Three-Year Changes in Adult Risk Drinking Behavior in Relation to the Course of Alcohol-Use Disorders*

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ABSTRACT. Objective: This study examines the associations between the course of alcohol-use disorder (AUD) and changes in average daily volume of ethanol intake, frequency of risk drinking, and maximum quantity of drinks consumed per day over a 3-year follow-up interval in a sample of U.S. adults. **Method:** Data were taken from a longitudinal study of a nationally representative sample of U.S. adults, who were 18 years of age and older (mean age = 46.4) when initially interviewed in 2001-2002 and successfully reinterviewed approximately 3 years later (n = 22,245 baseline drinkers). The time reference period for the drinking measures was the 12 months preceding the interview. Changes in consumption reflect differences between Wave 1 and Wave 2 measures for individuals with nonmissing values at both Waves (n = 22,003 for volume of intake, 22,132 for frequency of risk drinking and 21,942 for maximum quantity of drinks). **Results:** There were positive changes in all consumption measures associated with developing an AUD and negative changes associated with remission of an AUD, even among individuals who continued to drink. Increases and decreases associated with onset and offset of dependence exceeded those associated with onset/offset of abuse only, and the decreases associated with full remission from dependence exceeded those associated with partial remission. There were few changes in consumption among individuals whose AUD status did not change. Interactions of AUD transitions with other factors indicate that development of an AUD is associated with a greater increase in consumption among men, possibly reflecting their greater total body water and lower blood alcohol concentration in response to a given dose of ethanol, and among individuals with high baseline levels of consumption. **Conclusions:** Changes in consumption associated with onset and offset of AUD are substantial enough to have important implications for the risk of associated physical and psychological harm. (*J. Stud. Alcohol Drugs* **69:** 866-877, 2008)

NUMEROUS STUDIES HAVE EXAMINED changes in volume and pattern of drinking over the life course, with most focusing on the heavy drinking trajectories from adolescence to early adulthood (Chassin et al., 2002; Hill et al., 2000; Oesterle et al., 2004; Schulenberg et al., 1996; Tucker et al., 2003; Windle et al., 2005). Within the developmental framework of this rich literature, numerous factors have discriminated among different drinking trajectories. Virtually all studies have found male gender to be associated with higher baseline levels of heavy drinking and trajectories more indicative of increasing or continued heavy drinking. Other factors associated with change in drinking behavior over time have included baseline levels of alcohol and other substance use (Auerbach and Collins, 2006; Chassin et al., 2002; Hill et al., 2000; Sher and Rutledge, 2007; Windle et al., 2005); family history of alcoholism (Chassin et al., 2002, 2004; Jackson et al., 2001; Timberlake et al., 2007); borderline personality disorder (PD) and/or behavioral undercontrol and impulsivity (Rohde et al., 2001; Rutledge and Sher, 2001); externalizing behavior, delinquency, conduct disorder, and antisocial PD (Bucholz et al., 2000; Chassin et al., 2002; Hill et al., 2000; Jackson and Sher, 2005; Tucker et al., 2003); and early initiation of drinking (Casswell et al., 2002; Goudriaan et al., 2007; Windle et al., 2005).

Far fewer longitudinal studies have examined changes in drinking patterns past the early adult years. The decline in heavy drinking with age that has been reported consistently in cross-sectional studies implies a decline in heavy drinking with age, but cross-sectional data run the risk of confounding age, period, and cohort effects. However, using age-period-cohort models to distinguish these effects, Kerr et al. (2004) concluded that beer and distilled sprits consumption (although not wine consumption) did decline significantly with age. Similarly, in another study of a large, nationally representative U.S. sample of adults followed up four times over a 22-year period, the proportions of men and women classified as heavy drinkers (usual consumption of 5+/4+ drinks per drinking occasion, respectively) declined steadily with increasing age, independent of period effects.

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The gender differential also narrowed with advancing age. Other factors associated with a higher prevalence of heavy drinking across the span of ages included not being married, smoking, lower income, and lower education (Karlamangla et al., 2006). The education finding supports other longitudinal studies tracking individuals beyond the college years that have found that lower levels of heavy drinking in their late 20s and 30s by those who attended college (Lanza and Collins, 2006; Muthén and Muthén, 2000).

Consistent with the general age-related decline in heavy drinking reported in the studies above, a study based on retrospective lifetime drinking histories from small samples of non-Hispanic white, black, and Mexican-American men found that all three groups reported the highest levels of mean quantity, overall frequency, and total weekly volume of consumption during a drinking stage that occurred in their late teens to early 20s (Neff and Dassori, 1998). However, a large study of adult primary care patients interviewed at two points 5 years apart did not find a significant association of age with shifts from lighter (≥1 drinks per day) to heavier (≥3 drinks per day) drinking after controlling for other significant correlates of change. Rather, it found that individuals at risk of shifting to heavier drinking were disproportionately men, black, current or former smokers, and those who reported mood or anxiety problems (Midanik et al., 1990). Moreover, a meta-analysis of longitudinal data for adult samples conducted as part of the Collaborative Alcohol-Related Longitudinal Project determined that after age 30 the age-related decline in drinking frequency was no longer statistically significant (Johnstone et al., 1996). Other studies from this project found that changes in quantity, frequency, and volume of consumption were positively related to baseline values of those measures and were decreased among individuals who were married or got married over the follow-up interval (Leino et al., 1995).

Longitudinal studies of treatment and quasi-treatment samples have shown long-term decreases in consumption in association with recovery from an alcohol-use disorder (AUD), with the extent or trajectory of decrease demonstrating considerable instability over time and varying as a function of baseline characteristics and treatment experience (e.g., Delucchi et al., 2004; Maisto et al., 2002; Matzger et al., 2004; Moos and Moos, 2005; Shaw et al., 1997; Skog and Duckert, 1993). There are, however, virtually no general population studies charting changes in consumption as a function of the natural course of AUD. Based on retrospective data from two Canadian populations, Sobell et al. (1996) reported that the current consumption levels of nonabstinent, resolved problem drinkers were within sampling range of levels for social drinkers, implying a decrease in relation to remission of AUD, but they did not report on the actual extent to which the resolved problem drinkers' consumption levels had declined. Similarly, Dawson (1996) reported that individuals with prior alcohol dependence who had returned to drinking without an AUD had far lower volumes of ethanol intake and frequencies of risk drinking and intoxication than individuals with a current AUD, but these differences could reflect lower consumption levels to begin with for those who recovered as well as differences exclusively attributable to the change in AUD status. Also virtually absent in the literature are studies that document changes in consumption associated with the development of alcohol abuse and dependence.

