisocial peers than the control group (trend-level for the YAO-P group, $p = 0.036$). Further, the AO-D group also had significantly fewer prosocial peers than the control group (trend-level for the AO-P group, $p = 0.044$).

Adult Outcomes at Age 29

Descriptive statistics and AUD group comparisons for the adult substance use and mental health outcomes at age 29 are reported in Table 4. For period of heaviest use, each AUD group reported significantly greater average quantity and maximum consumption than the control group. Alcohol consumption variables were associated with persistent AUD, but not an adolescent onset. For substance use over the last 6 years, persistent AUD was associated with greater average quantity, maximum consumption, and nicotine dependence symptoms, along with a trend-level elevation in drug dependence symptoms ($p = 0.011$). All AUD groups reported significantly higher levels of nicotine dependence and illicit drug use than the control group. Each AUD group except the YAO-D group also reported greater average number of drinks and maximum consumption than the control group. The AO-P and YAO-P groups reported significantly more drug dependence symptoms. Persistent AUD was associated with significantly more adult antisocial behavior and major depression symptoms over the last 6 years. Compared to the control group, each AUD group reported significantly more adult antisocial behavior, and the persistent AUD groups endorsed significantly more major depression symptoms. A trend-level Onset x Course interaction was detected for mental health problems, $F(1, 112.9) = 4.6$, $\eta^2 = 0.042$, $p = 0.034$, with a trend-level main effect for an adolescent onset ($p = 0.013$). The AO-P group reported significantly more mental health problems than the control group.

For social outcomes in adulthood (Table 5), persistent AUD was associated with more legal and alcohol/drug problems in the last 6 years, and the AO-P and YAO-P groups had more legal and alcohol/drug problems than the control group. The AO-P group also reported more financial problems than the control group. There were trend-level associations between adolescent AUD and lower income, and between persistent AUD and lower occupational status. Persistent AUD had a trend-level association with lower odds for having a child by age 29 ($p = 0.027$). The YAO-P group was less likely to be married by age 29 than the control group. The AO-D group cohabitated and had a child at a younger age than the control group. There were trend-level associations for the AO-P group such that they were slightly more likely to cohabitate ($p = 0.012$) and be separated or divorced ($p = 0.016$), but less likely to be married ($p = 0.030$) compared to the control group.

Persistent AUD was associated with more antisocial peers at age 29, and each AUD group reported more antisocial peers than the control group. Additionally, all groups except the YAO-D group reported fewer prosocial peers. Persistent AUD was associated with a significantly greater number of sex partners over the last 6 years, an effect driven by the YA O -P group. Persistent AUD was also associated with greater drinking by a romantic partner, and t he AO-P a n d YAO-P groups reported more drinking by their romantic partner than the control group (trend-level for the AO-D group, $p = 0.038$). Finally, all AUD groups reported that their romantic partners had significantly more permissive attitudes toward substance use relative to the romantic partners of those in the control group.

Discussion

We examined adolescent risk factors and young adult consequences associated with an adolescent onset and persistent course of AUD in a community sample of women followed from age 17 to 29. Most women (71%) failed to reach our diagnostic threshold (2 or more symptoms of alcohol abuse or dependence) of AUD while a small percentage exhibited an adolescent onset (7.4%) or persistent course (6.4%). Two thirds (65%) of women with AUD desisted by age 29. Any endorsement of AUD was associated with high levels of risk exposure and prolonged consequences. In particular, women with an adolescent onset of AUD experienced a variety of risk factors and substantial psychosocial impairment in adolescence, while those with persistent AUD experienced continued problems in young adulthood. Notably, though measures of behavioral disinhibition in adolescence were consistent predictors of persistent AUD in our prior study of men (e.g., Hicks et al., 2010), they failed to differentiate the persistent and desistent AUD groups in women. Finally, the greater accumulation of risk exposure associated with AUD in women appears to have significant consequences for women's health and functioning, even after ostensible “recovery”.

An adolescent onset of AUD in women was associated with more substance use and abuse/dependence, antisocial behavior, behavioral disinhibition, mental health problems, academic problems, and deviant peer affiliation at age 17. Relative to the control group, however, even women with a young adult onset were elevated on most substance use and some behavioral disinhibition measures. Persistent AUD was associated with higher levels of heavy drinking, nicotine dependence, antisocial behavior, and major depression at age 29.

Persistent AUD was also uniquely related to more antisocial peers, a greater number of sex partners, and heavier drinking by a current romantic partner relative to women who desisted by age 29.

While early and protracted symptoms of AUD were associated with significant health consequences for women, remission of acute symptoms did not fully attenuate the negative outcomes associated with AUD. Of particular concern is that women who desisted from AUD by age 29 did not return to normative levels of alcohol use as defined by levels of alcohol use in the control group. Relative to women never diagnosed with AUD, the AO-D group continued to exhibit elevated alcohol, nicotine, and illicit drug use and antisocial behavior at age 29, while the YAO-D group also had elevated levels of nicotine and illicit drug use and antisocial behavior. Though the relative deviance of women who desisted by age 29 made it difficult to detect differences among the AUD groups, it is apparent that women who endorse any AUD during this developmental period exhibited a relatively long-lasting vulnerability to hazardous alcohol and substance use, even after the remission of AUD symptoms.

The features of AUD in women that we identified in the current study (i.e., lower prevalence and higher rates of desistence, but greater severity relative to non-diagnosed same gender participants) contrast with findings in men, and suggest gender-specific risks and consequences of AUD. In women, AUD is relatively infrequent and associated with significant impairment across multiple functional domains, suggesting it is a severe form of psychopathology requiring high levels of risk exposure to manifest. While women showed similar deficits as previously documented in men with AUD, we extended these deficits to include domains more specific to women, most notably the eating disorders of bulimia and binge eating disorder. Comorbidity across these eating disorders and AUD suggests that they may share a common liability, most likely behavioral disinhibition (Rosval et al., 2006; Vitousek et al., 1994; Westen & Harnden-Fischer, 2001). Along with the heightened severity of AUD for women, the health consequences for AUD are not fully eliminated by symptom desistence. Even women who “recover” from AUD (i.e., no longer exhibit symptoms but may still use alcohol) reported heightened levels of substance use, antisocial behavior, and dysfunction in the social environment relative to the non-diagnostic, control group. Continued vulnerability appeared to be unique to women as desistence in men was more clearly indicative of a traditional “recovery” that entails improvement in multiple domains of psychosocial functioning (Hicks et al., 2010). Alternatively, continued impairment may be due to impact of the accumulation of risk necessary to express AUD rather than to the effects of AUD per se. Of course, both processes could be at work; that is, relative to men, AUD in women has a more enduring negative effect on psychosocial functioning, while other risk factors that women with AUD are likely to experience also have unique and long-last effects on functioning.

