Evidence for a closing gender gap in alcohol use, abuse, and dependence in the United States population[☆]

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Abstract

Background: Descriptively, male–female differences in alcohol consumption and alcohol use disorders appear to have decreased in birth cohorts reaching adulthood since the 1970s compared to earlier birth cohorts. However, such birth cohort effects on gender differences have never been statistically tested in nationally representative data. The aim of this study was to test the hypothesis that gender differences in alcohol consumption, abuse, and dependence are decreasing over time.
Methods: Face-to-face survey conducted in the 2001–2002 National Epidemiologic Survey on Alcohol and Related Conditions among those aged <90 ($N = 42,693$). Birth cohort was divided into four categories: 1913–1932, 1933–1949, 1950–1967, 1968–1984. Outcomes included lifetime largest drinks, frequent binge drinking, DSM-IV defined alcohol abuse, and alcohol dependence, measured with the Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS-IV).
Findings: Birth cohort and gender interacted significantly in predicting lifetime largest drinks ($F = 27.6$, [d.f. = 3], $p < 0.0001$), frequent binge drinking ($F = 40.0$, [d.f. = 3], $p < 0.0001$), alcohol abuse ($F = 62.0$, [d.f. = 3], $p < 0.0001$) and alcohol dependence ($F = 15.3$, [d.f. = 3], $p < 0.0001$). Cohort-specific ORs indicated monotonic decreases in the gender ratio in more recent birth cohorts for all outcomes.
Conclusion: These results suggest that gender differences in the prevalence of all four outcomes are decreasing in younger age cohorts. While these changes are consistent with a cohort effect, the possibility of age and period effects cannot be ruled out but suggest important avenues for more specific hypothesis testing. Further, women in younger cohorts may be in need of new targeted prevention and intervention efforts.
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1. Introduction

Men have been consistently shown to drink more alcohol and have a higher likelihood of alcohol use disorders than women in the United States (Grant, 1997; Helzer and Pryzbeck, 1988; Warner et al., 1995) and internationally (Wilsnack et al., 2000). In the U.S., "traditional" gender roles have changed since the baby boom birth cohort reached adulthood in the 1970s. Since

then, the proportion of women working outside the home has increased, while the proportion of women having children has decreased (Thronton and Freedman, 1983; Echols, 1989; Rosen, 2000). Consistent with these changes in "traditional" behaviors, differences in gender-based drinking norms have diminished since the 1970s (Greenfield and Room, 1997). Diminished gender differences in the prevalence of alcohol use disorders as a function of birth cohort would be consistent with these other large social changes, but such information is lacking. A more comprehensive understanding of the relationship of birth cohort to alcohol disorders has important implications for our knowledge of etiology of these prevalent and often disabling disorders (Hasin et al., 2007). Evidence that gender differences in alcohol consumption and alcohol use disorders have changed across successive U.S. birth cohorts would indicate the need to investigate

how such gender differences in alcohol use disorders occurred within larger population-level gender role shifts. Moreover, the identification of secular trends in alcohol-related outcomes has important implications for researchers investigating risk factors at other levels of organization. For instance, the magnitude of association between a genetic risk factor and alcohol dependence could vary across populations as a function of the trends in alcohol disorder prevalence across time.

To understand birth cohort effects on gender differences in the likelihood of alcohol use disorders, overall birth cohort shifts in prevalence must be understood. U.S. per capita alcohol consumption began an upward trend at the repeal of Prohibition in 1933 that peaked in the early 1980s (Lakins et al., 2005). While National Alcohol Survey data indicate that cohort effects may vary by beverage type (Kerr et al., 2004), most large-scale cross-sectional studies have indicated an increase in the prevalence of alcohol use and alcohol use disorders by birth cohort. The National Household Surveys on Drug Abuse indicated a cohort effect for any alcohol use, with individuals born in cohorts after 1950 particularly more likely to use alcohol than those born previously (Johnson and Gerstein, 1998). The National Longitudinal Alcohol Epidemiologic Survey (NLAES) and the National Comorbidity Study (NCS) both suggest that birth cohorts after World War II evidence heightened risk for alcohol use (Grant, 1997) and lifetime alcohol disorders (Anthony et al., 1994; Warner et al., 1995; Grant, 1996, 1997) compared with earlier birth cohorts. Although cross-sectional studies cannot empirically establish the existence of a cohort effect as opposed to a period (factors impacting risk among all members of a population at a given point in time) or age effect (factors impacting risk at specific ages), these shifts in the prevalence of alcohol disorders in the population are consistent with a cohort effect because respondents within birth cohorts are similar to each other with respect to alcohol consumption and alcohol use disorders across the lifecourse, as opposed to all cohorts being similar at a single point in time (a period effect) or all individuals across birth cohorts having the same risk at a given age (age effect).