The scant literature on changes in consumption related to course of AUD is unfortunate, because decreases in consumption corresponding to recovery from AUD have been related to improved quality of life in treatment samples (Finney and Moos, 1992; Kraemer et al., 2002), and increased consumption levels have been correlated with increased risks of psychological, physical, social, and legal harm in both cross-sectional and longitudinal studies (Dawson et al., 2008; Murray et al., 2002; Perreira and Sloan, 2002; Pletcher et al., 2005). Thus consumption changes associated with onset and offset of AUD are of considerable public health importance. Moreover, the magnitude of these changes cannot be inferred from the more extensive body of research in which consumption as an independent variable has been used to predict AUD transitions as outcomes (e.g., Caetano et al., 1997; Dawson and Archer, 1993; Dawson et al., 2008; Grant and Harford, 1990; Woerle et al., 2007). In addition, changes in consumption associated with AUD transitions may enhance understanding of the etiology of AUD and help to address the debate as to the utility of the distinction between abuse and dependence and their hierarchy in the current Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) criteria (American Psychiatric Association, 1994).

The present study was designed to fill this gap in the literature. Using data from a longitudinal sample of U.S. adults interviewed at two points approximately 3 years apart, it classifies individuals into 12 categories reflecting transitions in DSM-IV AUD status over the follow-up interval. It then compares changes over the follow-up interval across these 12 categories for three commonly used measures of heavy or risk drinking: average daily volume (ADV) of ethanol intake, frequency of risk drinking, and maximum drinks consumed in a single day. These comparisons are based on models that adjust for baseline consumption levels, sociodemographic characteristics, and psychopathology as well as family history of alcoholism and age at first drink. On the basis of the extant literature and the DSM-IV articulation of alcohol abuse as secondary to and thus presumably milder than alcohol dependence, we anticipate that (1) development of an AUD will be associated with increases in all measures of consumption; (2) remission of AUD will be associated with decreases in consumption, even among individuals who continued to drink; (3) increases in consumption associated with developing dependence will be larger than those asso-

ciated with developing abuse only; (4) decreases associated with remission from dependence will be greater than those associated with remission from abuse only; and (5) decreases associated with full remission from dependence will be greater than those associated with partial remission.

Given the fairly short follow-up interval, we do not anticipate any change in consumption among individuals who did not have an AUD at either time, except for reductions related to individuals who stopped drinking altogether over the follow-up interval.

Method

Sample

The data in this analysis came from Waves 1 and 2 of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), designed by the National Institute on Alcohol Abuse and Alcoholism. The 2001-2002 Wave 1 NESARC sample, which represented the population residing in households and noninstitutional group quarters in all 50 states and the District of Columbia, consisted of 43,093 U.S. adults 18 years of age and older (response rate, 81.0%). Data were collected in personal interviews conducted by interviewers trained by the U.S. Bureau of the Census. In Wave 2, interviewers attempted to reinterview all respondents from Wave 1 who had not died, become incapacitated or institutionalized, left the country, or entered the military (eligible n = 39,959). The reinterview rate among those eligible was 86.7%, yielding a Wave 2 sample of 34,653 adults and a cumulative response rate of 70.2%. As in Wave 1, Wave 2 data were weighted to reflect design characteristics of the NESARC and account for oversampling. Adjustment for nonresponse across sociodemographic characteristics and the presence of any lifetime Wave 1 NESARC substance-use disorder or other psychiatric disorder was performed at the household and person levels. Weighted data were then adjusted to be representative of the civilian population of the United States on socioeconomic variables based on the 2000 Decennial Census. All potential NESARC respondents were informed in writing about the nature of the survey, the statistical uses of the survey data, the voluntary aspect of their participation, and the Federal laws that rigorously provide for the confidentiality of identifiable survey information. Only respondents consenting to participate after receiving this information were interviewed. The research protocol, including informed consent procedures, received full ethical review and approval from the U.S. Census Bureau and the U.S. Office of Management and Budget. This analysis is based on a subsample of respondents who had consumed at least one drink in the year immediately preceding the Wave 1 interview and were reinterviewed at Wave 2 (n = 22,245), including those who had stopped drinking by the time of the Wave 2 interview (n = 3,016).

Measures of risk drinking

Annual number of drinks was calculated as a weighted function of overall drinking frequency, usual quantity of drinks, maximum quantity of drinks and the frequency with which the maximum was consumed, and the frequency of drinking 5+ drinks, and was converted to annual volume of ethanol intake in ounces using additional information on usual size of drink and ethanol content of usual brand consumed. Volume was estimated separately for coolers, beer, wine, and distilled spirits and then summed across beverages and divided by 365 to obtain ADV of ethanol intake. Men's frequency of risk drinking was estimated as the largest of the frequency of drinking five or more (5+) alcoholic drinks in a single day and the frequencies of drinking ≥2.7 oz (≥4.5 standard drinks) of ethanol in a single day of coolers, beer, wine, or distilled spirits, where beverage-specific ounces of ethanol intake per day were calculated based on number of drinks, size of drink, and ethanol content of main brand consumed. Women's frequency of risk drinking was estimated as the largest of the frequency of drinking four or more (4+) alcoholic drinks in a single day and the frequencies of drinking ≥2.1 oz (≥3.5 standard drinks) of ethanol in a single day of coolers, beer, wine, or distilled spirits. All frequencies reflect number of risk drinking days in the year preceding the Wave 1 interview. Maximum quantity refers to the largest number of drinks consumed on any day in the year preceding the Wave 1 interview (all alcoholic beverage types combined). The question was asked in open-ended format. Based on a reinterview of a subsample of the Wave 1 NESARC respondents after 10 weeks, on average, test-retest reliability was good for ADV of ethanol intake and frequency of risk drinking, with intraclass correlation coefficients (ICC) of .72 and .70, respectively, and excellent for maximum quantity of drinks (ICC = .84) (Grant et al., 2003). To reduce skew, improve model fit, and avoid undue influence of outliers, ADV was top-coded to a high of 14.4 oz (affecting the upper 0.1% of cases), and maximum quantity of drinks to a high of 24 (affecting the upper 0.5% of cases).

