False Negatives in the Assessment of Lifetime Alcohol Use Disorders: A Serious But Unappreciated Problem

ANGELA M. HAENY, M.A.,[a,*] ANDREW K. LITTLEFIELD, PH.D.,[b] AND KENNETH J. SHER, PH.D.[a]

[a]*Midwest Alcoholism Research Center and Department of Psychological Sciences, University of Missouri–Columbia, Columbia, Missouri*
[b]*Psychology Department, Texas Tech University, Lubbock, Texas*

ABSTRACT. Objective: Some individuals will not meet criteria for a lifetime alcohol use disorder (AUD) at a baseline assessment but will at a follow-up measurement, but not because the disorder began after the initial evaluation. Despite several research implications, this type of unreliability of lifetime AUD estimates has not been studied extensively. The present study investigated the extent of false negatives in the assessment of lifetime AUDs using longitudinal data. **Method:** A prospective cohort of college freshmen (baseline $N = 489$) were assessed seven times between ages 18 and 34 years using the Diagnostic Interview Schedule. Individuals were categorized as false negatives at the index assessment using a retrospective (using *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* [DSM-III], and DSM-IV data), a prospective (using DSM-III data only), and a combined approach (using DSM-III data only). **Results:** For DSM-IV, of the 29 ostensible new onsets at a follow-up 5 years later (age approximately 34 years), 28 (96%) reported meeting AUD criteria before the index assessment (age approximately 29 years). For DSM-III, of the 25 ostensible new onsets, the retrospective, prospective, and combined approaches categorized 18 (72%) individuals as false negatives at the index assessment. **Conclusions:** These findings further demonstrate sensitivity issues with lifetime AUD assessments and call into question the validity of "onset" cases that rely on only two waves of data, especially when the follow-up assessment fails to reassess lifetime fully (i.e., across the entire drinking history). (*J. Stud. Alcohol Drugs, 75,* 530–535, 2014)

PREVIOUS RESEARCH HAS EXTENSIVELY documented limitations in the assessment of lifetime alcohol use disorders (AUDs) in terms of individuals meeting criteria for a lifetime AUD at an initial assessment but failing to do so at a subsequent assessment (i.e., negative prevalence; Haeny et al., 2014; Jackson et al., 2006; Robins, 1985; Shrout et al., 2011; Vandiver and Sher, 1991). In addition to negative prevalence, unreliability of lifetime estimates may be reflected by individuals not meeting criteria for a lifetime AUD at a baseline assessment but doing so at a subsequent assessment, an issue that has received much less consideration. Although such discrepancies can represent a legitimate new onset of the disorder, it is possible that the absence of a diagnosis at baseline followed by a diagnosis at follow-up indicates a false negative diagnosis at baseline. Evidence for the latter would include, for example, the onset of the disorder at follow-up being retrospectively dated to precede the baseline assessment. To our knowledge, no study has investigated the unreliability of lifetime AUDs with respect to the possibility that ostensible new onsets at follow-up are actually false negatives at baseline.

This type of measurement error could have a large number of adverse consequences in alcohol research. Not only will the estimated prevalence of lifetime AUDs (e.g., 18% in the National Longitudinal Alcohol Epidemiological Study [Grant and Hartford, 1995] and 30% in the National Epidemiological Survey on Alcohol Related Conditions [NESARC; Hasin et al., 2007]) be underestimates, but various kinds of studies that rely on lifetime AUDs for research phenotypes, such as genetic studies (Goodwin, 1971; Schuckit et al., 1996; Wang et al., 2012), studies of children of alcoholics (Buu et al., 2012; Macdonald and Blume, 1986; Sher, 1991), or health disparities (e.g., Edlund et al., 2012; Wells et al., 2001), could be inaccurate (and underpowered) if those with lifetime AUDs are grouped with those without.

Additionally, research on the course of AUDs requires temporal dating of various drinking milestones. A false negative assessment of AUD at baseline could lead to erroneous inferences as to the timing of drinking-related milestones. For example, the age-incidence curve for AUDs (e.g., Vergés et al., 2012) could have artificially lower hazard rates earlier in development and artificially higher rates later in development if individuals with true lifetime AUDs were false negatives at baseline but were later found to have an AUD. Similarly, research examining the relation between risk factors such as role transitions and the development of AUDs suggests that late-in-life onset can be predicted from remaining single and from becoming divorced (Chilcoat and Breslau, 1996) and getting a new job (Vergés et al., 2012). However, if the proportion of new onset cases of AUDs actually represents false negatives at an initial assessment, such findings are suspect. That is, studies on the course of AUDs require considerable sensitivity of lifetime AUDs.

