

# Sensitive Periods for Adolescent Alcohol Use Initiation: Predicting the Lifetime Occurrence and Chronicity of Alcohol Problems in Adulthood\*

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**ABSTRACT. Objective:** This study examined the association between age at alcohol use onset and adult alcohol misuse and dependence by testing the sensitive-period hypothesis that early adolescence (11–14) is a vulnerable period of development during which initiating alcohol use is particularly harmful. **Method:** Data came from a longitudinal panel of 808 participants recruited in 1981. Participants were followed through age 33 in 2008 with 92% retention. **Results:** Onset of alcohol use before age 11 (late childhood), when compared with initiation during early adolescence, was related to an increased chronicity of adult alcohol dependence, even after accounting for sociodemographic controls and other substance use in adolescence. The present study finds no evidence that early adolescence is a particularly sensitive period for the onset of

alcohol use. Findings related to the onset of regular alcohol use and the chronicity of alcohol dependence suggest that the onset of regular drinking before age 21 is problematic, but no one adolescent period is more sensitive than others. Specifically, although all age groups that started drinking regularly before age 21 had a greater rate of alcohol dependence in adulthood, initiation of regular use of alcohol at or before age 14 was not related to greater chronicity of alcohol dependence than the initiation of regular use of alcohol in middle or late adolescence. **Conclusions:** The findings suggest the importance of delaying the onset of alcohol use through prevention efforts as early as the elementary grades. In addition, prevention efforts should focus on preventing the onset of regular drinking before age 21. (*J. Stud. Alcohol Drugs*, 72, 221–231, 2011)

NATIONAL DATA INDICATE that almost one in four U.S. high school students began drinking alcohol before they entered high school (Centers for Disease Control and Prevention, 2008), yet adolescent drinking has the potential to profoundly affect future life trajectories and outcomes. Research has linked the age at onset of drinking with later alcohol problems. Some studies have suggested a linear relationship between age at onset and the probability of later problems, such that progressively earlier initiation carries increasing risk for later alcohol misuse (Hawkins et al., 1999), alcohol abuse, and other adolescent alcohol-related problem behaviors (Gruber et al., 1996). Similarly, Grant et al. (2001) found that the odds of adult alcohol abuse and alcohol dependence at a 12-year follow-up were significantly increased with each earlier year of onset of regular drinking, defined here as having two or more drinks a week.

## *Sensitive-period hypothesis*

In contrast to the linear “earlier onset is worse” hypothesis, some evidence suggests that there may be sensitive or vulnerable periods during early adolescence when the effects of alcohol onset are particularly harmful. Recent advances in the field of developmental neuroscience have augmented our understanding of ethanol exposure on development (for a review, see Witt, 2010). Studies indicate that physiological changes in early adolescence are not limited to biological changes that mark the onset of puberty but also include brain growth and neural remodeling marked by synaptic cortical remodeling and changes in neurotransmitter receptors and transporters (e.g., Crews et al., 2007; Giedd, 2004; Nixon et al., 2010). During the period of brain remodeling, young people may be more vulnerable to harmful environmental shocks, including exposure to ethanol, that could lead to long-term problems, psychopathology, and priming of neurochemical pathways involved in drug metabolism (De Bellis et al., 2005; Ehlers and Criado, 2010; Volkow and Li, 2005; Witt, 2010).

Furthermore, the transition from childhood to early adolescence is often socially difficult (Bronfenbrenner, 1979; Harter et al., 1992; Rudolph et al., 2001), which may contribute to a heightened vulnerability to alcohol initiation. Social changes that accompany development in early adolescence, such as the formation of self-concept and self-esteem

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(e.g., Guthrie et al., 1994; Harter, 2006), the development of an increased sense of belongingness to one's peer group (Steinberg and Morris, 2001), and the transition to middle school (Eccles et al., 1993) may increase vulnerability to negative outcomes associated with alcohol use onset. Vulnerability to environmental shocks may be particularly salient in the period of early adolescence (age 11-14), when the onset of puberty gives rise to changes in arousal and motivation mechanisms before the regulatory mechanisms are well established (Steinberg, 2005).

Although no study has explicitly examined early adolescence as a sensitive period for adolescent alcohol initiation, some evidence suggests the plausibility of this hypothesis. For example, studies using retrospective reports have found that the onset of alcohol use before age 14 (Grant and Dawson, 1997; Hingson et al., 2006) is related to elevated rates of lifetime alcohol use and dependence problems. Using cross-sectional data and retrospective reports, DeWit et al. (2000) found that the onset of drinking between the ages 11 and 14 resulted in the highest risk profile for later alcohol problems. However, they also found that about 20 years after exposure, those who initiated drinking before age 11 had a similar cumulative probability of alcohol dependence to the age 11-14 initiation group. Along the same lines, Dawson et al. (2008) reported a robust relationship between age at first drink, particularly if before age 15, and the risk of adult alcohol use disorders.