Changes in risk drinking

In the Wave 2 interview, individuals who had stopped drinking were assigned values of 0 for all consumption measures. Changes in risk drinking were measured by subtracting the Wave 1 value from the Wave 2 value, with negative values indicating a decrease and positive values indicating an increase in consumption over the follow-up period.

Course of alcohol-use disorder

At both Wave 1 and Wave 2, respondents had to report one or more symptoms of at least three of the seven DSM-IV dependence criteria (American Psychiatric Association,

1994) in the year preceding interview to be classified with alcohol dependence. (The withdrawal criterion was measured as a syndrome, requiring at least two symptoms.) To be classified with abuse, they had to report the occurrence of at least one symptom of any of the four DSM-IV abuse criteria (for further detail, see Grant et al., 2004a). The test-retest reliability for past-year AUDs was excellent ($\kappa = .76$) (Grant et al., 2003). Other studies have demonstrated the concurrent and construct validity of the Alcohol Use Disorders and Associated Disabilities Interview Schedule—DMS-IV Version (AUDADIS-IV; Canino et al., 1999; Cottler et al., 1997; Hasin et al., 1997; Muthén et al., 1993; Nelson et al., 1999; Pull et al., 1997).

Consistent with the hierarchy established in the DSM-IV, past-year AUD status at Wave 1 and Wave 2 was described by three categories: no AUD, abuse only, and dependence (with or without abuse). Transitions in AUD status were described by the 12 categories listed in the following. Individuals who remained without an AUD at both waves were separated to distinguish those who continued and stopped drinking, because the former comprise a more meaningful basis for comparison with individuals who developed an AUD over the follow-up interval.

(1) No AUD at Wave 1; remained without AUD and continued drinking at Wave 2.
(2) No AUD at Wave 1; remained without AUD and stopped drinking by Wave 2.
(3) No AUD at Wave 1; developed abuse only by Wave 2.
(4) No AUD at Wave 1; developed dependence (with or without abuse) by Wave 2.
(5) Abuse only at Wave 1; nonabstinent remission to no AUD by Wave 2.
(6) Abuse only at Wave 1; abstinent remission to no AUD by Wave 2.
(7) Abuse only at Wave 1; remained abuse only at Wave 2.
(8) Abuse only at Wave 1; developed dependence (with or without abuse) by Wave 2.
(9) Dependence at Wave 1; nonabstinent remission to no AUD by Wave 2.
(10) Dependence at Wave 1; abstinent remission to no AUD by Wave 2.
(11) Dependence at Wave 1; partial remission (abuse or subclinical dependence) at Wave 2.
(12) Dependence at Wave 1; remained dependent at Wave 2.

Measurement of baseline covariates

Dichotomous measures of other baseline substance-use and mental disorders were assessed using the AUDADIS-IV (Grant et al., 2001). Unless otherwise specified, all baseline measures reflect the 12 months immediately preceding the Wave 1 interview. The derivation and psychometric properties of the NESARC mood disorders, anxiety disorders, and PDs have been described in detail elsewhere (Grant et al., 2004b, 2005). Any mood disorder included major depressive episode, dysthymia, mania, or hypomania; and any anxiety disorder included panic disorder (with or without agoraphobia), specific or social phobia, or generalized anxiety disorder. Baseline disorders ruled out those that were exclusively illness-induced or substance-induced, and major depressive episode also ruled out bereavement. The NESARC measured 10 PDs: antisocial, borderline, avoidant, paranoid, dependent, schizoid, obsessive/compulsive, histrionic, narcissistic, and schizotypal disorders. All reflected lifetime behavioral patterns and not reactions to specific circumstances. Because of their prominence in the previously cited literature, borderline and antisocial PDs were considered separately from the other 8 PDs, which were combined into a single category. Any drug-use disorder consisted of either nicotine dependence, or abuse or dependence for any of the following types of illicit drugs: sedatives, tranquilizers, painkillers, stimulants, marijuana, cocaine/crack, hallucinogens, inhalants/solvents, heroin, or other illicit drugs. Abuse and dependence were classified using algorithms parallel to those described above for alcohol abuse and dependence.

Baseline demographics included age (top-coded to 90 and older on the data tape), gender, race/ethnicity (non-Hispanic white, black, Native American, Asian, and Hispanic, ultimately collapsed to black vs nonblack based on initial model testing), whether currently married, whether ever attended or completed college, and presence of children younger than age 18. Family history of alcoholism (positive vs negative) reflected respondent-reported symptoms of AUD in any of 14 different types of first and second degree relatives. Age at first drink, measured in single years of age, excluded small tastes or sips of someone else's drink and was top- and bottom-coded to a range of 12 to 25 years to avoid undue influence of outlier values.

Analysis

Analyses were performed using SUDAAN (Research Triangle Institute, 2001), a software package that uses Taylor series linearization to adjust variance estimates for complex survey designs. Multiple linear regression models were used to estimate the associations of the course of AUD with the magnitude of the change in each of the three consumption measures. Separate sets of models were run for (1) individuals with no AUD at baseline, (2) individuals with abuse only, and (3) individuals with dependence, to achieve the most meaningful comparisons for testing our expected findings. All models tested for interactions of the other model covariates with the dichotomous variables representing course of AUD. Because of the number of interactions tested, a p value of $<.005$ was required for an interaction to be deemed significant.

Results

The majority of Wave 1 past-year drinkers who were reinterviewed at Wave 2 had no AUD at either point in time (Table 1). This included individuals who were past-year drinkers at both Waves (67.9%) and individuals who had stopped drinking by Wave 2 (11.9%). Within the 3-year follow-up interval, 4.7% of drinkers developed alcohol abuse only, and 3.1% developed alcohol dependence. These figures include both first incidence and recurrence. In addition, 4.0% had abuse only at Wave 1 but remitted to no AUD (3.7% nonabstinent remission and 0.3% abstinent remission), 2.0% continued to have abuse only, and 1.0% had abuse only at Wave 1 that progressed to dependence. Finally, 1.3% had dependence at Wave 1 that had fully remitted (1.0% nonabstinent remission and 0.3% abstinent remission), 2.2% had dependence that had partially remitted, and 2.0% remained dependent.