Received: August 24, 2013. Revision: November 7, 2013.

This research was supported by National Institute on Alcohol Abuse and Alcoholism Grants F31 AA019596 (to Andrew K. Littlefield); T32 AA13526, R01 AA13987, R37 AA07231, and KO5 AA017242 (to Kenneth J. Sher); and P60 AA11998 (to Andrew Heath).

*Correspondence may be sent to Angela M. Haeny at 145 Psychology Building, Department of Psychological Sciences, University of Missouri, Columbia, MO 65211, or via email at: amhnq6@mail.missouri.edu.

Notably, there is no gold standard (e.g., a genetic marker or laboratory test) to determine if an individual was correctly diagnosed with a lifetime AUD, making it difficult to definitively distinguish false negatives from true negatives and false positives from true positives. However, given the availability of longitudinal data, we can investigate the apparent rate of false negatives in the assessment of lifetime AUDs. Evidence for this would come from contradictory information across prospective assessments, such as later assessments indicating that a lifetime AUD was present before the index assessment or assessments before the index assessment documenting a lifetime AUD preceding a negative lifetime AUD assessment.

Method

Participants

The data were drawn from the Alcohol Health and Behavior study (Sher et al., 1991), which began in 1987 with a sample of first-year undergraduate students (N = 489) from a large midwestern university. Seven assessments were conducted over a 16-year period (approximately at ages 18 [Wave 1], 19 [Wave 2], 20 [Wave 3], 21 [Wave 4], 25 [Wave 5], 29 [Wave 6], and 34 years [Wave 7]). The baseline sample consisted of 53% women with a mean age of 18.55 years (SD = 0.97), 94% White, and 52% reported a family history of paternal alcoholism (by design; see Sher et al., 1991).

Measures

The Diagnostic Interview Schedule (DIS) Version III-A (Robins et al., 1985) for *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (DSM-III), criteria was used to assess 12-month and lifetime AUD and alcohol dependence diagnoses at all seven time periods (consistent with the DSM criteria available at the time of the start of the study). The DIS is an instrument with acceptable to excellent reliability and validity for DSM-III diagnoses (Robins et al., 1981, 1982). In our previous work, we found that test–retest reliability for DSM-III AUD criteria varied (κ ranged from .37 to .71; Y ranged from .42 to .74) depending on the time between measurement occasions, and the rate of re-diagnosis was less than 80%, even for a 1-year interval between measurement occasions (Haeny et al., 2014). New DIS items (i.e., DIS-III-R for the DSM-III-R criteria [Robins et al., 1989] and the DIS-IV for the DSM-IV criteria [Robins et al., 1997]) were added to the interview as they became available. As a result, DSM-III (American Psychiatric Association, 1980) AUD data were collected at Waves 1–7, DSM-III-R (American Psychiatric Association, 1987) AUD data were collected at Waves 3–7, and DSM-IV (American Psychiatric Association, 1994) AUD data were collected at Waves 6 and 7. Supplementary questions regarding the age at onset for each symptom were added to the alcohol and other drug modules of the DIS. Ostensible new onsets were investigated between Waves 6 and 7 (the only two waves with DSM-IV data). For the purposes of the present study, the index assessment refers to Wave 6 (the assessment at age ≈ 29 years), and follow-up refers to Wave 7 (the assessment at age ≈ 34 years). There were 292 (71% of those assessed at Wave 6) and 247 (60% of those assessed at Wave 6) individuals without a lifetime AUD at the index assessment using DSM-III and DSM-IV criteria, respectively.

Data analysis

SAS version 9.2 (SAS Institute Inc., Cary, NC) was used to analyze the data. Potential false negatives at the index assessment were investigated using DSM-III (data available at all seven waves) and DSM-IV (data only available at Waves 6 and 7). For the DSM-III criteria, three methods (i.e., a retrospective approach, a prospective approach, and a combination of the two approaches) were used to identify potential false negatives at the index assessment. Given that DSM-IV data were only available at Waves 6 and 7, just the retrospective approach was used for these criteria.

For the retrospective approach, individuals were categorized as false negatives at the index assessment if ostensible onsets at follow-up (i.e., no lifetime diagnosis at baseline followed by a lifetime diagnosis at follow-up) retrospectively reported an age at onset earlier than their age at the index assessment. Assuming no error in recalling age at onset, "valid" new onsets at follow-up should not report having met syndromal criteria for lifetime diagnosis before the index assessment. For example, say Participant X appeared to have an onset for a DSM-IV AUD between the index assessment (age 29) and follow-up (age 34). However, if at the follow-up assessment Participant X reported an onset of three DSM-IV dependence symptoms within the same 12-month period when 18–19 years of age (and no earlier history of abuse), then Participant X would be categorized as a false negative for a DSM-IV lifetime AUD at the index assessment. Under the prospective approach, a later assessment occasion in a prospective study (e.g., Wave 6 of the current study) is arbitrarily designated as the "index assessment" and earlier waves (e.g., Waves 1–5 of the current study) for the presence of a prior lifetime diagnosis. "True" negatives for lifetime AUD at the index assessment should not have lifetime AUD diagnoses before the index assessment.