#### *Present study*

Although some research suggests that there may be sensitive periods during which alcohol initiation is particularly damaging, existing studies do not allow a definitive conclusion regarding the sensitive-period hypothesis. Some studies have used cross-sectional data or retrospective reports, which suffer from recall bias, particularly for those who initiated substance use at younger ages (Parra et al., 2003). Other studies operationalized the age at first alcohol use only as the age at which young people took their first drink and did not distinguish between the first drink of alcohol and the initiation of regular use. Yet, initiation of regular use could signal a more risky behavior leading to more adverse outcomes. Finally, most prior studies categorized early initiation as onset of drinking before age 14 or 15. However, this does not allow one to examine whether very early initiation (i.e., in late childhood) has similar or worse effects than initiation in early adolescence. The analyses presented here test the sensitive-period hypothesis using prospectively assessed data on the timing of alcohol initiation. We consider the possible effects of both initiation of any drinking and initiation of regular drinking. Specifically, we test whether initiation in early adolescence is more strongly associated with adult alcohol problems than initiation in pre-, middle, or late adolescence. We focus on predicting problems of alcohol misuse

and dependence and their chronicity ("misuse" is used in place of "abuse" throughout the article because the exact criteria for abuse according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition [DSM-IV; American Psychiatric Association, 1994], were not used here).

An alternative explanation for the association between early onset of alcohol use and increased incidence of later alcohol problems could be the result of individual, family, and social factors that have been linked to both age at initiation and adult alcohol problems. From this perspective, early alcohol use may simply be a marker or a symptom of other risk factors that are salient predictors of adult alcohol problems. Therefore, we consider demographic and risk variables commonly linked to alcohol problems, including ethnicity, childhood poverty, and gender (e.g., Hasin et al., 2007; Johnston et al., 2008; Stinson et al., 1998; Treno et al., 2000). We also control for adolescent tobacco, marijuana, and other illicit drug use, which has been linked to both adolescent alcohol initiation and adult alcohol misuse and dependence problems (Merline et al., 2008).

## **Method**

### *Sample*

Data were drawn from the Seattle Social Development Project, a 25-year longitudinal study that has followed 808 youths from elementary school to adulthood with the goal of understanding prosocial and antisocial development across the life span. In the fall of 1985, all 1,053 fifth-grade students in 18 Seattle public elementary schools serving high-crime areas were invited to participate in the study. Consenting participants were assessed in the fall of 1985 and spring of 1986. After that, they were surveyed annually through 10th grade, again in the 12th grade, and then every 3 years through age 33.

Eight hundred and eight (77%) of the eligible students received parental permission to participate in the longitudinal study and completed the fall 1985 survey. The retention rate was relatively high; 92% ( $n = 721$ ) of those still living ( $n = 785$ ) completed the age 33 assessment in 2008. The Human Subjects Review Committee at the University of Washington approved the Seattle Social Development Project procedures and measures.

This study focuses on sensitive periods for adolescent alcohol initiation; thus, we use data from every participant who initiated alcohol drinking before the legal age of 21 and who participated in at least one wave of assessment from ages 21 to 33. About 87.4% of participants ( $n = 706$ ) initiated drinking before the age of 21. These numbers are comparable to statistics from national surveys (e.g., Substance Abuse and Mental Health Services Administration, 2009). Of the 706, 14 did not have adult alcohol data available and

were excluded, resulting in a final analytic sample size of 692 participants (85.6% of the original sample, 88.2% of those still living). Of these 692 participants, 357 (51.6%) were male, 345 (49.9%) were White, 177 (25.6%) were African American, 130 (18.8%) were Asian American, and 40 (5.8%) were Native American. About half of the sample members ( $n = 347$ ; 50.1%) came from low-income families (as indicated by their participation in the free school lunch program in Grades 5-7). The analyses examining associations between the age at initiation of regular drinking and adult alcohol use problems include those participants who initiated at least some drinking before age 21 and regular drinking (as defined by drinking three or more times a month) by the last wave of assessment at age 33 ( $n = 549$ ). Regarding the excluded cases, 102 participants did not initiate any drinking before age 21 (of these, 64 did not initiate regular drinking before the last assessment, but 38 started regular drinking after age 21), and 143 participants had initiated some alcohol use before age 21, but they did not initiate regular drinking before the last assessment at the age of 33. The 38 participants who did not initiate any drinking before age 21 but initiated regular drinking at some point after age 21 were excluded to keep the data analytic sample consistent with models investigating initiation of any drinking. Sensitivity analyses included these cases, and, as expected, the pattern of results remained similar to those reported in the results section. We also conducted supplemental sensitivity checks where the 143 participants who did not initiate regular drinking before age 33 were included in the analyses as a separate dummy-coded group, and the results were as expected: The group of noninitiators of regular drinking had much lower odds of lifetime alcohol misuse and dependence diagnosis than did any other initiator group.

### Measures

**Outcomes.** The primary outcomes in this study were alcohol misuse and alcohol dependence. Participants reported on their alcohol use behavior using items from the short form of the Diagnostic Interview Schedule (Robins et al., 1981), which concur with DSM-IV diagnostic criteria. The alcohol-dependence items assessed whether respondents experienced clinically significant impairment in the form of tolerance, withdrawal, difficulty controlling alcohol use, continued use despite adverse consequences, failure to fulfill major role obligations as a result of drinking, spending significant effort on alcohol use, and repeated attempts to reduce alcohol use. A dependence diagnosis was given when three or more of these criteria were met. Alcohol misuse was assessed using items that indicated whether respondents experienced clinically significant impairment in the form of persistent drinking despite adverse consequences, failure to fulfill major role obligations as a result of drinking, drinking in physically hazardous situations, and recurrent alcohol-related legal

problems. Alcohol misuse in a given wave was identified when respondents indicated impairment in at least one of the aforementioned criteria. The items for alcohol misuse map onto alcohol abuse criteria from DSM-IV. However, in the DSM-IV, a diagnosis of alcohol dependence and not alcohol abuse is given when criteria for both are met. Because this study sought to understand the relationships of age at first alcohol use and regular drinking onset to both abuse and dependence and because abuse and dependence were analyzed separately, we did not discount abuse diagnoses when dependence criteria also were met. Because of this deviation from the DSM-IV criteria, the label of "alcohol misuse" instead of alcohol abuse is used in this article. Between 36.2% and 45.2% of those who met diagnostic criteria for alcohol abuse also met criteria for alcohol dependence at each time point.