As indicated in Table 1, the 12 groups varied strongly in terms of sociodemographic characteristics and comorbidity. Those who were consistently without an AUD were the oldest, least likely to be men, and least likely to have other substance-use disorders or antisocial PD at baseline. They were also the least likely to have a positive family history of alcoholism and had the oldest mean age at first drink. Individuals with alcohol dependence at either Wave 1 or Wave 2 had the highest rates of mood, anxiety, personality, and other substance-use disorders, followed by those with abuse only at either time. Among individuals who remitted from alcohol abuse over the follow-up interval, those with abstinent remission were more likely to be women, members of minority race/ethnic groups, have less than a college education, and have children in their households. They were also more likely to have had baseline anxiety disorders and PDs.

Table 2 shows mean values for the three consumption measures at Wave 1 and Wave 2, as well as the changes in consumption over the follow-up interval, in relation to transitions in AUD status. Among individuals with no AUD at baseline, those who developed abuse or dependence had significant increases in all three measures of consumption. For those who developed abuse, the largest proportional increase was in the frequency of risk drinking, which rose by 65% from 34.6 to 57.1 days. Among those who developed dependence, the proportional increases were equally large for ADV of ethanol intake and frequency of risk drinking, each of which more than doubled. Both the absolute and proportional increases were greater for individuals who developed dependence than for those who developed abuse.

Among individuals with abuse only at baseline, there were significant decreases in all measures of consumption associated with both nonabstinent and abstinent remission to no AUD and significant increases in all consumption measures associated with progression to dependence. Again, maximum quantity of drinks showed less sensitivity to these transitions in AUD status than did ADV and frequency of risk drinking. Among individuals with dependence at baseline, there were decreases in consumption associated with both partial and full remission. These were greatest for abstinent remission, intermediate for nonabstinent remission and smallest for partial remission.

Among individuals whose AUD status did not change, regardless of whether they had no AUD, abuse only, or dependence at baseline, changes in consumption generally were not statistically significant. However, there was a small but significant increase in maximum quantity of drinks among individuals who continued drinking without an AUD and a small but significant decrease in ADV of ethanol intake among individuals with abuse only at Wave 1 and Wave 2.

Also noteworthy in Table 2 is the fact that individuals who developed an AUD were already heavier drinkers at baseline, by any measure of consumption, than those who remained without an AUD. For example, individuals who developed abuse only had a baseline ADV of ethanol intake that was more than twice as great as those who continued drinking without an AUD (0.70 vs 0.33 oz) and a frequency of risk drinking that was almost three times as great (34.6 vs 12.6 days, respectively). The discrepancies were even greater relative to individuals who developed dependence, whose baseline values also exceeded those of individuals who developed abuse only.

Among individuals who remitted from abuse only, those with abstinent remission had almost twice as high a baseline volume of consumption as those with nonabstinent remission, and they also had significantly greater frequencies of risk drinking and maximum quantities of drinks consumed at baseline. In contrast, individuals with nonabstinent and abstinent remission from alcohol dependence had almost identical baseline volumes of intake and differed only in terms of frequency of risk drinking (118.3 vs 152.7 days).

Table 3 contains selected parameters from a series of main-effects multiple linear regression models whose beta parameters reflect changes in consumption associated with transitions in AUD status, after adjustment for baseline consumption, sociodemographic characteristics and psychiatric comorbidity. The top third of the table is based on individuals who did not have an AUD at baseline, comparing those who remained without an AUD but stopped drinking and those who developed abuse only or dependence with those who remained without an AUD and continued drinking. The middle third of the table compares individuals who continued to have abuse only with those who either remitted to no AUD or progressed to dependence, and the bottom third compares individuals who continued to be dependent with those who achieved partial or full remission. The model parameters associated with different AUD transitions cannot be compared directly with the changes in consumption for the comparable AUD transition categories in Table 2. First, the model parameters are adjusted for sociodemographic characteristics and

TABLE 1. Percentage distribution and characteristics of Wave 1 past-year drinkers, by transition in AUD status between Waves 1 and 2: Past-year drinkers at Wave 1