Subsequently, the current study provides an important gender-specific context for further study of AUD desistence. As women's impairment was similar across developmental groups, high rates of desistence prior to age 29 were not necessarily due to an accumulation of negative consequences as a function of prolonged alcohol use. Instead, the relative strength of gender-specific promoters of desistence like romantic relationships and young

parenthood may have a potent effect on the remission or maturing out of AUD symptoms in women. Though our power was limited, women with persistent AUD were less likely to be married and have a child, and more likely to be separated or divorced and in a romantic relationship with a partner endorsing high drinking levels. The formation of a stable family unit—in particular, child bearing for women—may then be implicated in the remission of AUD symptoms, though this may not generalize to other domains of functioning. Follow-up studies such as discordant twin analyses and direct gender comparisons are needed to provide a more rigorous test of these hypotheses and delineate potential causal mechanisms for desistence.

Though the current study provides one of the few comprehensive examinations of the course of AUD and its correlates from adolescence through young adulthood in a large, community sample of women, some limitations need to be acknowledged. The MTFS sample was comprised almost solely of European Americans, which limits generalizability to women of other racial and ethnic groups. While the sample was large, the low prevalence of AUD among women limited statistical power, despite requiring only 2 symptoms of abuse or dependence for a diagnosis of AUD. Though technically a sub-threshold diagnosis, requiring only 2 symptoms for an AUD provided greater sensitivity to emerging alcohol problems and improved statistical power, with prior MTFS studies demonstrating the validity of this approach (Elkins et al., 2004; Elkins et al., 2006; Elkins, McGue, & Iacono 2007; McGue & Iacono, 2005). Also, the design was primarily descriptive with few attempts to draw causal inferences from the many associations we detected. Importantly, these methods served our primary aim of examining patterns of associations across multiple domains in an effort to distinguish profiles of developmental risk for AUD rather than validating any single gender-specific correlate. Additionally, while our strategy of focusing on rationally derived groups has the advantage of theoretical clarity, additional insights may be provided by sophisticated statistical methods that identify trajectory groups empirically. Finally, though an initial investigation of the developmental course of AUD in women was critical, direct gender comparisons are needed to provide a rigorous test of whether the risk structure and consequences of AUD differs for men and women. In particular, direct gender comparisons will be needed to test the hypothesis that women with AUD are a more severe group than men with AUD, and so require a greater loading or exposure to risk factors in order to manifest the disorder. Also, given the unique patterns of desistence among women, it will also be important to examine potential gender differences in the mechanisms underlying the maintenance versus desistence of AUD.

Acknowledgments

Author Note: This research was supported in part by National Institute on Alcohol Abuse and Alcoholism R01 Award AA 009367. Katherine T. Foster was supported by National Institute on Alcohol Abuse and Alcoholism T32 Fellowship Award AA 007477. Brian M. Hicks was supported by National Institute of Drug Abuse K01 Award DA 025868.

References

Acker C. Neuropsychological deficits in alcoholics - the relative contributions of gender and drinking history. British Journal of Addiction. 1986; 81(3):395–403. [PubMed: 3461848]

Babor TF, Hofmann M, Del Boca F, Hesselbrock V, Meyer R, Dolinsky Z, Rounsaville B. Types of alcoholics I: Evidence for an empirically-derived typology based on indicators of vulnerability and severity. Archives of General Psychiatry. 1992; 49:599–608. [PubMed: 1637250]

Blazei RW, Iacono WG, Krueger RF. Intergenerational transmission of antisocial behavior: how do kids become antisocial adults? Applied Prevention Psychology. 2006; 11:230–253.

Borges G, Ye Y, Bond J, Cherpitel CJ, Cremonte M, Moskalewicz J, Rubio-Stipec M. The dimensionality of alcohol use disorders and alcohol consumption in a cross-national perspective. Addiction. 2010; 105(2):240–254. [PubMed: 20078482]

Brown S, Tapert S. Adolescence and the trajectory of alcohol use: Basic to clinical studies. Adolescent Brain Development: Vulnerabilities and Opportunities. 2004; 1021:234–244.

Caspi A, Henry B, McGee RO, Moffitt TE, Silva PA. Temperamental origins of child and adolescent behavior problems: from age three to age fifteen. Child Development. 1995; 66:55–68. [PubMed: 7497829]

Caspi A, Moffitt TE, Newman DL, Silva PA. Behavioral observations at age 3 years predict adult psychiatric disorders. Longitudinal evidence from a birth cohort. Archives of General Psychiatry. 1996; 53:1033–39. [PubMed: 8911226]

Chassin L, Flora DB, King KM. Trajectories of alcohol and drug use and dependence from adolescence to adulthood: The effects of familial alcoholism and personality. Journal of Abnormal Psychology. 2004; 113(4):483–498. [PubMed: 15535782]

Chen K, Kandel D. The natural-history of drug-use from adolescence to the mid-thirties in a general-population sample. American Journal of Public Health. 1995; 85(1):41–47. [PubMed: 7832260]

Clark DB. The natural history of adolescent alcohol use disorders. Addiction. 2004; 99(Suppl 2):5–22. [PubMed: 15488102]

Clark DB, Kirisci L, Tarter RE. Adolescent versus adult onset and the development of substance use disorder in males. Drug and Alcohol Dependence. 1998; 49:115–121. [PubMed: 9543648]

Cloninger CR. Neurogenetic adaptive mechanisms in alcoholism. Science. 1987; 236:410–416. [PubMed: 2882604]

Cotto JH, Davis E, Dowling GJ, Elcano JC, Staton AB, Weiss SRB. Gender effects on drug use, abuse, and dependence: A special analysis of results from the national survey on drug use and health. Gender Medicine. 2010; 7(5):402–413. [PubMed: 21056867]

Dawson DA, Goldstein RB, Moss HB, Li T, Grant BF. Gender differences in the relationship of internalizing and externalizing psychopathology to alcohol dependence: Likelihood, expression and course. Drug and Alcohol Dependence. 2010; 112(1-2):9–17. [PubMed: 20558014]

Diehl A, Croissant B, Batra A, Mundle G, Nakovics H, Mann K. Alcoholism in women: Is it different in onset and outcome compared to men? European Archives of Psychiatry and Clinical Neuroscience. 2007; 257(6):344–351. [PubMed: 17629733]

Dishion TJ, Patterson GR, Stoolmiller M, Skinner ML. Family, school, and behavioral antecedents to early adolescent involvement with antisocial peers. Developmental Psychology. 1991; 27:172–180.