While studies of adolescent alcohol use have consistently shown a convergence in rates of alcohol use initiation in younger birth cohorts (Engs and Hanson, 1990; Johnson and Gerstein, 1998; Johnston et al., 2004, 2005; Schulenberg et al., 2001), studies of adults are less consistent. In The Netherlands, convergence was suggested by linear increases in average weekly alcohol consumption during a time when consumption among men decreased (Saelan et al., 1992; Neve et al., 1993), although this effect was not seen for weekly heavy (≥6 drinks) drinking (Neve et al., 1996). Converging rates of mean weekly drinking due mainly to increases among women by cohort were found in a survey in Finland, but not in Germany, Switzerland, or the Netherlands (Bloomfield et al., 2001). In New Zealand, gender differences in alcohol consumption decreased from 1995 to 2000, due mainly to increases among women in typical quantity/frequency (e.g., consuming more than 20 l of alcohol per annum and drinking enough to feel drunk once per week (McPherson et al., 2004)). In surveys of U.S. women from 1981 to 2001, complete abstention and heavy episodic drinking (≥6 drinks/day) both decreased over time (Wilsnack et al.,

2006), but these surveys did not include males, so direct gender comparisons could not be made. These studies of alcohol consumption differed widely in their measures and study design, and thus their lack of consensus on gender differences in alcohol consumption is difficult to interpret.

In contrast to the literature on alcohol consumption, studies of DSM-IV alcohol use disorders have been more consistent, although most evidence comes from family genetic studies, which are not representative samples. Among relatives of 300 Caucasian alcohol dependent individuals (Reich et al., 1988), secular trends in the consumption of alcohol contributed to an increased prevalence of alcohol dependence over time, and sex differences in alcohol consumption and problem alcohol behaviors decreased. In a Midwestern Caucasian twin study, individuals born after 1951 were at higher risk for DSM-IV alcohol dependence than those born earlier, a risk that was more pronounced for women (Holdcraft and Iacono, 2002). The Collaborative Study on the Genetics of Alcoholism (COGA) noted a decrease in the gender gap in the prevalence of DSM-IV defined alcohol dependence among those born in younger cohorts compared to earlier-born cohorts, but did not directly test for the presence of effect modification (Rice et al., 2003). These studies were all limited in terms of generalizeability. In contrast, the NLAES, a 1991–1992 U.S. national survey of over 42,000 adults, provided a substantial advantage in terms of sample size and representativeness. Descriptively, male and female prevalences by birth cohort suggested that gender differences in alcohol use disorders had decreased in younger cohorts (Grant, 1997). However, this apparent difference was never directly tested. These studies suggest birth cohort effects on the gender ratio in the prevalence of DSM-IV alcohol use disorders, and point to the need for direct testing in a recent, large, representative sample.

In sum, there is evidence to suggest that non-parallel male and female trends in recent birth cohorts have decreased the gender gap in alcohol consumption. However, the existence of such a phenomenon has never been directly tested in a representative sample. Accordingly, the present study uses such a sample, the U.S. National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), to investigate whether there is evidence consistent with a birth cohort effect on gender differences in the prevalence of DSM-IV alcohol abuse and dependence. We also examined such effects for drinking and frequent binge drinking to determine if the effects were consistent across different alcohol indicators, and to provide links to earlier studies. The NESARC sample is uniquely situated to address these questions. The sample size is large enough to detect interaction effects, the state-of-the-art diagnostic instrument provides reliable, valid and complete diagnostic information on both alcohol dependence and abuse, and the sample includes individuals born more recently than previous large-scale studies.

2. Methods

2.1. Sample

This sample consists of participants in the 2001–2002 the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), a nationally

representative United States survey of 43,093 civilian non-institutionalized participants aged 18 and older, sampled cross-sectionally and interviewed in person. Details of the sampling frame are described elsewhere (Grant et al., 2003, 2004). The National Institute on Alcohol Abuse and Alcoholism (NIAAA) sponsored the study and supervised the fieldwork, conducted by the U.S. Bureau of the Census. There was oversampling of young adults, Hispanics, and African-Americans, and rates are weighted to the 2000 decennial census in terms of age, race, sex, and ethnicity and are further weighted to adjust for sampling probabilities. The study achieved an overall response rate of 81%. The research protocol, including informed consent procedures, received full ethical review and approval from the U.S. Census Bureau and U.S. Office of Management and Budget. By sex, 47.9% of the sample was male. The youngest age group in the NESARC, 18–29, composed 21.8% of the total sample; 30.9% were 30–44, 31.1% were 45–64, and 16.2% were 65 or older. White subjects comprised 70.9% of the sample, African-Americans, 11.1%, Hispanics, 11.6%, Asian or Pacific Islander, 4.4%, and American Indians and Alaska Natives, 2.1%.