	Individuals without an AUD at baseline				Individuals with abuse only at baseline				Individuals with dependence[a] at baseline			
	Remained no AUD											
Variable	Continued drinking	Stopped drinking	Developed abuse only	Developed dependence[a]	Nonabst. remission to no AUD	Abst. remission to no AUD	Remained abuse only	Developed dependence[a]	Nonabst. remission to no AUD	Abst. remission to no AUD	Partially remitted	Remained dependent[a]
Cases in category, *n*	14,988	2,867	1,011	685	824	73	423	202	216	76	458	422
Baseline drinkers in category, % (SE)	67.9 (0.5)	11.9 (0.4)	4.7 (0.2)	3.1 (0.2)	3.7 (0.2)	0.3 (0.0)	2.0 (0.1)	1.0 (0.1)	1.0 (0.1)	0.3 (0.0)	2.2 (0.1)	2.0 (0.1)
Characteristics												
Age, mean (SE)	44.4 (0.2)	46.8 (0.4)	38.0 (0.6)	34.7 (0.6)	38.6 (0.6)	36.8 (1.6)	38.0 (0.7)	32.8 (1.0)	30.5 (1.0)	31.6 (1.4)	30.2 (0.6)	32.8 (0.7)
Male, % (SE)	48.5 (0.6)	46.5 (1.3)	71.3 (1.5)	64.4 (2.2)	67.1 (1.8)	58.9 (7.1)	80.5 (2.1)	76.3 (3.8)	62.2 (4.3)	73.4 (5.8)	65.9 (2.4)	72.4 (2.5)
Non-Hisp. white, % (SE)	77.3 (1.3)	65.2 (2.2)	82.3 (1.4)	67.6 (2.5)	78.5 (2.0)	56.6 (7.5)	82.9 (2.2)	66.6 (4.4)	65.7 (4.4)	56.2 (7.0)	70.4 (2.8)	73.6 (3.3)
Married, % (SE)	68.1 (0.7)	63.4 (1.1)	58.0 (1.7)	47.5 (2.3)	53.5 (2.1)	50.0 (7.1)	59.2 (2.9)	49.7 (4.0)	39.6 (4.0)	32.5 (6.1)	33.7 (2.6)	34.3 (2.9)
Attended college, % (SE)	67.0 (0.7)	50.9 (1.2)	66.5 (1.8)	56.2 (2.6)	66.7 (2.0)	34.8 (6.8)	62.4 (2.7)	58.0 (4.9)	58.7 (4.0)	54.2 (6.6)	61.8 (2.8)	53.4 (3.2)
Children <18 years, % (SE)	43.6 (0.6)	42.6 (1.2)	42.5 (1.8)	36.6 (2.3)	40.7 (2.2)	62.3 (6.4)	47.8 (2.9)	47.0 (4.5)	38.4 (4.0)	40.9 (6.5)	37.2 (2.6)	30.7 (2.6)
FHP alcoholism, % (SE)	53.8 (0.8)	51.8 (1.4)	57.5 (1.8)	65.6 (2.4)	65.0 (2.0)	76.0 (6.0)	64.9 (2.7)	67.2 (4.1)	66.3 (3.9)	78.0 (6.0)	69.4 (2.6)	79.6 (2.3)
Age 1st drink, mean (SE)	19.0 (0.0)	19.8 (0.1)	17.9 (0.1)	17.8 (0.1)	17.6 (0.1)	16.9 (0.5)	17.3 (0.1)	17.2 (0.2)	16.9 (0.3)	16.7 (0.2)	17.0 (0.1)	16.7 (0.2)
Any past-year mood dx, % (SE)	8.0 (0.3)	9.9 (0.7)	8.3 (1.0)	14.2 (1.7)	12.5 (1.5)	18.9 (5.2)	8.5 (1.6)	17.5 (3.0)	30.0 (3.9)	27.6 (6.0)	23.8 (2.3)	30.7 (3.0)
Any past-year anxiety dx, % (SE)	10.7 (0.4)	11.6 (0.8)	7.7 (1.0)	13.7 (1.7)	11.8 (1.3)	26.7 (6.9)	11.5 (2.0)	11.4 (2.4)	21.9 (3.4)	17.4 (5.2)	24.0 (2.3)	28.6 (2.9)
Antisocial PD, % (SE)	2.8 (0.2)	3.1 (0.5)	4.9 (0.9)	7.1 (1.3)	7.9 (1.3)	14.3 (5.0)	5.9 (1.3)	10.2 (2.1)	16.5 (3.2)	20.5 (6.2)	14.7 (2.2)	22.8 (2.5)
Borderline PD, % (SE)	4.5 (0.2)	6.7 (0.6)	5.3 (0.7)	22.3 (1.8)	4.9 (0.9)	4.7 (2.4)	6.7 (1.4)	15.6 (2.6)	11.0 (2.8)	17.5 (4.9)	13.4 (1.8)	30.3 (2.8)
Any other PD, % (SE)	16.5 (0.4)	18.6 (1.0)	19.3 (1.4)	35.8 (2.4)	19.5 (1.7)	30.6 (6.2)	23.6 (2.5)	34.4 (4.2)	37.0 (3.9)	42.9 (7.0)	34.4 (3.0)	49.3 (2.8)
Any other substance use dx, % (SE)	11.9 (0.4)	12.9 (1.0)	17.7 (1.5)	26.7 (1.9)	25.9 (2.0)	40.6 (7.3)	29.6 (2.6)	32.3 (3.9)	49.2 (4.3)	46.2 (7.1)	45.9 (2.9)	60.7 (3.2)

Notes: Standard errors of 0.0 denote <0.05. AUD = Alcohol-use disorder; nonabst. = nonabstinent; abst. = abstinent; Hisp. = Hispanic; FHP = family-history positive; dx = diagnosis; PD = personality disorder. [a]With or without abuse.

TABLE 2. Wave 1 and Wave 2 past-year consumption measures and changes in consumption between Waves 1 and 2, by transition in AUD status: Past-year drinkers at Wave 1

	Average daily volume of ethanol intake (oz)			Frequency of risk drinking, days			Maximum quantity of drinks consumed		
AUD transition	Wave 1	Wave 2	Net change	Wave 1	Wave 2	Net change	Wave 1	Wave 2	Net change
Individuals without an AUD at Wave 1									
Remained without AUD and continued drinking	0.33 (0.01)	0.33 (0.01)	-0.00 (0.01)	12.6 (0.5)	12.5 (0.5)	-0.1 (0.5)	3.4 (0.1)	3.7 (0.1)	0.3 (0.1)
Remained without AUD and stopped drinking	0.15 (0.01)	0.00 (0.00)	-0.15 (0.01)	6.1 (0.7)	0.0 (0.0)	-6.1 (0.7)	2.4 (0.1)	0.0 (0.0)	-2.4 (0.0)
Developed abuse only	0.70 (0.04)	1.00 (0.05)	0.30 (0.04)	34.6 (2.4)	57.1 (3.1)	22.5 (3.2)	5.8 (0.2)	8.2 (0.2)	2.4 (0.2)
Developed dependence[a]	0.84 (0.06)	1.84 (0.11)	1.00 (0.11)	49.5 (4.2)	105.4 (5.2)	55.9 (5.5)	6.2 (0.2)	9.9 (0.3)	3.7 (0.3)
Individuals with abuse only at Wave 1									
Nonabst. remission to no AUD	0.98 (0.06)	0.70 (0.04)	-0.28 (0.06)	52.5 (3.5)	33.9 (2.9)	-18.6 (3.2)	7.6 (0.2)	6.2 (0.2)	-1.4 (0.2)
Abst. remission to no AUD	1.82 (0.64)	0.00 (0.00)	-1.82 (0.64)	95.8 (21.9)	0.0 (0.0)	-95.8 (21.9)	8.8 (1.1)	0.0 (0.0)	-8.8 (1.1)
Remained abuse only	1.30 (0.08)	1.11 (0.07)	-0.19 (0.07)	69.9 (5.2)	62.3 (5.2)	-7.6 (5.3)	9.3 (0.3)	9.5 (0.3)	0.2 (0.3)
Developed dependence[a]	1.56 (0.16)	2.32 (0.24)	0.76 (0.19)	84.9 (8.3)	127.1 (42.2)	42.2 (9.5)	10.2 (0.5)	12.6 (0.5)	2.4 (0.5)
Individuals with dependence[a] at Wave 1									
Nonabst. remission to no AUD	2.57 (0.26)	0.58 (0.07)	-1.99 (0.25)	118.3 (9.6)	38.7 (5.7)	-79.6 (9.2)	11.2 (0.6)	6.4 (0.4)	-4.8 (0.6)
Abst. remission to no AUD	2.65 (0.37)	0.00 (0.00)	-2.65 (0.37)	152.7 (18.4)	0.0 (0.0)	-152.7 (18.4)	12.2 (1.0)	0.0 (0.0)	-12.2 (1.0)
Partially remitted	2.46 (0.19)	1.26 (0.11)	-1.20 (0.21)	119.4 (6.7)	77.2 (5.7)	-42.2 (7.5)	12.0 (0.4)	9.6 (0.3)	-2.3 (0.4)
Remained dependent[a]	3.44 (0.21)	3.24 (0.21)	-0.21 (0.23)	152.9 (7.4)	149.5 (7.6)	-3.4 (8.9)	13.2 (0.5)	13.9 (0.4)	0.6 (0.3)