Elkins I, King S, McGue M, Iacono W. Personality traits and the development of nicotine, alcohol, and illicit drug disorders: Prospective links from adolescence to young adulthood. Journal of Abnormal Psychology. 2006; 115(1):26–39. [PubMed: 16492093]

Elkins IJ, McGue M, Iacono WG. Prospective effects of attention-deficit/hyperactivity disorder, conduct disorder, and sex on adolescent substance use and abuse. Archives of General Psychiatry. 2007; 64(10):1145–1152. [PubMed: 17909126]

Elkins IJ, McGue M, Iacono WG, Tellegen A. Genetic and environmental influence on parent-son relationships: evidence for increasing genetic influence during adolescence. Developmental Psychology. 1997; 33:351–363. [PubMed: 9147842]

Elkins I, McGue M, Malone S, Iacono W. The effect of parental alcohol and drug disorders on adolescent personality. American Journal of Psychiatry. 2004; 161(4):670–676. [PubMed: 15056513]

Granic I, Patterson GR. Toward a comprehensive model of antisocial development: A dynamic systems approach. Psychological Review. 2006; 113:101–131. [PubMed: 16478303]

Grant S, LaBrie JW, Hummer JF, Lac A. How drunk am I? Misperceiving one's level of intoxication in the college drinking environment. Psychology of Addictive Behaviors. 2012; 26(1):51–58. [PubMed: 21604830]

Hernandez-Avila C, Rounsaville B, Kranzler H. Opioid-, cannabis- and alcohol-dependent women show more rapid progression to substance abuse treatment. Drug and Alcohol Dependence. 2004; 74(3):265–272. [PubMed: 15194204]

Hicks BM, South SC, DiRago AC, Iacono WG, McGue M. Environmental adversity and increasing genetic risk for externalizing disorders. Archives of General Psychiatry. 2009; 66:640–648. [PubMed: 19487629]

Hicks BM, Iacono WG, McGue M. Consequences of an adolescent onset and persistent course of alcohol dependence in men: Adolescent risk factors and adult outcomes. Alcoholism-Clinical and Experimental Research. 2010; 34(5):819–833.

Hommer D, Momenan R, Rawlings R, Ragan P, Williams W, Rio D, Eckardt M. Decreased corpus callosum size among alcoholic women. Archives of Neurology. 1996; 53(4):359–363. [PubMed: 8929159]

Iacono WG, Carlson SR, Taylor J, Elkins IJ, McGue M. Behavioral disinhibition and the development of substance-case disorders: Findings from the minnesota twin family study. Development and Psychopathology. 1999; 11(4):869–900. [PubMed: 10624730]

Iacono WG, Malone SM, McGue M. Behavioral disinhibition and the development of early-onset addiction: common and specific influences. Annual Review of Clinical Psychology. 2008; 4:12.1–12.24.

Iacono WG, McGue M, Krueger RF. Minnesota center for twin and family research. Twin Research and Human Genetics. 2006; 9(6):978–984. [PubMed: 17254440]

Johnson W, McGue M, Iacono WG. Genetic and environmental influences on academic achievement trajectories during adolescence. Developmental Psychology. 2006; 43:514–532. [PubMed: 16756442]

Johnstone BM, Leino EV, Ager CR, Ferrer H, Fillmore KM. Determinants of life-course variation in the frequency of alcohol consumption: Meta-analysis of studies from the collaborative alcohol-related longitudinal project. Journal of Studies on Alcohol. 1996; 57(5):494–506. [PubMed: 8858547]

Kendler KS, Prescott CA, Myers J, Neale MC. The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. Archives of General Psychiatry. 2003; 60:929–37. [PubMed: 12963675]

Keyes KM, Grant BF, Hasin DS. Evidence for a closing gender gap in alcohol use, abuse, and dependence in the united states population. Drug and Alcohol Dependence. 2008; 93(1-2):21–29. [PubMed: 17980512]

Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M. Etiologic connections among substance dependence, antisocial behavior, and personality: modeling the externalizing spectrum. Journal of Abnormal Psychology. 2002; 111:411–24. [PubMed: 12150417]

Krueger RF, Markon RF, Patrick CJ, Benning SD, Kramer MD. Linking antisocial behavior, substance use, and personality: an integrative quantitative model of the adult externalizing spectrum. Journal of Abnormal Psychology. 2007; 116:645–666. [PubMed: 18020714]

Krueger RF, Nichol PE, Hicks BM, Markon KE, Patrick CJ, Iacono WG, McGue M. Using latent trait modeling to conceptualize an alcohol problems continuum. Psychological Assessment. 2004; 16(2):107–19. [PubMed: 15222807]

Mann K, Batra A, Gunther A, Scroth G. Do women develop alcoholic brain-damage more readily than men. Alcoholism-Clinical and Experimental Research. 1992; 16(6):1052–1056.

Masse LC, Tremblay RE. Behavior of boys in kindergarten and the onset of substance use during adolescence. Archives of General Psychiatry. 1997; 54:62–68. [PubMed: 9006402]

McGue M, Iacono W. The association of early adolescent problem behavior with adult psychopathology. American Journal of Psychiatry. 2005; 162(6):1118–1124. [PubMed: 15930060]

Niaura R, Nathan P, Frankenstein W, Shapiro A, Brick J. Gender differences in acute psychomotor, cognitive, and pharmacokinetic response to alcohol. Addictive Behaviors. 1987; 12(4):345–356. [PubMed: 3687517]

Nixon S. Cognitive deficits in alcoholic women. Alcohol Health & Research World. 1994; 18(3):228–232.