2.2. Interviewers, training, and field quality control

Interviewing was conducted by 1800 professional interviewers from the Census Bureau, using computer-assisted software with built-in skip, logic, and consistency checks. All interviewers had experience with other national health-related surveys with an average of 5 years of experience, and were further trained for 10 days under the direction of NIAAA. Regional supervisors re-contacted a random 10% of all respondents to verify the interviewer and for quality control purposes. In addition, a randomly selected subset of respondents was re-interviewed with one to three complete sections of the AUDADIS-IV. This served as a test–retest reliability study of NESARC measures (Grant et al., 2003). In the few cases when accuracy was uncertain, the data were discarded and a supervising interviewer repeated the interview.

2.3. Measures

The Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS-IV (Grant et al., 2003)), a state-of-the-art structured diagnostic interview, was administered to the NESARC participants. This instrument was specifically designed for experienced lay interviewers and was developed to advance measurement of substance use and mental disorders in large-scale surveys. The AUDADIS-IV used an extensive list of over 40 questions to assess alcohol abuse and dependence. Diagnoses were indicated according to the DSM-IV (American Psychiatric Association, 1994); at least three of seven criteria for alcohol dependence and at least one of four criteria for alcohol abuse. Withdrawal syndromes were also assessed according to DSM-IV criteria; the presence of at least two symptoms out of eight is necessary for a withdrawal diagnosis. Time frames for diagnosis included the previous 12-month period and prior to the previous 12-month period. Diagnoses made in either time frame were combined for a "lifetime" diagnosis of alcohol abuse and a "lifetime" diagnosis of alcohol dependence. Consistent with DSM-IV, those diagnosed with dependence were not additionally diagnosed with abuse. Respondents were also asked a series of questions about consumption behavior during the period of heaviest drinking. For these analyses, two consumption-related measures were used: the largest number of drinks ever consumed in one sitting during period of heaviest drinking, and frequent binge drinking, defined as drinking 5+ drinks at least one per week during period of heaviest drinking. Both measures have been validated as accurately capturing problem alcohol use (Wechsler et al., 1994; Hasin et al., 1999; Dawson, 2003). A test retest reliability study indicated good to excellent reliability for largest drinks variables (ICC = 0.70–0.84), and fair to good reliability for 5+ drinks variables (ICC = 0.47–0.69) (Grant et al., 2003).

The reliability and validity of alcohol dependence diagnosis has been extensively documented in the U.S. and abroad. The reliability of the alcohol dependence diagnosis has achieved a minimum kappa of 0.74 (Canino et al., 1999; Grant et al., 1995, 2003; Chatterji et al., 1997). The alcohol abuse diagnosis has been shown to have adequate reliability when measured independently of the alcohol dependence diagnosis (Chatterji et al., 1997; Canino et al., 1999). The validity of these diagnoses has been documented in numerous studies including the World Health Organization/National Institutes of Health and

Reliability and Validity Study (Chatterji et al., 1997; Cottler et al., 1997; Pull et al., 1997; Ustun et al., 1997; Hasin et al., 1997b; Vrasti et al., 1998; Canino et al., 1999) and others (Grant et al., 2003, 1995; Hasin et al., 1997a, 1997c; Hasin and Paykin, 1999). Further, the symptom items have been validated using clinical reappraisals conducted by psychiatrists (Canino et al., 1999); good validity of the alcohol diagnoses was documented ($K = 0.60, 0.76$). Validity coefficients were similar for alcohol disorders in the WMH-CIDI ($K = 0.56, 0.70$, Kessler et al., 2005) and higher than the DIS (≤ 0.50, Anthony et al., 1985).