Alcohol misuse and dependence in the past year were assessed in 1996, 1999, 2002, 2005, and 2008 (ages 21, 24, 27, 30, and 33). The lifetime alcohol-misuse and alcohol-dependence incidence variables were coded as 0 for those who never met the diagnostic threshold and coded as 1 for those who met the diagnostic threshold in at least one assessment. In our sample, the rate of lifetime alcohol misuse was 43.2% and the rate of lifetime alcohol dependence was 25.8%. Between the ages of 21 and 33, annual rates ranged from 13.6% to 23% for alcohol misuse and from 7.7% to 13.5% for alcohol dependence. These prevalence rates are higher than those reported in national samples (e.g., Grant, 1994), which may be expected given our higher risk, urban sample. Chronicity of alcohol misuse and alcohol dependence were assessed using count variables indicating the number of times participants met diagnostic criteria between the ages of 21 and 33. However, not all participants were interviewed at all five waves in adulthood. Therefore, using the simple count variables could produce biased Poisson regression estimates. To explicitly model missingness in the data, we added the amount of exposure (i.e., the length of time an individual was followed in adulthood) to adjust the Poisson regression estimates.

**Predictors.** The primary predictor was the age at first alcohol use. Between 1985 (age 10) and 1989 (age 14), respondents were asked if they ever drank alcohol. Starting in 1990 (age 15) until 2002, participants were asked if they ever drank alcohol "other than a sip or two." No significant changes in the pattern of initiation were detected as a result of this wording change (Kosterman et al., 2000). Starting in 2002 (age 27), and every 3 years thereafter, participants were asked if they had drunk alcohol other than a sip or two in the past 3 years. The prospective measure of age at alcohol initiation was derived by calculating the respondents' age at each interview and was defined as the earliest age at which respondents reported having drunk alcohol.

In the first set of analyses, age at initiation of any alcohol use was defined by a series of mutually exclusive dummy variables representing onset before age 11 (pre-adolescence),

between ages 11 to 14 (early adolescence), between ages 15 and 17 (mid-adolescence), and between ages 18 and 20 (late adolescence). The reference group in the first set of analyses was the hypothesized vulnerable group of 11- to 14-year-old initiators. The second set of analyses examined the age at onset of regular drinking (drinking beer, wine, wine coolers, whiskey, gin, or other distilled spirits three or more times in the past month). Only five participants reported onset of regular drinking before age 11; therefore, a separate pre-adolescent onset group was not created for regular drinking. Instead, age at onset of regular drinking was represented in a series of mutually exclusive dummy variables indicating onset at or before the age of 14, between ages 15 and 17, between ages 18 and 20, and at or after age 21. The reference group in this second set of analyses consisted of those who initiated regular drinking at or after the age of 21 ( $n = 261$ ; 47.5% of the sample for these analyses). The rationale for selecting this reference group was twofold. First, the hypothesized sensitive-period group of early adolescent initiators could not be clearly specified because the earliest group included a combination of pre-adolescent and early adolescent initiators of regular drinking (owing to the sample size limitations described earlier). Second, in light of recent efforts by college administrators to lower the legal drinking age (Amethyst Initiative, 2008), setting the referent group as those who initiated regular drinking at or after the legal age of 21 allows for policy-relevant comparisons.

To address the issue of potential dependence between the reference group in these analyses and the chronicity of alcohol use disorder diagnoses, which were assessed during the same time period, we conducted additional sensitivity analyses, in which the reference group was defined as those who began regular drinking between ages 21 and 24, and the outcomes were respecified as the chronicity of alcohol use disorders between ages of 24 and 33, excluding those who began regular drinking at age 27 or after ( $n = 87$ ). The results of these analyses were similar to those presented in the results section.

*Control variables.* Control variables included self-reports of gender (coded as male = 1, female = 0) and ethnicity (dummy variables for African American, Asian American, Native American and "other" ethnicity, with European American as the reference group). We also controlled for childhood poverty, defined as participation in the National School Lunch/School Breakfast program collected from participants' school records (coded as 1 = eligible for free school lunch between ages 10 and 12, 0 = not eligible), and the average frequency of substance use during adolescence, including tobacco, marijuana, and other illicit drug use (including cocaine, crack, psychedelics, as well as amphetamines, tranquilizers, sedatives, and narcotics without a doctor's permission). Specifically, at each wave between 6th and 12th grade, participants were asked about the number of times in the past month they had used each type of substance. The reports

for each substance were averaged across the five waves to yield the final tobacco, marijuana, and other illicit drug use control variables. A portion of the sample was exposed to a multicomponent preventive intervention in the elementary grades, consisting of teacher training, parenting classes, and social competence training for children (see Hawkins et al., 1999, for a description and analysis of the intervention and effects). Although differences in prevalences and means have been observed between intervention and control groups, prior analyses have shown few differences in the relationships among variables related to the etiology of substance use and the covariance structures of the groups (e.g., Catalano et al., 1996). Similarly, analyses for this report were based on the full sample after determining that there were no substantial group differences in the relationships of the predictors with adult alcohol problems. To address the issue of potential differences in prevalences and means between intervention and control groups, all of the final models were also run with a variable indicating intervention group status. There were no intervention effects on any of the outcomes included in this study, and none of the predictor or control variable coefficients changed after the inclusion of the intervention effects (results not shown but available from the first author).