Nolen-Hoeksema S. Gender differences in risk factors and consequences for alcohol use and problems. Clinical Psychology Review. 2004; 24(8):981–1010. [PubMed: 15533281]

Nolen-Hoeksema S, Hilt. Possible contributors to the gender differences in alcohol use and problems. The Journal of General Psychology, 2006. 2006; 133(4):357–374.

Olenick NL, Chalmers DK. Gender-specific drinking styles in alcoholics and nonalcoholics. Journal of Studies on Alcohol. 1991; 52(4):325–330. [PubMed: 1875705]

Peugh JL, Enders CK. Using the SPSS mixed procedure to fit crosssectional and longitudinal multilevel methods. Educational and Psychological Measurement. 2005; 65:717–741.

Patterson, GR.; Capaldi, DM. A mediational model for boys' depressed mood. In: Rolf, JE.; Masten, A.; Cicchetti, D.; Nuechterlein, K.; Weintraub, S., editors. Risk and protective factors in the development of psychopathology. Cambridge, UK: University of Cambridge; 1990. p. 141-163.

Piazza NJ, Vrbka JL, Yeager RD. Telescoping of alcoholism in women alcoholics. International Journal of the Addictions. 1989; 24(1):19–28. [PubMed: 2759762]

Randall C, Roberts J, Del Boca F, Carroll K, Connors G, Mattson M. Telescoping of landmark events associated with drinking: A gender comparison RID A-7526-2009. Journal of Studies on Alcohol. 1999; 60(2):252–260. [PubMed: 10091964]

Robins, LM.; Baber, T.; Cottler, LB. Composite International Diagnostic Interview: Expanded Substance Abuse Module. St. Louis: 1987.

Robins LN, Wing J, Wittchen HU, Helzer JE, Babor TF, Burke J, Farmer A, Jablenski A, Pickens R, Regier DA, Sartorius N, Towle LH. The composite international diagnostic interview. Archives of General Psychiatry. 1988; 45:1069–1077. [PubMed: 2848472]

Ross HE. Alcohol and drug-abuse in treated alcoholics - a comparison of men and women. Alcoholism-Clinical and Experimental Research. 1989; 13(6):810–816.

Rosval L, Steiger H, Bruce K, Israel M, Richardson J, Abut M. Impulsivity in women with eating disorders: Problem of response inhibition, planning, or attention? International Journal of Eating Disorders. 2006; 39:590–593. [PubMed: 16826575]

Schuckit M, Anthenelli R, Bucholz K, Hesselbrock V, Tipp J. The time-course of development of alcohol-related problems in men and women. Journal of Studies on Alcohol. 1995; 56(2):218–225. [PubMed: 7760569]

Slutske WS, Heath AC, Madden PA, Bucholz KK, Statham DJ, Martin NG. Personality and the genetic risk for alcohol dependence. Journal of Abnormal Psychology. 2002; 111:124–33. [PubMed: 11871377]

Snyder J, Dishion TJ, Patterson GR. Determinants and consequences of associating with deviant peers during preadolescence and adolescence. Journal of Early Adolescence. 1986; 6:29–43.

Sher K, Gotham H. Pathological alcohol involvement: A developmental disorder of young adulthood. Development and Psychopathology. 1999; 11(4):933–956. [PubMed: 10624733]

Stockwell T, McLeod R, Stevens M, Phillips M, Webb M, Jelinek G. Alcohol consumption, setting, gender and activity as predictors of injury: A population-based case-control study. Journal of Studies on Alcohol. 2002; 63(3):372–379. [PubMed: 12086138]

Tangney JP, Miller RS, Flicker L, Barlow DH. Are shame, guilt, and embarrassment distinct emotions? Journal of Personality and Social Psychology. 1996; 70:1256–1269. [PubMed: 8667166]

Taylor J, McGue M, Iacono WG, Lykken DT. A behavioral genetic analysis of the relationship between the socialization scale and self-reported delinquency. Journal of Personality. 2000; 68:29–50. [PubMed: 10820680]

Tellegen, A.; Waller, NG. Exploring personality through test construction: development of the Multidimensional Personality Questionnaire, in Handbook of Personality Theory and Testing: Vol. II. Personality Measurement and Assessment. Boyle, GJ.; Matthews, G.; Saklofske, DH., editors. Sage; Thousand Oaks, CA: 2008. p. 261-292.

Vitousek K, Manke F. Personality variables and disorders in anorexia nervosa and bulimia nervosa. Journal of Abnormal Psychology. 1994; 103:137–147. [PubMed: 8040475]

Wechsler, D. WAIS-R Manual: Wechsler Adult Intelligence Scale Revised. Psychological Corporation; New York: 1981.

Westen D, Harnden-Fischer J. Personality profiles in eating disorders: rethinking the distinction between axis I and axis II. American Journal of Psychiatry. 2001; 158:547–562. [PubMed: 11282688]

Wilsnack RW, Wilsnack SC, Kristjanson AF, Vogeltanz-Holm ND, Gmel G. Gender and alcohol consumption: Patterns from the multinational GENACIS project. Addiction. 2009; 104(9):1487–1500. [PubMed: 19686518]

Wong MM, Nigg JT, Zucker RA, Puttler LI, Fitzgerald HE, et al. Behavioral control and resiliency in the onset of alcohol and illicit drug use: a prospective study from preschool to adolescence. Child Development. 2006; 77:1016–33. [PubMed: 16942503]

Young SE, Stallings MC, Corley RP, Krauter KS, Hewitt JK. Genetic and environmental influences on behavioral disinhibition. American Journal of Medical Genetics. 2000; 96:684–95. [PubMed: 11054778]

Zucker, RA.; Fitzgerald, HE.; Moses, HD. Emergence of Alcohol Problems and the Several Alcoholisms: A Developmental Perspective on Etiologic Theory and Life Course Trajectory, in Developmental Psychopathology: Vol. 2. Risk, Disorder, and Adaption. Cicchetti, D.; Cohen, DJ., editors. Wiley; New York: 1994. p. 677-711.