Birth cohort was divided into four categories for the analysis based on previous evidence and statistical examination of homogeneity of risk within category. Respondents older than 90 were removed from the analyses ($N = 400$) as lifetime responses would be most affected by recall bias, leaving a total N of 42,693. Cohort 1 was born between 1913 and 1932, Cohort 2 between 1933 and 1949, Cohort 3 between 1950 and 1967, and Cohort 4 between 1968 and 1984. As previously noted, epidemiologic evidence has indicated that the cohorts born after approximately 1950 are at heightened risk for alcohol disorders; thus the category definition between Cohort 2 and Cohort 3 was set at 1950. Further, previous studies have indicated that the per capita alcohol consumption in the U.S. peaked in the mid 1980s, and thus Cohort 3 and Cohort 4 were divided at 1968 to capture those who entered the period of risk for alcohol disorders after the U.S. alcohol consumption peak (Lakins et al., 2005). Note that neither minor variations in the category definitions nor variations in number of cohort groups meaningfully changed the results presented below.

2.4. Statistical analysis

To adjust for the complex sample characteristics of the NESARC, all analyses were conducted using SUDAAN (Research Triangle Institute, 2004) to obtain accurate standard errors. The mean number of lifetime largest drinks was calculated with univariate statistics and subset by birth cohort and sex. While the distribution of largest drinks was right-skewed, model fit statistics indicated that linear regression coefficients were robust to the skewness of the distribution and thus are reported here. The lifetime prevalence of frequent binge drinking, alcohol abuse, and alcohol dependence by birth cohort and sex was calculated with cross-tabulations. Odds ratios (ORs) and 95% confidence intervals were derived from logistic regressions to assess the effect of gender on the likelihood of aforementioned alcohol outcomes by birth cohort; interactions terms were included in the models to assess the possible interaction of birth cohort and gender. Control covariates were income, race, education, urbanicity and marital status; both cohort category and gender were adjusted for these demographic variables. Wald F-tests were used to estimate the statistical significance of the inclusion of interaction terms in the model, and cohort-specific odds ratio and confidence interval estimates were calculated using the beta estimate for the interaction and the beta estimate for gender in each model. Parameter estimates for cohort were dummy-coded, allowing the effects across levels of gender and cohort to be non-linear.

3. Results

3.1. Mean largest drinks

Table 1 indicates the mean lifetime largest number of drinks consumed by birth cohort and gender. In the total sample, men consumed 6.94 mean drinks (inter-quartile range 2–10) compared to 2.98 mean drinks (inter-quartile range 0–4) for women. This represented a 2.33:1 male/female ratio. When dividing by cohort, there are increases in mean drinks consumed with each successively younger cohort in both men and women. Additionally, there is a slight reduction in the male/female ratio. In Cohort 1, born 1913–1932, the male/female ratio was 2.91:1. By Cohort 4, born 1968–1984, there is a 2.10:1 male to female ratio. Birth cohort and sex interacted significantly in predicting lifetime largest drinks ($F = 27.6$, [d.f. = 3], $p < 0.0001$).

Table 1
Mean largest number of drinks by birth cohort and gender

	Birth cohort	N (total 42,693)	Mean (S.E.)			Male to female ratio[a]
			Total (42,693)	Men (18,413)	Women (24,280)	
			4.87 (0.1)	6.94 (0.1)	2.98 (0.1)	2.33:1
Cohort 1	1913–1932	6,153	2.21 (0.1)	3.58 (0.1)	1.23 (0.0)	2.91:1
Cohort 2	1933–1949	9,149	3.98 (0.1)	5.89 (0.2)	2.27 (0.1)	2.59:1
Cohort 3	1950–1967	15,434	5.51 (0.1)	7.65 (0.2)	3.45 (0.1)	2.22:1
Cohort 4	1968–1984	11,957	5.92 (0.1)	8.05 (0.2)	3.83 (0.1)	2.10:1

[a] Birth cohort and sex significantly interacted in a linear regression model ($F = 27.6$, [d.f. = 3], $p < 0.0001$).

Table 2
Prevalence of lifetime frequent binge drinking (5+ drinks once per week or more during period of heaviest use) by birth cohort and gender

	Birth cohort	N (total 42,693)	% (S.E.)			Adjusted odds ratio (men compared to women, within cohort)[a]
			Total (42,693)	Men (18,413)	Women (24,280)	OR (95% C.I.)
			22.2 (0.5)	33.5 (0.8)	11.9 (0.4)	3.82 (3.57–4.10)
Cohort 1	1913–1932	6,153	8.9 (0.5)	18.0 (0.9)	2.3 (0.3)	10.55 (7.88–14.12)
Cohort 2	1933–1949	9,149	19.4 (0.6)	32.1 (1.0)	7.8 (0.5)	6.50 (5.62–7.50)
Cohort 3	1950–1967	15,434	26.3 (0.8)	38.2 (1.1)	14.8 (0.6)	3.88 (3.50–4.32)
Cohort 4	1968–1984	11,957	25.3 (0.8)	34.5 (1.1)	16.2 (0.7)	2.66 (2.36–3.00)

[a] Birth cohort and sex significantly interacted in a logistic regression model ($F = 40.0$, [d.f. = 3], $p < 0.0001$); cohort-specific odds ratios are derived from this logistic regression model.