### *Analysis*

We conducted a series of regression analyses (logistic and Poisson models) to predict the dichotomous lifetime alcohol problem diagnoses and the chronicity count of alcohol misuse and dependence. All analyses were conducted with Stata 10 (StataCorp LP, College Station, TX). The predictors were entered in a series of steps. First, we entered the age at alcohol initiation variables (Model 1). Then we added demographic and background control variables (Model 2). In the last step, we added controls for average adolescent substance use (including tobacco, marijuana, and other illicit drug use; Model 3).

## **Results**

Table 1 provides descriptives for the analytic sample overall and by the age at initiation groups. About 46.7% of those who initiated some alcohol use in adolescence evidenced alcohol misuse and about 28.5% evidenced alcohol dependence at some point in young adulthood. Of those who initiated regular drinking before age 33, 55% evidenced alcohol misuse and 34.6% evidenced alcohol dependence. On a bivariate level, there were no significant differences among the age at onset groups for the lifetime and chronicity of alcohol misuse. However, there were bivariate differences by the age at onset groups in the lifetime and chronicity of alcohol dependence. Ethnicity and childhood poverty were significantly related to age at alcohol use initiation. In general, more European Americans than African, Native, and Asian

TABLE 1. Descriptive statistics for the covariates and risk factors by age at any and regular alcohol use initiation

Covariates	Age at any alcohol use initiation					Age at regular alcohol use initiation				
	Overall	<11 yrs	11-14 yrs	15-17 yrs	18-20 yrs	Overall	≤14 yrs	15-17 yrs	18-20 yrs	≥21 yrs
<i>n</i>	692	72	456	122	42	549	97	106	85	261
Gender										
Male, %	51.6	52.8	50.0	59.0	45.2	55.6	54.6	56.6	63.5	52.9
Ethnicity										
African American, %	25.6	22.2	24.3	33.6	21.4	25.0	29.9	36.8	23.5	18.8
Native American, %	5.8	2.8	6.1	6.6	4.8	6.0	7.2	5.7	4.7	6.1
Asian American, %	18.8	11.1	15.1	30.3	38.1	15.5	7.2	12.3	8.2	22.2
European American, %	49.9	63.9	54.4	29.5	35.7	53.6	55.7	45.3	63.5	52.9
Poverty										
Eligible for free lunch at school, %	50.1	34.7	46.9	65.6	66.7	47.7	49.5	64.2	38.8	43.3
Other substance use in adolescence										
Tobacco use, <i>M</i> ( <i>SD</i> )	1.42 (3.32)	1.11 (2.71)	1.55 (3.62)	1.55 (2.92)	0.26 (0.64)	1.64 (3.55)	2.48 (4.69)	3.50 (4.88)	1.77 (3.44)	0.52 (1.45)
Marijuana use, <i>M</i> ( <i>SD</i> )	0.80 (2.10)	0.61 (1.43)	0.84 (2.19)	0.97 (2.37)	0.18 (0.93)	0.96 (2.29)	1.79 (3.20)	1.72 (2.87)	1.28 (2.53)	0.25 (0.94)
Hard drug use, <i>M</i> ( <i>SD</i> )	0.22 (0.82)	0.25 (0.70)	0.26 (0.95)	0.12 (0.31)	0.02 (0.06)	0.25 (0.88)	0.51 (1.44)	0.45 (1.14)	0.25 (0.75)	0.07 (0.27)
Lifetime diagnosis										
Alcohol misuse, %	46.7	45.8	46.3	54.1	31.0	55.0	52.6	63.2	62.4	50.2
Alcohol dependence, %	28.5	36.1	28.3	31.1	9.5	34.6	35.1	43.4	44.7	27.6
Chronicity of diagnosis										
Alcohol misuse, <i>M</i> ( <i>SD</i> )	0.92 (1.25)	1.08 (1.44)	0.92 (1.24)	0.98 (1.21)	0.55 (0.94)	1.12 (1.31)	1.16 (1.40)	1.29 (1.35)	1.33 (1.33)	0.97 (1.25)
Alcohol dependence, <i>M</i> ( <i>SD</i> )	0.50 (0.97)	0.78 (1.25)	0.49 (0.94)	0.50 (0.96)	0.17 (0.54)	0.62 (1.05)	0.73 (1.21)	0.79 (1.14)	0.87 (1.24)	0.43 (0.83)

Note: Yrs = years.

Americans were in the pre-adolescent and early adolescent initiator groups when any alcohol use was considered. Those who had experienced childhood poverty, as indicated by eligibility for free lunch in fifth to seventh grade, were less likely to be among the earlier any alcohol initiation groups. This is likely because the ethnic minority participants, while having a higher age at any alcohol use initiation, also had higher rates of poverty than the European Americans. Those who initiated regular alcohol use at or after age 21 had lower average tobacco, marijuana, and other illicit drug use during adolescence than any of the adolescent initiator groups.