Table 1

Substance Use and Abuse, Psychopathology and Behavioral Disinhibition at age 17

	Never (*n* = 449)	YA O -D (*n* = 43)	YA O -P (*n* = 27)	AO-D (*n* = 33)	AO-P (*n* = 14)	Adolescent Onset		Persistent Course	
						F-statistic	Partial η2	*F*-statistic	Partial η2
Substance use and abuse									
Access to substances (t-score)	48.5 (9.0)	51.2 (9.6)	52.1 (9.9)	58.1 (10.6)**	56.5 (6.8)**	*F*(1,98.5)=8.3	0.093*	*F*(1,95.5)=0.8	0.000
Age of 1st drink	17.1 (2.1)	15.7 (1.5)**	15.8 (1.8)*	14.7 (1.3)**	13.9 (2.2)**	*F*(1,103.1)=17.1	0.138**	*F*(1,113.4)=0.5	0.005
Frequency of alcohol use past year	1.5 (1.5)	2.5 (1.5)**	2.1 (1.3) ‡	3.8 (1.3)**	3.7 (1.3)**	*F*(1,102.3)=26.7	0.197**	*F*(1,113.7)=0.4	0.009
Average alcohol quantity past year	0.9 (1.84)	2.2 (2.5)**	1.6 (2.1) ‡	4.5 (3.4)**	5.3 (3.9)**	*F*(1,113.0)=25.6	0.185**	*F*(1,113.0)=0.0	0.000
# Alcohol Intoxications	2.0 (4.6)	6.5 (12.0)**	3.0 (5.0)	33.4 (35.1)**	29.4 (38.0)**	*F*(1,107.3)=30.2	0.236**	*F*(1,111.9)=0.7	0.006
Maximum # drinks 24 hours	3.4 (4.6)	7.1 (5.9)**	6.0 (4.1)*	15.9 (6.5)**	13 (8.6)**	*F*(1,113.0)=50	0.307**	*F*(1,113.0)=2.2	0.019
Nicotine use (# days per month)	4.1 (9.2)	9.1 (12.1)**	10.5 (13.2)**	21.6 (13.2)**	16.5 (14.4)**	*F*(1,106.1)=15.8	0.132**	*F*(1,111.8)=0.2	0.005
# Cigarettes smoked per day	1.2 (4.0)	2.5 (4.4)*	2.6 (4.1) ‡	7.8 (7.1)**	7.4 (8.0)**	*F*(1,95.4)=21.4	0.160**	*F*(1,114.0)=1.1	0.010
Nicotine dependence symptoms	0.4 (1.2)	1.1 (1.9)**	0.7 (1.3)	3.0 (2.4)**	3.2 (3.1)**	*F*(1,107.7)=24.6	0.201**	*F*(1,111.5)=0.2	0.001
# Marijuana uses	1.7 (10.2)	9.1 (23.2)**	1.6 (3.2)	23.8 (34.4)**	22.6 (34.2)**	*F*(1,112.0)=11.6	0.094*	*F*(1,112.0)=1.0	0.009
# Different drug classes tried	0.2 (0.6)	0.6 (0.9)**	0.5 (0.7) ‡	1.5 (1.4)**	1.1 (1.4)**	*F*(1,112.0)=13	0.104**	*F*(1,112.0)=0.4	0.003
# Drug dependence symptoms	0.0 (0.3)	0.2 (0.6)*	0.1 (0.4)	1.5 (2.0)**	2.1 (3.2)**	*F*(1,107.6)=22.6	0.182**	*F*(1,113.9)=0.2	0.003
Axis I Disorders									
# Major Depression symptoms	1.2 (2.4)	1.1 (2.4)	1.4 (2.8)	2.0 (2.7)	3.3 (3.8)*	*F*(1,107.7)=6.6	0.047 ‡	*F*(1,110.0)=1.4	0.013
# Anorexia Nervosa symptoms	0.4 (0.9)	0.6 (1.0)	0.2 (0.8)	0.7 (1.1)	0.9 (1.1) ‡	*F*(1,104.5)=3.1	0.026	*F*(1,112.9)=0.7	0.006
# Bulimia Nervosa symptoms	0.2 (0.7)	0.2 (0.7)	0.2 (0.6)	0.0 (0.3)	1.3 (1.8)**	*F*(1,112.0)=8.0	0.067*	*F*(1,112.0)=12.9	0.103**
# Binge Eating Disorder symptoms	0.4 (1.5)	0.4 (1.5)	0.3 (1.4)	0.1 (0.8)	2.0 (3.2)**	*F*(1,111.8)=4.1	0.039 ‡	*F*(1,81.5)=6.3	0.062 ‡
Antisocial Behavior/Disinhibition									
Early adolescent problem behavior	0.5 (0.9)	1.2 (1.3)**	1.1 (1.3)*	2.1 (1.5)**	2.2 (1.5)**	*F*(1,94.3)=13.6	0.109**	*F*(1,113.9)=0.0	0.000
Delinquent behavior inventory	2.7 (2.7)	3.4 (2.5)	3.6 (3.4)	6.9 (3.6)**	10.1 (7.0)**	*F*(1,103.4)=32.5	0.255**	*F*(1,99.3)=4.0	0.024 ‡
Behavioral disinhibition scale	1.9 (1.8)	2.6 (1.8) ‡	3.2 (2.3)*	5.2 (2.2)**	6.1 (3.4)**	*F*(1,97.9)=35.7	0.268**	*F*(1,96.0)=2.2	0.021
# Conduct disorder symptoms	0.4 (0.8)	0.5 (0.8)	0.7 (0.7)	1.5 (1.4)**	2.3 (2.6)**	*F*(1,96.4)=22.2	0.165**	*F*(1,113.4)=3.1	0.027
# Adult antisocial behavior symptoms	0.3 (0.6)	0.7 (1.1)**	0.5 (0.7)	2.0 (1.6)**	2.4 (2.4)**	*F*(1,108.6)=31.9	0.229**	*F*(1,111.7)=0.1	0.000

Psychol Addict Behav. Author manuscript; available in PMC 2015 June 01.

‡ $p < .05$;

* $p < .01$;

** $p < .001$.

YAO-D = Young Adult Onset- Desist; YAO-P = Young Adult Onset-Persist; AO-D = Adolescent Onset-Desist; AO-P = Adolescent Onset-Persist. AUD = Alcohol use disorder. Adolescent onset = AUD onset by age 17; Young adult onset = AUD onset by age 20 or 24; Desist = 0 AUD symptoms at age 29; Persist = AUD at age 29. Values in the columns for the AUD groups are the means and standard deviations for the respective group and measure. Symbols indicating significance levels on these values indicate whether the mean for the given AUD group is significantly different from the mean of the never group (i.e., individuals who did not meet criteria for AUD between age 17 to 29). The partial eta squared (η2) in the Adolescent Onset and Persistent Course columns is a measure of predictive power of the given after controlling for the other. The non-integer and varying values for the degrees of freedom for the *F*-tests are due to adjustments for the correlated twin observations and different levels of missing data for certain variables.