3.2. Frequent binge drinking

In Table 2, birth cohort and gender stratify the prevalence of frequent binge drinking (drinking 5+ drinks once per week or more during period of heaviest drinking). The prevalence of lifetime frequent binge drinking among men increased by cohort from 18.0% among those in Cohort 1 to 38.2% among those in Cohort 3, then decreased to 34.5% in the youngest cohort, Cohort 4. Among women, the prevalence of frequent binge drinking increased monotonically, from 2.3% among those in Cohort 1 to 16.2% among those in Cohort 4. After inclusion of a statistically significant interaction term ($F = 40.0$, [d.f. = 3], $p < 0.0001$), cohort-specific odds ratios (shown in Table 2) comparing men to women were calculated using the beta coefficients from the regression model. For instance, the cohort-specific beta estimate for the effect of gender when the interaction term is in the model (0.98) indicates the odds of binge drinking among men compared to women at what is the reference group in this

model, the youngest cohort level (Cohort 4). Specifically, among those in Cohort 4, men are 2.66 times (Table 2) more likely to evidence binge drinking compared with women. To estimate the odds ratio for Cohort 3, the beta estimate for the interaction of gender and cohort at Cohort 3 (0.38) is added to the beta estimate for gender when the interaction term is in the model (0.98) and then exponentiated to get an odds ratio estimate. As shown in Table 2, this estimate indicates that among those in Cohort 3, men are 3.88 times more likely to evidence binge drinking compared with women.

In sum, these estimates indicate consistent monotonic decreases in the odds of frequent binge drinking among males compared to among females by each successively younger cohort: the odds ratio in Cohort 1 was 10.55 (95% C.I. 7.88–14.12) compared to 2.66 (95% C.I. 2.36–3.00) in Cohort 4, the youngest cohort. This indicated substantial effect modification by birth cohort of the odds of frequent binge drinking among men compared to women.

Table 3
Prevalence of lifetime DSM-IV alcohol dependence by birth cohort and gender

	Birth cohort	N (total 42,693)	% (S.E.)			Adjusted odds ratio (men compared to women, within cohort)[a]
			Total (42,693)	Men (18,413)	Women (24,280)	OR (95% C.I.)
			12.5 (0.4)	17.4 (0.5)	8.0 (0.3)	2.59 (2.38–2.82)
Cohort 1	1913–1932	6,153	2.8 (0.3)	5.2 (0.5)	1.1 (0.2)	5.07 (3.29–7.80)
Cohort 2	1933–1949	9,149	8.6 (0.4)	13.2 (0.7)	4.5 (0.3)	3.86 (3.21–4.65)
Cohort 3	1950–1967	15,434	14.4 (0.5)	19.5 (0.7)	9.5 (0.6)	2.67 (2.31–3.07)
Cohort 4	1968–1984	11,957	17.2 (0.6)	22.1 (0.9)	12.3 (0.6)	1.97 (1.75–2.22)

[a] Birth cohort and sex significantly interacted in a logistic regression model ($F = 15.3$ [d.f. = 3], $p < 0.0001$); cohort-specific odds ratios are derived from this logistic regression model.

Table 4
Prevalence of lifetime DSM-IV alcohol abuse by birth cohort and gender

	Birth cohort	N (total 42,693)	% (S.E.)			Adjusted odds ratio (men compared to women, within cohort)[a]
			Total (42,693)	Men (18,413)	Women (24,280)	OR (95% C.I.)
			17.8 (0.5)	24.6 (0.7)	11.5 (0.4)	2.22 (2.06–2.39)
Cohort 1	1913–1932	6,153	11.9 (0.6)	23.0 (1.2)	3.8 (0.4)	7.14 (5.59–9.13)
Cohort 2	1933–1949	9,149	19.0 (0.7)	29.4 (1.1)	9.6 (0.6)	3.78 (3.26–4.38)
Cohort 3	1950–1967	15,434	22.1 (0.7)	28.1 (0.9)	16.3 (0.7)	1.79 (1.61–1.99)
Cohort 4	1968–1984	11,957	14.3 (0.6)	17.7 (0.8)	11.0 (0.6)	1.63 (1.41–1.87)

[a] Birth cohort and sex significantly interacted in a logistic regression model ($F = 62.0$ [d.f. = 3], $p < 0.0001$); cohort-specific odds ratios are derived from this logistic regression model.