Notes: Figures in parentheses are SEs of estimates. AUD = alcohol-use disorder; nonabst. = nonabstinent; abst. = abstinent. [a]With or without abuse.

comorbidity, whereas the data in Table 2 are unadjusted. Second, the model parameters reflect not the absolute value of the change but rather the *differential* change in consumption for the AUD transition category in question relative to the reference category. Despite these differences, the pattern of these model parameters closely replicates the results of Table 2, and the adjusted differences in change among categories are, in general, somewhat greater (more strongly positive or negative) than the unadjusted changes.

Table 4 contains parameters from models that included significant ($p < .005$) interactions, denoting factors that moderated the associations between transitions in AUD status and changes in consumption. With respect to ADV of ethanol intake among individuals who did not have an AUD at baseline (top third, left data column), a positive interaction between baseline volume and developing abuse only indicated that this transition was associated with a *greater* increase in volume among those who had higher ADV at baseline. In addition, the increase in volume associated with developing dependence was doubled among men ($\Delta = 0.57 + 0.61$ for men vs $\Delta = 0.57$ for women) and more than doubled among individuals with PD other than borderline or antisocial disorders. The increases in frequency of risk drinking days (top third, middle data column) were not modified by any significant interactions; however, the increases in maximum quantity of drinks associated with developing abuse only and dependence (top third, right data column) were greater for men than women. In addition, the increases associated with developing abuse only were reduced among individuals who had attended or completed college and decreased with advancing age.

When individuals with abuse only at Wave 1 were examined (middle third), there were negative interactions between all three baseline consumption measures and changes in those measures for individuals who remitted to no AUD (abstinent remission only). That is, as would necessarily be the case, there were greater reductions among those who had the highest initial levels of consumption. In addition, the increase in ADV in association with progressing from abuse to dependence was more than three times as great for men as women (the increase among women in fact fell just short of statistical significance), and the increase in frequency of risk drinking associated with developing dependence was greater among individuals with another substance-use disorder. The reduction in maximum quantity of drinks in association with abstinent remission from abuse was less with advancing age (a positive interaction offsetting the negative main effect of the transition) and increased among men compared with women (a negative interaction adding to the negative main effect).

Among individuals with alcohol dependence at Wave 1 (lower third), the reduction in all measures of consumption again were greatest among those with the highest initial consumption levels, although these interactions fell short of significance for some consumption measures among individuals with nonabstinent and partial remission. Among those with nonabstinent remission from dependence, the reduction in frequency of risk drinking was greater among persons with borderline PD, and among those with abstinent remission, the reduction in maximum quantity was greater for men than for women.

Because of the possibility that interactions might simply indicate that the combined effects of multiple risk factors for

TABLE 3. Selected parameters from main effects multiple linear regression models representing net change in consumption measures between Wave 1 and Wave 2 for various transitions in AUD status: Past-year drinkers at Wave 1

	Average daily volume of ethanol intake, oz		Frequency of risk drinking		Maximum quantity of drinks consumed in a day	
AUD transition	β (SE)	*p*	β (SE)	*p*	β (SE)	*p*
Individuals without an AUD at Wave 1						
Remained without AUD and continued drinking	0.00 (0.00)	(ref.)	0.0 (0.0)	(ref.)	0.0 (0.0)	(ref.)
Remained without AUD and stopped drinking	-0.26 (0.01)	.000	-11.3 (0.6)	.000	-3.3 (0.1)	.000
Developed abuse only	0.46 (0.04)	.000	35.0 (3.1)	.000	3.0 (0.2)	.000
Developed dependence	1.22 (0.10)	.000	75.6 (5.9)	.000	4.4 (0.2)	.000
Model goodness of fit	Adj. $\chi^2 = 686.5$, $p < .001$, $R^2 = .260$		Adj. $\chi^2 = 2{,}060.2$, $p < .001$, $R^2 = .343$		Adj. $\chi^2 = 3{,}158.8$, $p < .001$, $R^2 = .392$	
Individuals with abuse only at Wave 1						
Nonabst. remission to no AUD	-0.29 (0.07)	.000	-23.9 (5.2)	.000	-2.4 (0.2)	.000
Abst. remission to no AUD	-1.29 (0.22)	.000	-70.5 (8.7)	.000	-9.2 (0.6)	.000
Remained abuse only	0.00 (0.00)	(ref.)	0.0 (0.0)	(ref.)	0.0 (0.0)	(ref.)
Developed dependence	1.13 (0.19)	.000	60.9 (10.0)	.000	2.6 (0.5)	.000
Model goodness of fit	Adj. $\chi^2 = 166.1$, $p < .001$, $R^2 = .456$		Adj. $\chi^2 = 550.6$, $p < .001$, $R^2 = .466$		Adj. $\chi^2 = 443.0$, $p < .001$, $R^2 = .473$	
Individuals with dependence at Wave 1						
Nonabst. remission to no AUD	-2.14 (0.21)	.000	-91.3 (9.5)	.000	-6.6 (0.5)	.000
Abst. remission to no AUD	-2.97 (0.23)	.000	-143.2 (9.5)	.000	-13.5 (0.5)	.000
Partially remitted	-1.46 (0.23)	.000	-52.3 (9.9)	.000	-3.7 (0.4)	.000
Remained dependent	0.00 (0.00)	(ref.)	0.0 (0.0)	(ref.)	0.0 (0.0)	(ref.)
Model goodness of fit	Adj. $\chi^2 = 604.5$, $p < .001$, $R^2 = .599$		Adj. $\chi^2 = 624.1$, $p < .001$, $R^2 = .513$		Adj. $\chi^2 = 838.3$, $p < .001$, $R^2 = .583$	