Psychol Addict Behav. Author manuscript; available in PMC 2015 June 01.

Table 2

Personality, Academic Functioning, and Intellectual Ability at age 17

	Never (*n* = 449)	YA O -D (*n* = 43)	YA O -P (*n* = 27)	AO-D (*n* = 33)	AO-P (*n* = 14)	Adolescent Onset		Persistent Course	
						F-statistic	Partial η2	*F*-statistic	Partial η2
Personality (*t-scores*)									
Positive Emotionality	50.2 (9.7)	53.0 (11.5)	50.0 (8.9)	46.6 (11.7)	45.4 (8.7)	*F*(1,106.0)=7.2	0.063*	*F*(1,106.0)=1.4	0.013
Negative Emotionality	48.9 (9.3)	51.3 (11.6)	54.5 (9.8)*	55.8 (10.8)**	55.2 (11.3)‡	*F*(1,102.7)=2.3	0.020	*F*(1,102.2)=0.3	0.006
Behavioral Constraint	51.0 (9.4)	48.5 (10.0)	48.2 (8.1)	43.9 (11.2)**	43.0 (12.6)*	*F*(1,88.0)=5.2	0.050 ‡	*F*(1,105.6)=0.1	0.001
Academics									
GPA	3.2 (0.6)	3.1 (0.6)	2.9 (0.7) ‡	2.7 (0.8)**	2.8 (0.8)*	*F*(1,95.9)=3.7	0.038	*F*(1,108.8)=0.3	0.002
Expected attainment	4.9 (0.8)	4.9 (0.6)	4.7 (0.8)	4.2 (1.4)**	4.2 (1.1)*	*F*(1,104.3)=12.7	0.097*	*F*(1,111.8)=0.5	0.005
Engagement (*t-score*)	52.0 (8.8)	49.7 (9.2)	47.0 (7.3)*	41.4 (12.3)**	41.7 (12.9)**	*F*(1,108.5)=14.9	0.110**	*F*(1,102.4)=0.5	0.005
Disciplinary problems (*t-score*)	48.3 (9.2)	51.7 (8.7) ‡	54.7 (10.8)**	57.5 (11.1)**	59.3 (11.4)**	*F*(1,102.0)=7.1	0.064*	*F*(1,111.2)=2.0	0.015
Intellectual Ability									
Verbal IQ	95.4 (13.8)	94.5 (11.9)	87.7 (8.9)*	91.5 (13.2)	88.5 (10.2)	*F*(1,102.5)=0.2	0.005	*F*(1,59.7)=3.2	0.048
Performance IQ	103.6 (16.8)	106.2 (16.6)	96.8 (16.1) ‡	96.8 (15.4) ‡	103.5 (11.4)	*F*(1,110.3)=0.4	0.002	*F*(1,102.6)=0.1	0.002
Full scale IQ	98.6 (13.7)	99.2 (12.8)	91.0 (11.3)*	93 (12.7) ‡	94.1 (8.7)	*F*(1,109.9)=2.1	0.016	*F*(1,70.2)=2.0	0.034

‡ $p < .05$;

* $p < .01$;

** $p < .001$.

YAO-D = Young Adult Onset- Desist; YAO-P = Young Adult Onset-Persist; AO-D = Adolescent Onset-Desist; AO-P = Adolescent Onset-Persist. AUD = Alcohol use disorder. Adolescent onset = AUD onset by age 17; Young adult onset = AUD onset by age 20 or 24; Desist = 0 AUD symptoms at age 29; Persist = AUD at age 29. Values in the columns for the AUD groups are the means and standard deviations for the respective group and measure. Symbols indicating significance levels on these values indicate whether the mean for the given AUD group is significantly different from the mean of the never group (i.e., individuals who did not meet criteria for AUD between age 17 to 29). The partial eta squared (η2) in the Adolescent Onset and Persistent Course columns is a measure of predictive power of the given after controlling for the other. The non-integer and varying values for the degrees of freedom for the *F*-tests are due to adjustments for the correlated twin observations and different levels of missing data for certain variables.

Table 3

Environmental Characteristics at age 17

	Never (*n* = 449)	YA O -D (*n* = 43)	YA O -P (*n* = 27)	AO-D (*n* = 33)	AO-P (*n* = 14)	Adolescent Onset		Persistent Course	
						F-statistic	Partial η2	*F*-statistic	Partial η2
Parent Characteristics (*t-scores*)									
Mother Socioeconomic Status	50.2 (9.9)	52.2 (7.9)	49.9 (9.6)	48.9 (12.1)	48.0 (13.0)	*F*(1,114.0)=2.1	0.018	*F*(1,114.0)=0.8	0.007
Father Socioeconomic Status	50.4 (9.9)	52.2 (9.6)	49.6 (10.4)	48.6 (11.3)	46.1 (12.9)	*F*(1,114.0)=3.2	0.027	*F*(1,114.0)=1.6	0.014
Mother Externalizing	49.5 (9.8)	51.2 (11.1)	52.0 (8.6)	52.1 (8.9)	50.0 (15.7)	*F*(1,114.0)=0.0	0.000	*F*(1,114.0)=0.0	0.001
Father Externalizing	49.5 (9.7)	47.4 (8.1)	50.7 (10.8)	56.9 (12.0)**	52.1 (12.1)	*F*(1,90.0)=9.8	0.098*	*F*(1.90.0)=0.0	0.000
Mother-child relationship quality	51.0 (9.7)	48.4 (10.2)	49.6 (6.8)	44.8 (10.8)*	43.9 (9.7)*	*F*(1,100.3)=4.6	0.048 ‡	*F*(1,56.7)=0.5	0.000
Father-child relationship quality	51.2 (9.5)	49.7 (8.9)	43.3 (12.9)**	46.0 (10.5)*	42.0 (11.6)**	*F*(1,37.3)=2.7	0.017	*F*(1,23.3)=1.1	0.060
Stressful life events									
# Family-level stressful life events	2.5 (2.7)	2.7 (2.5)	2.9 (3.1)	4.6 (3.2)**	4.5 (3.2)*	*F*(1,107.1)=18.5	0.081**	*F*(1,65.8)=0.1	0.000
# School problems	1.1 (1.1)	1.5 (1.2)	1.5 (1.3)	2.4 (1.4)**	2.1 (1.4)*	*F*(1,110.2)=7.1	0.081*	*F*(1,109.5)=0.3	0.001
# Alcohol/drug and legal problems	0.3 (0.6)	0.7 (0.9)*	0.5 (0.9)	1.5 (1.3)**	1.8 (1.3)**	*F*(1,105.4)=21.4	0.166**	*F*(1,114.0)=0	0.000
Peers (*t-scores*)									
Prosocial peers	50.8 (8.9)	52.5 (9.7)	50.3 (10.3)	44.0 (10.1)**	45.7 (8.4) ‡	*F*(1,94.8)=11.9	0.120*	*F*(1,93.0)=0.5	0.001
Antisocial peers	48.0 (8.8)	53.4 (9.3)**	52.5 (10.8) ‡	61.4 (11.4)**	58.8 (8.2)**	*F*(1,64.9)=9.3	0.119*	*F*(1,47.9)=0.8	0.006