3.3. Alcohol dependence

Table 3 shows the prevalence of lifetime DSM-IV alcohol dependence (with or without abuse) stratified by birth cohort and gender. The prevalence of dependence increased monotonically among men and women by birth cohort. Among men, the prevalence increased from 5.2% in Cohort 1 to 22.1% in Cohort 4. Among women, the prevalence increased from 1.1% in Cohort 1 to 12.3% in Cohort 4. Birth cohort and sex interacted significantly in predicting lifetime alcohol dependence ($F = 15.3$ [d.f. = 3], $p < 0.0001$), signaling the presence of significant effect modification of cohort on the gender ratio of alcohol dependence. The odds ratios comparing the risk in men and women (calculated by the same method described above for frequent binge drinking) indicated consistent decreases across birth cohorts. Among those in Cohort 1, men are 5.07 (95% C.I. 3.29–7.80) times more likely to have alcohol dependence. By the youngest cohort, Cohort 4, men are 1.97 (95% C.I. 1.75–2.22) times more likely to have alcohol dependence.

3.4. Alcohol abuse

Table 4 indicates the prevalence of lifetime DSM-IV alcohol abuse (without dependence) stratified by birth cohort and gender. For alcohol abuse, prevalence increased by birth cohort for Cohorts 1–3, and then decreased for both men and women in Cohort 4. An overall interaction of birth cohort and sex in predicting lifetime alcohol abuse was highly significant ($F = 62.0$ [d.f. = 3], $p < 0.0001$), signaling the presence of substantial effect modification of cohort on the gender ratio for alcohol abuse. The odds of alcohol abuse in men compared to women monotonically decreased by birth cohort (odds ratios calculated by the same method described above for frequent binge drinking), which indicated the presence of significant effect modification, although the difference in the odds ratio in Cohorts 3 and 4 was slight.

3.5. Alcohol abstention

Table 1 indicates the prevalence of lifetime alcohol abstention (% of respondents who have never had more than a sip or taste of alcohol). While women were more likely to be alcohol abstainers in each cohort, the odds ratio for the gender difference in alcohol abstention indicated converging gender differences

similar to the other measures presented. Birth cohort and sex significantly interacted in a logistic regression model ($F = 11.1$, [d.f. = 3], $p < 0.0001$) to predict lifetime alcohol abstention.

4. Discussion

We examined overall rates and gender differences in alcohol consumption, binge drinking, alcohol abuse and dependence by birth cohort in the U.S. general population, and conducted statistical tests of whether birth cohort modified the magnitude of gender differences in the lifetime prevalence of these four important alcohol variables. The results showed substantial, monotonic decreases in gender differences between the oldest and youngest cohorts for all alcohol variables, confirmed by the significance of the interaction tests for effects of birth cohort and gender. The odds ratios for gender differences in risk of frequent binge drinking and alcohol dependence decreased from the oldest to youngest cohorts by a factor of about four, and the corresponding odds ratios for alcohol abuse decreased across cohorts by a factor of about 2.65. Across measures of consumption and alcohol diagnosis, we observed the greatest gender conversion for binge drinking (from an odds ratio of 10.6 in the oldest cohort to an odds ratio of 2.7 in the youngest), but binge drinking remained the alcohol measure with the greatest discrepancy between men and women. Alcohol abuse was the indicator with the smallest gender difference in the youngest cohort, with men in the youngest cohort being 1.63 times as likely to have abuse compared to women.