#### *Sensitive-period hypothesis and alcohol misuse*

Table 2 summarizes the regression models for age at onset of any adolescent drinking predicting the occurrence (lifetime diagnosis by age 33) and chronicity (number of occurrences between 21 and 33, inclusive) of alcohol misuse in adulthood. Participants who initiated any alcohol use between ages 11 and 14 were neither significantly more nor significantly less likely to meet the criteria for lifetime

alcohol misuse than those who initiated at other ages. Initiation of any alcohol use between ages 11 and 14 was not associated with more chronic alcohol misuse. These findings persisted in Models 2 and 3 with the inclusion of sociodemographic and tobacco, marijuana, and other illicit drug control variables. Thus, the sensitive-period hypothesis for the onset of *any* drinking was not supported for the lifetime or chronicity of alcohol misuse.

Consistent with previous studies, males were much more likely to have lifetime alcohol misuse (odds ratio [OR] = 2.54,  $p < .01$ ), and their rate of alcohol misuse was more than two times greater than that of females (incidence rate ratio [IRR] = 2.11,  $p < .01$ ), even when controlling for ethnicity, childhood poverty, and other substance use during adolescence. While holding other variables constant, Native Americans had a rate of alcohol misuse 1.46 times greater than European Americans. Conversely, Asian Americans had a lower rate of alcohol misuse than European Americans (IRR = 0.72,  $p < .05$ ). More frequent marijuana use in adolescence predicted an increased likelihood of lifetime alcohol misuse as well as the duration or chronicity of misuse over

TABLE 2. Regression models for age at onset of any adolescent drinking and alcohol misuse in adulthood

Variable	Alcohol misuse					
	Lifetime diagnosis			Chronicity of diagnosis		
	Model 1 OR	Model 2 OR	Model 3 OR	Model 1 IRR	Model 2 IRR	Model 3 IRR
Age at alcohol use initiation <sup>a</sup>						
Alcohol initiation before 11 years	0.98 (0.25)	0.94 (0.25)	0.96 (0.26)	1.17 (0.20)	1.15 (0.19)	1.20 (0.20)
Alcohol initiation 11–14 years, referent	–	–	–	–	–	–
Alcohol initiation 15–17 years	1.37 (0.28)	1.40 (0.30)	1.40 (0.31)	1.11 (0.14)	1.11 (0.14)	1.13 (0.14)
Alcohol initiation 18–20 years	0.52 <sup>§</sup> (0.18)	0.58 (0.21)	0.64 (0.23)	0.63 <sup>§</sup> (0.17)	0.72 (0.18)	0.77 (0.20)
Gender						
Male		2.70** (0.44)	2.54** (0.42)		2.23** (0.24)	2.11** (0.23)
Ethnicity						
African American <sup>b</sup>		0.73 (0.15)	0.72 (0.16)		0.81 (0.11)	0.81 (0.11)
Native American <sup>b</sup>		1.59 (0.57)	1.58 (0.58)		1.47* (0.26)	1.46* (0.25)
Asian American <sup>b</sup>		0.66 <sup>§</sup> (0.15)	0.74 (0.17)		0.69* (0.11)	0.72* (0.11)
Poverty						
Eligible for free lunch at school vs. not		0.99 (0.17)	0.94 (0.17)		1.02 (0.11)	1.00 (0.11)
Other substance use in adolescence						
Average tobacco use			1.02 (0.03)			1.01 (0.01)
Average marijuana use			1.12* (0.06)			1.05** (0.02)
Average other illicit drug use			1.21 (0.16)			1.08* (0.04)

Notes: Estimates for lifetime diagnosis are odds ratios (OR). Estimates for chronicity of diagnosis are incidence rate ratios (IRR). Standard errors in parentheses. <sup>a</sup>Reference group is alcohol initiation between 11 and 14 years; <sup>b</sup>reference group is European American.

<sup>§</sup> $p < .10$ ; \* $p < .05$ ; \*\* $p < .01$ .

the course of young adulthood (OR = 1.12,  $p < .05$ ; and IRR = 1.05,  $p < .01$ , respectively). Similarly, more frequent other illicit drug use predicted more chronic alcohol misuse (IRR = 1.08,  $p < .05$ ).

Table 3 summarizes the regression models for the age at regular drinking initiation predicting alcohol misuse in young adulthood. Without considering any other covariates, we found some evidence of worse alcohol misuse outcomes for those who initiated regular drinking before the age of 21, particularly for those adolescents who initiated regular drinking between the ages of 15 and 17 (OR = 1.70,  $p < .05$ ; IRR = 1.35,  $p < .05$ ) and the ages of 18 and 20 (OR = 1.64,  $p < .10$ ; IRR = 1.38,  $p < .05$ ) when compared with those who initiated regular drinking after the age of 21. However, after we included demographic characteristics and average other substance use in adolescence, there were no differences in the lifetime occurrence or chronicity of alcohol misuse between those who initiated regular drinking before age 21 when compared with those who delayed the initiation of regular drinking until after age 21 (although those who initiated between ages 15 and 17 still evidenced a trend toward

somewhat higher odds of lifetime and rate of alcohol misuse than those who initiated after age 21). Thus, the sensitive-period hypothesis was not supported for the lifetime or chronicity of alcohol misuse. Being male more than doubled the odds of alcohol misuse (OR = 2.45,  $p < .01$ ), and chronicity of alcohol misuse among males was also greater than that of females (IRR = 1.89,  $p < .01$ ). While controlling other covariates, Native Americans had more chronic misuse than European Americans (IRR = 1.46,  $p < .05$ ). Finally, higher frequency of marijuana and other illicit drug use in adolescence were also related to more chronic adult alcohol misuse (IRR = 1.03,  $p < .05$ ; and IRR = 1.08,  $p < .05$ , respectively).