‡ $p < .05$;

* $p < .01$;

** $p < .001$.

YAO-D = Young Adult Onset- Desist; YAO-P = Young Adult Onset-Persist; AO-D = Adolescent Onset-Desist; AO-P = Adolescent Onset-Persist. AUD = Alcohol use disorder. Adolescent onset = AUD onset by age 17; Young adult onset = AUD onset by age 20 or 24; Desist = 0 AUD symptoms at age 29; Persist = AUD at age 29. Values in the columns for the AUD groups are the means and standard deviations for the respective group and measure. Symbols indicating significance levels on these values indicate whether the mean for the given AUD group is significantly different from the mean of the never group (i.e., individuals who did not meet criteria for AUD between age 17 to 29). The partial eta squared (η2) in the Adolescent Onset and Persistent Course columns is a measure of predictive power of the given after controlling for the other. The non-integer and varying values for the degrees of freedom for the *F*-tests are due to adjustments for the correlated twin observations and different levels of missing data for certain variables.

Table 4

Adult Substance Use and Mental Health Outcomes at age 29

	Never (*n* = 449)	YA O -D (*n* = 43)	YA O -P (*n* = 27)	AO-D (*n* = 33)	AO-P (*n* = 14)	Adolescen Onset		Persistent Course	
						F-statistic	Partial η2	*F*-statistic	Partial η2
Period of heaviest use									
Average alcohol quantity	3.5 (1.9)	5.7 (2.3)**	7.6 (3.5)**	6.1 (3.3)**	7.6 (2.4)**	*F*(1,114.0)=0.2	0.002	*F*(1,114.0)=10	0.081*
Max # drinks in 24 hours	7.5 (4.7)	12.2 (5.3)**	16.0 (7.3)**	12.3 (5.9)**	19.6 (7.3)**	*F*(1,104.6)=0.9	0.009	*F*(1,113.4)=18.3	0.132**
Substance Use (Last 6 years)									
Average alcohol quantity	2.2 (1.4)	2.6 (1.2)	5.3 (2.6)**	3.5 (2.5)**	6.1 (2.5)**	*F*(1,109.8)=2.2	0.038	*F*(1,112.0)=38	0.259**
Max # drinks in 24 hours	5.2 (3.6)	6.4 (3.6)	14.3 (6.2)**	7.9 (5.0)**	16.7 (7.8)**	*F*(1,111.4)=2.3	0.027	*F*(1,108.6)=80.1	0.366**
# Nicotine dependence symptoms	0.5 (1.2)	1.3 (1.8)**	2.9 (2.2)**	2.0 (2.0)**	2.9 (2.1)**	*F*(1,112.4)=3.1	0.015	*F*(1,101.2)=12.6	0.093*
# Marijuana uses	2.8 (14.6)	7.7 (23.2) ‡	20.2 (39.0)**	9.7 (25.5)*	16.6 (36.2)*	*F*(1,100.6)=0.0	0.000	*F*(1,114.0)=3.4	0.028
# Different drug classes used	0.5 (0.8)	1.3 (1.3)**	1.4 (1.4)**	1.5 (1.5)**	2.0 (1.6)**	*F*(1,109.7)=0.5	0.013	*F*(1,110.1)=1.0	0.009
Drug dependence symptoms	0.1 (0.4)	0.1 (0.5)	0.9 (1.8)**	0.3 (1.6)	0.8 (1.7)**	*F*(1,93.0)=0.1	0.001	*F*(1,108.9)=6.8	0.054 ‡
Mental Health/Stressful Life Events (Last 6 years)									
# Adult antisocial behavior symptoms	0.2 (0.6)	0.7 (0.8)*	1.3 (1.0)**	0.7 (0.9)*	1.3 (0.6)**	*F*(1,108.3)=0.1	0.000	*F*(1,110.8)=11.9	0.088*
# Major depression symptoms	1.2 (2.4)	1.3 (2.5)	3.1 (3.6)**	1.7 (2.9)	4.0 (3.9)**	*F*(1,106.7)=0.9	0.008	*F*(1,113.8)=10.2	0.087*
# Mental health problems	0.2 (0.5)	0.4 (0.8)	0.3 (0.6)	0.5 (0.6) ‡	1.1 (1.3)**	*F*(1,105.7)=6.3	0.062 ‡	*F*(1,112.7)=3.1	0.034

‡ $p < .05$;

* $p < .01$;

** $p < .001$.

YAO-D = Young Adult Onset- Desist; YAO-P = Young Adult Onset-Persist; AO-D = Adolescent Onset-Desist; AO-P = Adolescent Onset-Persist. AUD = Alcohol use disorder. Adolescent onset = AUD onset by age 17; Young adult onset = AUD onset by age 20 or 24; Desist = 0 AUD symptoms at age 29; Persist = AUD at age 29. Values in the columns for the AUD groups are the means and standard deviations for the respective group and measure. Symbols indicating significance levels on these values indicate whether the mean for the given AUD group is significantly different from the mean of the never group (i.e., individuals who did not meet criteria for AUD between age 17 to 29). The partial eta squared (η2) in the Adolescent Onset and Persistent Course columns is a measure of predictive power of the given after controlling for the other. The non-integer and varying values for the degrees of freedom for the *F*-tests are due to adjustments for the correlated twin observations and different levels of missing data for certain variables.