This research represents an important contribution to the study of gender differences in alcohol disorders; using the best cross-sectional data available, this work supports and extends accumulating evidence in less representative samples that gender differences in alcohol disorders are decreasing as suggested previously by Reich et al. (1988), Holdcraft and Iacono (2002), and Rice et al. (2003) for alcohol dependence, indicating support for a cohort effect on gender differences in the population. Additionally, we extended the earlier work of Grant in the NLAES (Grant, 1997) by testing directly for a gender by birth cohort interaction in predicting alcohol abuse and dependence. We additionally showed that frequent binge drinking increased monotonically among women by birth cohort. This finding is contrary to other large studies of women only (Wilsnack et al., 2006; Neve et al., 1996) that suggested declines in heavy episodic drinking (6+ drinks once per week or more). The present study is perhaps

more representative of recent overall trends in frequent binge drinking in the United States due to (1) a larger, epidemiologic sample of both males and females in the U.S. population and (2) a cohort of more recently born participants. Finally, we observed differences in the magnitude of gender convergence across measures of consumption and disorder, underscoring the concept that different alcohol measures are tapping into distinct constructs. Differences in the effect of gender across alcohol-related constructs is an important avenue for further research, as it may have implications for understanding the biological underpinning of gender differences in alcohol. However, similar to previous literature in this area, it should be noted that a cross-sectionally designed study cannot empirically test for the presence of a birth cohort effect as opposed to an age or period effect; while these data are consistent with a cohort effect, age and period effects could be simultaneously influencing the results.

There are several possible explanations for the decreased male/female differences in alcohol use disorders. First, there could be a true cohort effect such that drinking behaviors and risk for alcohol disorders among women and men are converging in more recent cohorts. If so, the potential mechanisms, both social and biological, involved in alcohol-related gender differences should be considered. While early twin and adoption studies suggested greater genetic contribution to alcoholism among men (Cloninger et al., 1981; Light et al., 1996; Jang et al., 1997), larger, population-based twin samples show no gender difference in heritability (Heath et al., 1997; Prescott et al., 1999; Prescott and Kendler, 2000). Other biological factors include male–female differences in alcohol metabolism (Jones and Jones, 1976; Sutker et al., 1987; Thomasson, 1995; Lieber, 1997), greater sensitivity to adverse health effects due to heavy drinking among women. Despite the decreases in gender differences in cohorts shown above, the prevalence of all alcohol disorders remained higher in men than women in all birth cohorts. Thus, these biological differences may explain some part of the remaining gender gap, although they clearly do not account for the changes by birth cohort. For such changes, social-environmental explanations must be sought.

Some investigators hypothesized that stress among women due to pursuing both career and family leads to increased alcohol use and misuse (Fillmore, 1984; Johnson and Gerstein, 1998). However, since other studies indicated that women with multiple roles were at lower risk for alcohol disorders, this explanation seems unlikely (LaRosa, 1990; Wilsnack and Wilsnack, 1991). An association between frequency of alcohol consumption among women and the number of men in their workplace (Haavio-Mannila, 1991) was interpreted as showing an imitation effect (Holmila and Raitasalo, 2005). A study of medical students in the 1980s found that at the start of medical school, female students had fewer alcohol-related problems than men, but by the start of clinical training, the gender difference had disappeared (Richman and Rospenda, 1992). Perhaps imitation as well as increased socialization to traditionally male medical roles decreased constraints against drinking originally shown by the women at the beginning of medical school. Finally, from 2001 to 2002, the proportion of young girls exposed to print advertising of low-alcohol beverages (e.g. wine coolers)

increased by 216% (Jernigan et al., 2004). These and other time trends in advertising exposure to young women may have increased the social acceptability of alcohol use by women in younger generations.

We noted above clear increases in the proportion of women working outside the home and decreases in the proportion of women having children (Thronton and Freedman, 1983; Echols, 1989; Rosen, 2000). We also noted changes in gender-based drinking norms. More specifically, social norms for drinking in various situations were compared over three national surveys conducted between 1979 and 1990. During those years, there was no change in the proportion of respondents who felt that "a man drinking at a bar with friends" was acceptable. However, there was a significant increase in the proportion that felt "a woman drinking at a bar with friends" was acceptable (Greenfield and Room, 1997). This indicates a decrease in the negative perception associated with drinking in women, potentially leading to greater opportunities for them to experience alcohol problems.

The changes in social norms for drinking and drunkenness lead to a second potential explanation for the observed decrease in gender differences in alcohol disorders; that differential changes in stigma by gender associated with the reporting of drinking could give rise to the appearance of a cohort effect on gender differences due to social desirability effects on self-reports of drinking. Qualitative historical research has focused on adherence by women and men to norms of "moral" behavior. Alcohol researchers observed that in the early 20th century, female sex roles were characterized by greater "conventionality" and "acceptance of the dominant 'official' standards of morality and propriety" that included alcohol consumption (Clark, 1967). Women that appeared to abstain or drink very little were thus more closely following the official standards of morality and propriety in the time period, while men, less bound by these standards, were more likely to drink and develop chronic alcohol problems. As these norms changed, the reduced need to adhere to such moral norms was greater for women, potentially affecting not only drinking but also self-reports of drinking among women. Such ideas are intriguing, but unfortunately, few means to verify them empirically are available in the absence of time trend data comparing reported versus true alcohol use among men and women. To definitively rule out social desirability as an explanation of the findings reported above, alternative indicators of alcohol use not based on self-report should be identified and analyzed, work that is currently in progress. However, this issue does not negate the importance of statistically demonstrating a decreased gender difference in successively younger birth cohorts, as we have done above.