#### *Sensitive-period hypothesis and alcohol dependence*

Table 4 summarizes the regression models for the age at onset of any adolescent drinking and alcohol dependence in young adulthood. In the unadjusted analyses, initiation of any drinking between ages 18 and 20 was associated with a lower likelihood of lifetime alcohol dependence than initiation between ages 11 and 14 (OR = 0.27,  $p < .05$ ), but this

TABLE 3. Regression models for age at onset of adolescent regular drinking and alcohol misuse in adulthood

Variable	Alcohol misuse					
	Lifetime diagnosis			Chronicity of diagnosis		
	Model 1 OR	Model 2 OR	Model 3 OR	Model 1 IRR	Model 2 IRR	Model 3 IRR
Age at regular alcohol use initiation <sup>a</sup>						
Regular use initiation at/before 14 years	1.10 (0.26)	1.12 (0.28)	1.01 (0.26)	1.28 <sup>§</sup> (0.18)	1.24 (0.17)	1.16 (0.16)
Regular use initiation 15–17 years	1.70* (0.40)	1.83* (0.46)	1.64 <sup>§</sup> (0.44)	1.35* (0.17)	1.35* (0.16)	1.25 <sup>§</sup> (0.15)
Regular use initiation 18–20 years	1.64 <sup>§</sup> (0.42)	1.55 (0.41)	1.44 (0.39)	1.38* (0.18)	1.28 <sup>§</sup> (0.17)	1.23 (0.16)
Regular alcohol use initiation ≥ 21 years, referent	–	–	–	–	–	–
Gender						
Male		2.53** (0.46)	2.45** (0.45)		1.94** (0.21)	1.89** (0.21)
Ethnicity						
African American <sup>b</sup>		0.79 (0.18)	0.77 (0.19)		0.84 (0.11)	0.84 (0.11)
Native American <sup>b</sup>		1.96 (0.80)	1.88 (0.77)		1.48* (0.25)	1.46* (0.25)
Asian American <sup>b</sup>		0.98 (0.27)	1.01 (0.28)		0.86 (0.13)	0.86 (0.13)
Poverty						
Eligible for free lunch at school vs. not		0.90 (0.18)	0.89 (0.18)		0.98 (0.10)	1.00 (0.11)
Other substance use in adolescence						
Average tobacco use			1.00 (0.03)			1.00 (0.01)
Average marijuana use			1.08 (0.06)			1.03* (0.02)
Average other illicit drug use			1.12 (0.15)			1.08* (0.03)

Notes: Estimates for lifetime diagnosis are odds ratios (OR). Estimates for chronicity of diagnosis are incidence rate ratios (IRR). Standard errors in parentheses. <sup>a</sup>Reference group is initiated regular drinking after age 21; <sup>b</sup>reference group is European American.

<sup>§</sup> $p < .10$ ; \* $p < .05$ ; \*\* $p < .01$ .

TABLE 4. Regression models for age at onset of any adolescent drinking and alcohol dependence in adulthood

Variable	Alcohol dependence					
	Lifetime diagnosis			Chronicity of diagnosis		
	Model 1 OR	Model 2 OR	Model 3 OR	Model 1 IRR	Model 2 IRR	Model 3 IRR
Age at alcohol use initiation <sup>a</sup>						
Alcohol initiation before 11 years	1.43 (0.38)	1.41 (0.39)	1.49 (0.42)	1.57* (0.33)	1.57* (0.34)	1.68* (0.36)
Alcohol initiation 11–14 years, referent	–	–	–	–	–	–
Alcohol initiation 15–17 years	1.15 (0.25)	1.16 (0.27)	1.16 (0.28)	1.06 (0.21)	1.03 (0.19)	1.04 (0.19)
Alcohol initiation 18–20 years	0.27* (0.14)	0.30* (0.16)	0.35 <sup>§</sup> (0.19)	0.36* (0.18)	0.40 <sup>§</sup> (0.21)	0.46 (0.24)
Gender						
Male		3.15** (0.58)	2.92** (0.56)		2.38** (0.37)	2.19** (0.35)
Ethnicity						
African American <sup>b</sup>		0.69 (0.16)	0.73 (0.18)		0.78 (0.15)	0.78 (0.16)
Native American <sup>b</sup>		1.56 (0.58)	1.50 (0.57)		1.81* (0.47)	1.74* (0.44)
Asian American <sup>b</sup>		0.53* (0.14)	0.63 <sup>§</sup> (0.17)		0.71 (0.16)	0.78 (0.18)
Poverty						
Eligible for free lunch at school vs. not		1.06 (0.21)	0.97 (0.20)		1.11 (0.17)	1.02 (0.16)
Other substance use in adolescence						
Average tobacco use			1.06* (0.03)			1.03 <sup>§</sup> (0.02)
Average marijuana use			1.10* (0.05)			1.08** (0.03)
Average other illicit drug use			1.08 (0.13)			0.99 (0.07)

Notes: Estimates for lifetime diagnosis are odds ratios (OR). Estimates for chronicity of diagnosis are incidence rate ratios (IRR). Standard errors in parentheses. <sup>a</sup>Reference group is alcohol initiation between 11 and 14 years; <sup>b</sup>reference group is European American.