Table 5

Social Outcomes at age 29

	Never (*n* = 449)	YA O -D (*n* = 43)	YA O -P (*n* = 27)	AO-D (*n* = 33)	AO-P (*n* = 14)	Adolescen Onset		Persistent Course	
						F-statistic	Partial η2	*F*-statistic	Partial η2
Education, Work, Finances									
Educational attainment (years)	13.5 (2.9)	13.9 (3.3)	12.6 (1.9)	13.3 (2.6)	13.1 (1.7)	*F*(1,92.1)=0.3	0.003	*F*(1,108.6)=2.6	0.021
Occupational Status	3.8 (2.0)	3.3 (1.8)	4.7 (2.1) ‡	4.5 (2.2) ‡	4.6 (1.9)	*F*(1,100)=4.1	0.027	*F*(1,114)=5.1	0.068 ‡
Income	33.5K (23.6K)	40.1K (36.8K)	28.5K (19.6K)	24.5K (17.9K) ‡	27.6K (14.6K)	*F*(1,111.7)=3.2	0.038 ‡	*F*(1,73.8)=1.7	0.012
# Financial problems	0.5 (0.8)	0.6 (0.8)	0.6 (0.7)	0.6 (0.9)	1.0 (0.8)*	*F*(1,109)=1.4	0.013	*F*(1,109)=1	0.010
# Legal and alcohol/drug problems	0.1 (0.4)	0.1 (0.4)	0.4 (0.8)**	0.2 (0.6)	0.9 (1.1)**	*F*(1,74.4)=1.5	0.013	*F*(1,113.9)=12.4	0.097*
Social Environment									
Prosocial peers (*t-score*)	51.0 (9.0)	49.8 (10.6)	45.4 (10.4)*	44.4 (16.9)**	43.36 (8.25)*	*F*(1,98.4)=2.7	0.028	*F*(1,106.6)=2.6	0.015
Antisocial peers (*t-score*)	48.1 (9.1)	52.9 (9.3)*	58.4 (8.8)**	54.1 (11.0)**	66.4 (8.1)**	*F*(1,93.2)=3.2	0.030	*F*(1,109.9)=17.5	0.135**
# sex partners	1.4 (1.3)	1.7 (1.4)	2.9 (2.3)**	1.6 (1.5)	1.9 (0.8)	*F*(1,102.5)=2.8	0.014	*F*(1,103.4)=12.8	0.068*
Partner's drinking (*t-score*)	48.8 (9.4)	50.7 (7.4)	58.0 (11.4)**	53.1 (10.2) ‡	60.4 (8.7)**	*F*(1,77.2)=1.1	0.016	*F*(1,89.7)=12.0	0.125*
Partner's substance use attitudes (*t-score*)	48.5 (9.5)	54.8 (9.4)**	55.7 (7.6)*	54.4 (9.7)*	60.4 (8.3)**	*F*(1,80.7)=0.3	0.005	*F*(1,89)=1.9	0.022
Marriage and Children									
Age 1st cohabitate	22.1 (3.1)	22.1 (2.7)	21.9 (3.1)	20.3 (3.1)*	21.8 (4.5)	*F*(1,99.9)=3.6	0.035	*F*(1,94.2)=0.4	0.004
Age 1st married	23.9 (2.8)	24.8 (2.4)	24.8 (2.4)	24.1 (3.0)	26.2 (2.3) ‡	*F*(1,62)=0.1	0.001	*F*(1,62)=1.3	0.020
Age 1st had a child	24.6 (3.6)	24.6 (3.0)	21.8 (4.5) ‡	1.2 (4.9)**	23.2 (4.1)	*F*(1,54)=3.8	0.066	*F*(1,53.6)=0.9	0.010
						Wald Chi-square	Odds Ratio	Wald Chi-square	Odds Ratio
Ever Cohabitated	68.8%	86.0% ‡	77.8%	78.8%	100.0% ‡	$\chi^2(1) = 0.4$	1.35	$\chi^2(1) = 0.0$	1.14
Cohabited by age 23	44.5%	48.8%	48.2%	63.6%	57.1%	$\chi^2(1) = 1.9$	1.71	$\chi^2(1) = 0.2$	0.85
Ever married	71.0%	60.5%	44.4% *	66.7%	42.9% ‡	$\chi^2(1) = 0.1$	1.12	$\chi^2(1) = 1.0$	0.65
Ever separated or divorced	6.6%	8.1%	13.0%	9.8%	25.0% ‡	χ^2 (1) = 0.8	1.66	χ^2 (1) = 2.7	2.61
Ever had a child	51.6%	55.8%	33.3% ‡	54.5%	42.9%	χ^2 (1) = 0.0	1.01	χ^2 (1) = 4.9	0.43 ‡
Had a child by age 23	18.3%	18.6%	22.2%	36.4% ‡	14.3%	χ^2 (1) = 1.0	1.57	χ^2 (1) = 0.5	0.72

Psychol Addict Behav. Author manuscript; available in PMC 2015 June 01.

‡ $p < .05$;

* $p < .01$;

** $p < .001$.

YAO-D = Young Adult Onset- Desist; YAO-P = Young Adult Onset-Persist; AO-D = Adolescent Onset-Desist; AO-P = Adolescent Onset-Persist. AUD = Alcohol use disorder. Adolescent onset = AUD onset by age 17; Young adult onset = AUD onset by age 20 or 24; Desist = 0 AUD symptoms at age 29; Persist = AUD at age 29. Values in the columns for the AUD groups are the means and standard deviations for the respective group and measure. Symbols indicating significance levels on these values indicate whether the mean for the given AUD group is significantly different from the mean of the never group (i.e., individuals who did not meet criteria for AUD between age 17 to 29). The partial eta squared (η2) in the Adolescent Onset and Persistent Course columns is a measure of predictive power of the given after controlling for the other. The non-integer and varying values for the degrees of freedom for the *F*-tests are due to adjustments for the correlated twin observations and different levels of missing data for certain variables.