A third alternative hypothesis could be that the observed increase in the prevalence of alcohol disorders in younger cohorts is due to the inability of the cross-sectional design to capture all cases of alcohol disorders in the older cohorts (i.e. those respondents in the oldest cohorts are those that survived to be surveyed between 2001 and 2002). As adults with active alcohol disorders have a higher mortality rate than the general population, some lifetime cases of alcohol dependence are probably missed in older cohorts, underestimating the prevalence of

disorder in these groups. However, as we are comparing men to women within each cohort, the observed odds ratios will only be affected if there is differential alcohol-related mortality between men and women in the oldest cohorts. While there is some evidence that among those with active, chronic alcohol dependence, women have a greater sensitivity to adverse health effects due to heavy drinking (Deal and Gavaler, 1994; Hanna et al., 1997; Singletary and Gapstur, 2001; Hommer et al., 2001), this literature is based on men and women with unremitting long-term cases of alcohol dependence. As most cases of alcohol dependence in the general population across gender remit at some point (Dawson et al., 2005), it is unlikely that the death of women in the general population with alcohol use disorders account for the observed effect. However, the extent of differential mortality by gender among lifetime cases of alcohol dependence in the general population is unknown and should be determined.

As with all cross-sectional data, these data are limited by recall bias. The conclusion that these results are most consistent with a birth cohort effect relies on the assumption that the lifetime measure of alcohol use and alcohol use disorder is accurate. Previous literature has established that recall bias is an issue in the reporting of past alcohol consumption (Liu et al., 1996; Caldwell et al., 2006), thus these results should be interpreted with caution. This issue is exemplified in the NESARC sample as we observed few lifetime cases of alcohol disorders in respondents older than 90 at the time of survey, although this effect may be a combination of recall bias and selective mortality. However, to account for this possibility we removed those respondents at or older than 90 years old. Additionally, since our findings for binge drinking and alcohol abuse follow the patterns of the yearly per capita alcohol consumption in the United States, it is unlikely that the observed cohort effect is entirely a reflection of poor recall among heavy drinkers in the older cohorts (Johnson and Gerstein, 1998; Lakins et al., 2005). Additionally, because the NESARC sampled those 18 and older, some people in the youngest-born cohort may have been misclassified as unaffected because they have not had time to develop alcohol disorders. If this is true, however, then the prevalence of alcohol disorders in the younger cohort was underestimated. This could be re-examined using data from the 3-year follow-up of the NESARC sample that will become available in the next few years. Finally, future large-scale surveys using the AUDADIS instrument could also be used to simultaneously estimate the effect of cohort, age, and period effects on trends in alcohol consumption and alcohol use disorders in the United States, an important area for continued follow-up research.

Despite the necessity to test alternative hypotheses, the study has several substantial strengths that make these findings a contribution to our understanding of the epidemiology of alcohol disorders. First, the state-of-the-art instrument used for data collection (the AUDADIS-IV) increases the sensitivity and specificity of the estimates of alcohol consumption and alcohol disorders. Second, the large, representative sample of men and women represent an improvement over previous cross-sectional studies attempting to examine birth cohort effects on gender differences, which previously were conducted in samples from family genetic studies. Third, in contrast to information from previous epidemiologic samples, this study specifically tested a hypothesis regarding a cohort by gender interaction, and had sufficient power to detect such interactions.

In conclusion, these data suggest that gender-related differences in drinking and alcohol use disorders in the U.S. are declining. Particularly noteworthy is the finding that frequent binge drinking decreased among men in the youngest birth cohort but showed a monotonic increase in younger cohorts among women. Women may thus need specifically targeted prevention and treatment efforts, and should not be disregarded by researchers and clinicians as a group unlikely to develop alcohol problems. Future research should examine the sociocultural factors that have encouraged the expression of alcohol abuse and dependence in women for better empirical information on the decline in gender differences in alcohol use disorders.

Conflict of interest

None.

Acknowledgement

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.drugalcdep.2007.08.017.

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