Notes: Models adjusted for baseline value of the consumption of the alcohol measure in question, age, race/ethnicity, marital status, whether attended/completed college, family history of alcoholism, age at first drink, any past-year mood disorder, any past-year anxiety disorder, antisocial personality disorder (PD), borderline PD, other PD, and other past-year substance-use disorder. AUD = alcohol-use disorder; nonabst. = nonabstinent; abst. = abstinent.

change in consumption are multiplicative rather than additive in nature (additivity being implicit in the use of linear regression models), all interactions were tested in multiplicative models that were created by using logged values of the consumption outcomes. Constants were added to the change measures to remove negative and zero values and allow log transformation. Eighteen of the 21 significant interactions remained highly significant (p = .000-.008), but 3 were no longer significant. These included the interactions of male gender with abstinent remission from both abuse and dependence for the outcome of change in maximum quantity of drinks and the interaction of borderline PD and nonabstinent remission for the outcome of change in frequency of risk drinking.

Discussion

The changes in consumption documented in this study are consistent with our initial expectations in that even after adjustment for a wide range of sociodemographic characteristics and comorbid substance-use and mental disorders: (1) there were positive changes associated with developing an AUD; (2) there were negative changes associated with remission of an AUD, even among individuals who continued to drink; (3) the increase associated with developing dependence was larger than that associated with developing abuse only; (4) the decrease associated with remission from dependence was greater than that associated with remission from abuse only; and (5) the decrease associated with full remission from dependence was greater than that associated with partial remission. To the extent that heavy drinking is a marker of AUD severity, these findings thus support the argument that abuse is a milder disorder than dependence, even though some of its symptoms have been shown to tap into the more severe range of the underlying latent construct of AUD as a unidimensional entity (Saha et al., 2006, 2007). In fact, the strong differences in sociodemographic and baseline clinical characteristics of abusers who achieved abstinent as compared with nonabstinent remission—characteristics that more closely matched those of individuals with dependence than those of other abusers—may reflect a greater prevalence of the more severe abuse criteria among the latter and support the findings of Saha et al. (2006, 2007), which indicated that abuse is quite heterogeneous in terms of severity. These differences also support the argument that the more severe abuse criteria (e.g., continued drinking despite social and interpersonal problems caused by drinking) might more appropriately be considered as criteria for dependence. However, definitive recommendations require more research into differential patterns of criterion endorsement.

It is noteworthy that the Wave 2 consumption levels of individuals who had developed AUD over the preceding 3 years were still considerably lower than the baseline consumption levels of individuals already positive for AUD. For example, the average Wave 2 ADV of ethanol intake for persons who developed dependence was 1.84 oz, the equivalent

TABLE 4. Selected parameters from multiple linear regression models representing net change in consumption measures between Wave 1 and Wave 2 for various transitions in AUD status: Past-year drinkers at Wave 1

	Average daily volume (ADV) of ethanol intake, oz		Frequency of risk drinking		Maximum quantity of drinks consumed in a day	
AUD transition	β (SE)	*p*	β (SE)	*p*	β (SE)	*p*
Individuals without an AUD at Wave 1						
Remained without AUD and continued drinking	0.00 (0.00)	(ref.)	0.0 (0.0)	(ref.)	0.0 (0.0)	(ref.)
Remained without AUD and stopped drinking	-0.26 (0.01)	.000	-11.3 (0.6)	.000	-3.3 (0.1)	.000
Developed abuse only	0.29 (0.05)	.000	35.0 (3.1)	.000	5.1 (0.5)	.000
Developed dependence	0.57 (0.15)	.000	75.6 (5.0)	.000	3.4 (0.3)	.000
Developed Abuse Only × ADV Ethanol Intake	0.27 (0.08)	.001	–	–	–	–
Developed Abuse Only × Age	–	–	–	–	-0.1 (0.0)	.000
Developed Abuse Only × Male	–	–	–	–	1.8 (0.3)	.000
Developed Abuse Only × College	–	–	–	–	-1.1 (0.3)	.001
Developed Dependence × Male	0.61 (0.19)	.003	–	–	1.7 (0.4)	.000
Developed Dependence × Other PD	0.81 (0.25)	.002	–	–	–	–
Model goodness of fit	Adj. $\chi^2 = 541.6$, $p < .001$, $R^2 = .275$		Adj. $\chi^2 = 2{,}060.2$, $p < .001$, $R^2 = .343$		Adj. $\chi^2 = 3{,}153.5$, $p < .001$, $R^2 = .402$	
Individuals with abuse only at Wave 1						
Nonabst. remission to no AUD	-0.27 (0.07)	.000	-23.2 (5.1)	.000	-2.3 (0.3)	.000
Abst. remission to no AUD	-0.57 (0.12)	.000	-37.0 (5.6)	.000	-6.2 (0.7)	.000
Remained abuse only	0.00 (0.00)	(ref.)	0.0 (0.0)	(ref.)	0.0 (0.0)	(ref.)
Developed dependence	0.37 (0.19)	.056	39.8 (11.9)	.001	2.6 (0.5)	.000
Abst. Remission × ADV Ethanol Intake	-0.43 (.08)	.000	–	–	–	–
Abst. Remission × Frequency of Risk Drinking	–	–	0.3 (0.1)	.000	–	–
Abst. Remission × Maximum Quantity of Drinks	–	–	–	–	-0.4 (0.1)	.000
Abst. Remission × Age	–	–	–	–	0.1 (0.0)	.001
Abst. Remission × Male	–	–	–	–	-2.0 (0.4)	.000
Developed Dependence × Male	0.96 (0.32)	.004	–	–	–	–
Developed Dependence × Other Substance Dx	–	–	62.4 (21.1)	.004	–	–
Model goodness of fit	Adj. $\chi^2 = 175.1$, $p < .001$, $R^2 = .488$		Adj. $\chi^2 = 535.7$, $p < .001$, $R^2 = .484$		Adj. $\chi^2 = 461.9$, $p < .001$, $R^2 = .490$	
Individuals with dependence at Wave 1						
Nonabst. remission to no AUD	-0.85 (0.23)	.001	-84.1 (10.1)	.000	-2.7 (0.7)	.001
Abst. remission to no AUD	-1.33 (0.23)	.000	-95.4 (9.9)	.000	-5.5 (0.7)	.000
Partially remitted	-0.41 (0.23)	.082	-51.1 (9.9)	.000	-1.0 (0.7)	.200
Individuals without an AUD at Wave 1						
Nonabst. Remission × ADV Ethanol Intake	-0.43 (0.08)	.000	–	–	–	–
Nonabst. Remission × Borderline PD	–	–	-54.1 (17.9)	.004	–	–
Nonabst. Remission × Maximum Quantity of Drinks	–	–	–	–	-0.3 (0.1)	.000
Abst. Remission × ADV Ethanol Intake	-0.51 (0.07)	.000	–	–	–	–
Abst. Remission × Frequency of Risk Drinking	–	–	-0.3 (0.1)	.000	–	–
Abst. Remission × Maximum Quantity of Drinks	–	–	–	–	-0.5 (0.1)	.000
Abst. Remission × Male	–	–	–	–	-2.3 (0.4)	.000
Partially Remitted × ADV Ethanol Intake	-0.39 (0.09)	.000	–	–	–	–
Partially Remitted × Maximum Quantity of Drinks	–	–	–	–	-0.2 (0.1)	.000
Model goodness of fit	Adj. $\chi^2 = 595.2$, $p < .001$, $R^2 = .633$		Adj. $\chi^2 = 624.7$, $p < .001$, $R^2 = .520$		Adj. $\chi^2 = 840.5$, $p < .001$, $R^2 = .608$	