<sup>§</sup> $p < .10$ ; \* $p < .05$ ; \*\* $p < .01$ .

relationship was reduced to marginal significance when we controlled for sociodemographic and other substance use covariates (OR = 0.35,  $p < .10$ ). Other age groups did not differ from those who initiated any drinking between ages 11 and 14 in lifetime alcohol dependence.

In contrast, age at initiation of any drinking was related to the chronicity of alcohol dependence in young adulthood. Specifically, after including the sociodemographic covariates and adolescent other substance use, those who initiated alcohol use before age 11 had more chronic dependence (IRR = 1.68,  $p < .05$ ) than those who initiated between ages 11 and 14. In addition, the post hoc comparisons with the other age groups revealed the same pattern: Those who initiated alcohol use before age 11 had a rate of alcohol dependence 1.62 times greater ( $p < .01$ ) than those who delayed initiation until they were 15–17 years of age and 3.67 times greater ( $p < .05$ ) than those who did not start drinking alcohol until they were 18–20 years of age. Thus, the very early onset of any drinking but not the hypothesized early adolescent sensitive period was salient in the prediction of alcohol dependence.

Table 5 summarizes regression models for age at onset of regular drinking and alcohol dependence in adulthood. With the exception of the youngest group, the odds of lifetime alcohol dependence were significantly higher for those who initiated regular alcohol use before age 21 (OR = 1.71,  $p < .05$ , for those who initiated between ages 15 and 17; OR = 1.76,  $p < .05$ , for those who initiated between ages 18 and 20). Initiation of regular alcohol use at any time before age 21 was related to a greater rate of or more chronic alcohol dependence in young adulthood than initiation of regular alcohol use at age 21 or later, even after controlling for the demographic and other risk factors. Specifically, controlling for gender, ethnicity, poverty, and other substance use in adolescence, those who initiated regular alcohol use at or before age 14 had more chronic alcohol-dependence problems: a rate that was 1.67 times greater ( $p < .05$ ) than that of those who initiated regular use at or after age 21. Similarly, those who initiated between 15 and 17 years of age and between 18 and 20 years of age also had more chronic alcohol dependence problems: more than 1.5 times the rate of alcohol dependence (IRR = 1.70,  $p < .01$ ; and IRR = 1.85,  $p < .01$ ,



TABLE 5. Regression models for age at onset of adolescent regular drinking and alcohol dependence in adulthood

Variable	Alcohol dependence					
	Lifetime diagnosis			Chronicity of diagnosis		
	Model 1 OR	Model 2 OR	Model 3 OR	Model 1 IRR	Model 2 IRR	Model 3 IRR
Age at regular alcohol use initiation <sup>a</sup>						
Regular use initiation at/before	1.42	1.39	1.19	1.83**	1.80**	1.67*
14 years	(0.36)	(0.37)	(0.33)	(0.38)	(0.36)	(0.34)
Regular use initiation 15–17 years	2.01**	2.11**	1.71*	1.89**	1.90**	1.70**
(0.48)	(0.54)	(0.47)	(0.35)	(0.35)	(0.31)	
Regular use initiation 18–20 years	2.12**	1.95*	1.76*	2.06**	1.95**	1.85**
(0.55)	(0.52)	(0.48)	(0.41)	(0.38)	(0.36)	
Regular alcohol initiation ≥ 21 years, referent	–	–	–	–	–	–
Gender						
Male		2.74**	2.62**		1.96**	1.89**
		(0.54)	(0.52)		(0.31)	(0.30)
Ethnicity						
African American <sup>b</sup>		0.68	0.72		0.76	0.76
		(0.17)	(0.19)		(0.15)	(0.16)
Native American <sup>b</sup>		1.72	1.64		1.75*	1.72*
		(0.68)	(0.65)		(0.46)	(0.44)
Asian American <sup>b</sup>		0.70	0.76		0.94	0.96
		(0.21)	(0.23)		(0.21)	(0.22)
Poverty						
Eligible for free lunch at school vs. not		0.96	0.91		1.03	0.99
		(0.20)	(0.20)		(0.16)	(0.16)
Other substance use in adolescence						
Average tobacco use			1.04			1.01
			(0.03)			(0.02)
Average marijuana use			1.06			1.05 <sup>§</sup>
			(0.05)			(0.03)
Average other illicit drug use			1.08			0.99
			(0.13)			(0.06)

Notes: Estimates for lifetime diagnosis are odds ratios (OR). Estimates for chronicity of diagnosis are incidence rate ratios (IRR). Standard errors in parentheses. <sup>a</sup>Reference group is initiated regular drinking after age 21; <sup>b</sup>reference group is European American. <sup>§</sup> $p < .10$ ; \* $p < .05$ ; \*\* $p < .01$ .

respectively) as those who delayed the initiation of regular drinking until age of 21 years or older. Post hoc analyses revealed no statistically significant differences in the chronicity of alcohol dependence among any of the groups that initiated regular alcohol use before age 21.