Notes: Models adjusted for baseline value of the consumption of the alcohol measure in question, age, race/ethnicity, marital status, whether attended/completed college, family history of alcoholism, age at first drink, any past-year mood disorder, any past-year anxiety disorder, antisocial personality disorder (PD), borderline PD, other PD, and other past-year substance-use disorder. AUD = alcohol-use disorder; nonabst. = nonabstinent; abst. = abstinent; dx = diagnosis.

of a relatively modest three standard drinks per day. Individuals who were dependent at baseline and remained dependent had a mean baseline ADV of 3.44 oz, almost twice as high. These data illustrate that the severity of AUD, as indicated by alcohol consumption levels, continues to increase over time and that the 3-year changes reported in this study represent only a portion of the increase to be expected in the progression of these disorders. They also illustrate the importance of early intervention, when consumption levels of individuals with AUD are still relatively low and may not yet have resulted in irreversible changes in their neurobiological responses to alcohol (e.g., in terms of craving and withdrawal). Finally, they suggest that additional research, using general population data over a longer follow-up period, is needed to determine when levels of consumption begins to stabilize in the natural course of AUD.

The changes in consumption associated with AUD transitions were modified by a number of significant interactions. The increase in volume of consumption associated with the onset of abuse was larger among individuals with higher

baseline volumes of consumption, suggesting that the risk of developing abuse may be more strongly correlated with proportional than with absolute level of increase—possibly because of higher tolerance or lower response to the effects of alcohol among those already habituated to greater levels of intake. Male gender was associated with greater consumption increases in association with developing both abuse and dependence, including progressing from abuse to dependence, and these interactions were independent of both the initial levels of consumption and any interactions of AUD onset with baseline consumption. Notably, these interactions were apparent for changes in volume of consumption and/or maximum quantity of drinks consumed but not for frequency of risk drinking. This suggests that these gender interactions may reflect weaker physiological response to the effects of large doses of ethanol among men, consistent with their greater total body water and lower blood alcohol concentration in response to a given level of intake. Following this same logic, the reduced increases in maximum quantity among older drinkers in association with onset of abuse might reflect their lower total body water and higher blood alcohol concentration.

The smaller increase in maximum quantity of drinks associated with the development of AUD among individuals who attended college might reflect differential beverage preferences. More highly educated drinkers have been shown to drink a higher proportion of wine (McCann et al., 2003; Paschall and Lipton, 2005) than less educated drinkers, and the amount of ethanol consumed in a typical glass of wine is greater than that in a typical 12-oz bottle or can of beer (Kerr et al., 2005). Thus, college-educated drinkers may be increasing their maximum ethanol intake to the same degree as other drinkers but doing so with fewer drinks.

The augmented increase in ADV among individuals with PD other than borderline and antisocial disorders in association with the transition from no AUD to dependence requires further investigation. Within this group, increases in ADV were greatest among individuals with paranoid and schizotypal PDs, neither of which has demonstrated unusually strong rates of co-occurrence with AUD (Grant et al., 2005; Pulay et al., in press). However, the relationships of these PDs to drinking volume and pattern have not been measured in the general population.

The interactions modifying remission from AUD are more predictable and easily understood. The greater reductions among individuals with higher initial levels of intake simply reflect the greater reduction required for these individuals to achieve abstinence or the moderate levels of consumption necessary for nonabstinent recovery.

This study has several methodological strengths that increase confidence in the validity of its findings. Because of the longitudinal design of the NESARC, both transitions in AUD status and changes in alcohol consumption were measured directly by comparison of past-year data at two points in time; thus they were not subject to the level of recall error that might bias retrospective reports of change. In addition, these past-year measures, and those used as model covariates, have demonstrated high levels of reliability in test-retest and validity studies. These strengths notwithstanding, there are limitations to the analysis. First, the length of the follow-up period may not have been long enough to capture the full extent of increase or reduction in alcohol consumption associated with the course of AUD. Second, the study did not consider the potentially confounding effects of changes over the follow-up period in the model covariates. Some of these changes, particularly transitions in marital status, have been strongly correlated with drinking trajectories in prior research (Arnett, 1998; Bachman et al., 1997; Mudar et al., 2002; Prescott and Kendler, 2001). Finally, although the follow-up rate was high among those remaining eligible for interview, our inability to study changes in consumption among individuals who died or became incapacitated over the follow-up interval may have led to understatement of the magnitude of the changes or their associations with the course of AUD.

Despite these limitations, the results of this study have important implications for the prevention and treatment of AUD. They demonstrate that substantial reductions in consumption occur with the remission of AUD, even among continuing drinkers and those in partial remission. They also illustrate the large increases in consumption associated with developing an AUD, to levels that in prior research have been shown to increase the risk of numerous other adverse health outcomes. Finally, they indicate that these changes occur gradually enough to provide a window of opportunity for intervention before consumption stabilizes. Future research with the NESARC data will examine whether these changes in consumption were associated with changes in health-related quality of life in relation to transitions in AUD status.

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