Associations between the control variables and alcohol dependence were similar to those in the alcohol misuse models. Male gender was related to both increased likelihood of lifetime occurrence and longer chronicity of alcohol dependence. Compared with females and independent of ethnicity, childhood poverty, and other substance use during adolescence, young males were more than twice as likely to have lifetime alcohol dependence and their rate of chronicity of alcohol dependence in adulthood was also greater (OR = 2.92,  $p < .01$ , and IRR = 2.19,  $p < .01$ , respectively, for the age at any drinking; OR = 2.62,  $p < .01$ , and IRR = 1.89,  $p < .01$ , respectively, for the age at regular drinking initiation models). Native Americans had more chronic alcohol dependence than European Americans in both alcohol use initiation (IRR = 1.74,  $p < .05$ ) and regular alcohol use initiation models (IRR = 1.72,  $p < .05$ ). Finally, the frequency

of marijuana use during adolescence was related to a greater rate of alcohol dependence in alcohol initiation (IRR = 1.08,  $p < .01$ ) and regular alcohol use initiation (IRR = 1.05,  $p < .10$ ) models. Overall, the results in Table 5 suggest the onset of regular drinking at any point during adolescence places youths at risk for later alcohol dependence.

## Discussion

This study examined the nature of the association between age at alcohol use onset and adult alcohol misuse and dependence by testing the hypothesis that early adolescence is a sensitive period during which the initiation of alcohol use is particularly risky. This study found that the onset of alcohol use before age 11, when compared with initiation during early adolescence, was related to an increased chronicity of adult alcohol dependence, even after accounting for sociodemographic controls and other substance use in adolescence. The present study finds no evidence in support of age 11–14 (early adolescence) as a sensitive period for the onset of any drinking. Findings related to the onset of

regular alcohol use and chronicity of alcohol dependence suggest that the onset of regular drinking before age 21 is problematic, but there is no one period that is more sensitive than others. Specifically, although those who started drinking regularly before age 21 had a greater rate of alcohol dependence, we did not find evidence that initiation of regular use of alcohol at or before age 14 was related to greater chronicity of alcohol dependence in adulthood than the initiation of regular use of alcohol in middle or late adolescence.

The majority (59.2%) of this sample began drinking alcohol between the ages of 11 and 14 and the onset of alcohol use during this early adolescent period did not predict increased likelihood or rate of later alcohol use disorders. Earlier studies that reported worse outcomes for those who initiated alcohol use during or before early adolescence were based on retrospective reports (Dawson et al., 2008; DeWit et al., 2000; Grant and Dawson, 1997; Hingson et al., 2006). However, retrospective reports of routine behaviors such as the onset and use of alcohol can be subject to forward telescoping bias (e.g., Groves, 1989). Thus, our finding of worse adult alcohol outcomes for those who reported prospectively that they have initiated alcohol use before age 11 could correspond to the time frame of early adolescence reported in studies based on retrospective reports.

Furthermore, we did not find a specific adolescent sensitive period for the onset of regular drinking. In this study, the onset of regular drinking at any age before age 21 predicted greater chronicity of adult alcohol-dependence diagnosis through age 33. This relationship persisted even after we controlled for sociodemographic risk and other substance use in adolescence. Moreover, even the likelihood of lifetime alcohol-dependence diagnosis was greater for those who initiated regular alcohol use between ages 15 and 20 years than those who initiated at age 21 or older.

The association between the onset of regular alcohol use during adolescence and young adult alcohol problems could be the result of compromised subsequent social, emotional, and cognitive development. For example, adolescents who begin drinking regularly before they are of legal age are exposed to social and physical environments that may promote problem behaviors and exposure to other risk factors associated with maladaptive outcomes (Jessor, 1991). Future studies should examine possible social and environmental mediators of the relationships between alcohol use in adolescence and later alcohol misuse or dependence.

The finding that onset of regular drinking at any age before 21 is related to a greater chronicity of alcohol dependence could also be the result of compromised biological and cognitive development. Recent research suggests that brain maturation proceeds into the late teenage years (Steinberg, 2005). Regular alcohol consumption can affect brain development and disrupt the development of important behavioral mechanisms such as impulse inhibition and regulation of motivation, which can lead to later alcohol

use problems (e.g., Crews et al., 2007). Disruption in any of these domains—whether socioemotional, cognitive, or biological—could lead to an increased likelihood and more chronic occurrence of alcohol dependence in adulthood.

### *Limitations*

The following limitations should be considered when interpreting the present findings. First, the age at onset of regular drinking was determined based on questions about the frequency of drinking in the past month. It is possible that some of those who were considered abstainers from regular drinking at a given time point were in fact drinkers who did not engage in this behavior in the month before the interview. Similarly, in this study, alcohol misuse and dependence in young adulthood were assessed every 3 years between the ages of 21 and 33, and each interview assessed alcohol problems in the year before the interview. Thus, the incidence and rate of alcohol misuse and dependence represent repeated snapshots of alcohol-related problems and might underestimate actual prevalence. Second, this study relied on self-reports of alcohol use, which was illegal when participants were adolescents. Thus, the findings may be subject to underreporting. However, in the context of longitudinal studies where trust and rapport are developed over years, the potential sources of response bias in substance use reporting can be minimized (e.g., Del Boca and Darkes, 2003; Langenbucher and Merrill, 2001).

### *Prevention implications*

The finding that the onset of alcohol use before age 11 predicted greater chronicity of adult alcohol dependence than did alcohol use onset during ages 11-14 suggests the importance of delaying the onset of alcohol use through prevention efforts in the elementary grades. In addition, the finding that the onset of regular drinking at any time before age 21 was predictive of later alcohol dependence suggests that prevention efforts should focus especially on preventing the onset of regular drinking before age 21. It is noteworthy that the odds and rate of alcohol misuse and dependence problems were increased for those participants who reported more frequent marijuana use during adolescence. Thus, prevention efforts should focus on shared risk factors for adolescent drinking and the use of other drugs.

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