With a combined approach, both retrospective age-at-onset data at follow-up and data before the index assessment are used to categorize individuals as false negatives at the index assessment. Consider Participant Y as an example. Participant Y appeared to have an onset for DSM-III AUD between the index assessment (age 29 years) and follow-up (age 34 years). For Participant Y to be deemed as a "valid"

negative at the index assessment (or conversely, a "valid" onset at follow-up), Participant Y should not have any lifetime AUD diagnoses before the index assessment and should report an age at onset of AUD syndromal criteria older than age 29 years.

Results

Retrospective approach: Using age-at-onset data at follow-up to identify likely false negatives and new onsets

DSM-IV. There were 247 individuals without a DSM-IV lifetime AUD at the index assessment (Wave 6), of which 227 provided data at follow-up (Wave 7). Twenty-nine individuals (13%) met criteria for a DSM-IV lifetime AUD at follow-up but not at the index assessment, superficially suggesting new onset of the disorder. Of the 29 ostensible new onsets at follow-up, 28 (96%) reported having an AUD before the index assessment (27 abuse diagnoses, 1 dependence diagnosis). Interestingly, these false negatives at the index assessment retrospectively reported (at follow-up) meeting AUD syndromal criteria at a median age of 18.8 years, on average 10.1 years younger than their age at the index assessment (≈ 29 years old). Thus, only a very small minority of individuals who reported no lifetime diagnosis at the index assessment followed by a follow-up lifetime diagnosis (i.e., 1 of 29; 3%) appear to represent "valid" new onsets for DSM-IV lifetime AUDs.

DSM-III. There were 292 individuals without a DSM-III lifetime AUD at the index assessment, of which 269 provided data at follow-up. Twenty-five individuals (9%) reported a DSM-III lifetime AUD at follow-up but not at the index assessment, superficially suggesting new onset of the disorder. Of the 25 ostensible new onsets at follow-up, 13 (52%) reported AUD diagnoses before the index assessment (12 abuse diagnoses, 1 dependence diagnosis), suggesting that these individuals were likely false negatives at the index assessment. These apparent false negatives at the index assessment retrospectively reported at follow-up experiencing onset of an AUD at a median age of 20 years, on average 8.7 years younger than their age at the index assessment (≈ 29 years old). Thus, only 48% (i.e., 12 people) appeared to be valid new onsets for DSM-III lifetime AUDs.

Although the retrospective approach to DSM-III and DSM-IV yielded somewhat different estimates of false negative rates (12% in DSM-IV, 5% in DSM-III) and likely new onset rates (<1% in DSM-IV, 4% in DSM-III) among those with a negative lifetime AUD diagnosis at the index assessment, it is clear from both sets of analyses that only a minority of individuals with a negative lifetime diagnosis at the index assessment who subsequently are diagnosed with a lifetime AUD are likely to be true new onsets in this sample. As noted above, the high false negative rate was largely restricted to abuse diagnoses.

The possibility exists that the individuals we deemed as false negatives at the index assessment actually unreliably reported their retrospectively assessed age at onset for each symptom and therefore were indeed new onsets of AUD at follow-up. However, we have no way of distinguishing whether they incorrectly reported not meeting criteria for an AUD at the index assessment or incorrectly reported the age at onset for each AUD symptom at follow-up. An approach examining diagnoses before the index assessment was used as another technique less burdened by this issue. We partially attribute the inconsistency in reporting to the relatively low sensitivity of retrospective assessments; therefore, the examination of diagnoses before the index assessment may provide a stronger case for evidence of false negatives at the index assessment.

Prospective approach: Using data before the index assessment

DSM-III. The method using data before the index assessment (i.e., Wave 6) was limited to DSM-III criteria, given that these data were available at all seven waves. There were 292 individuals without a DSM-III lifetime AUD at the index assessment, of which 269 were reassessed at follow-up. If this were a cross-sectional study that only had data at the index assessment, we would assume that none of these 292 individuals ever had a lifetime AUD. However, 69 (24%) individuals met the criteria for a lifetime AUD (46 abuse diagnoses, 23 dependence diagnoses) at one or more waves before the index assessment, suggesting that they were false negatives at the (Wave 6) index assessment. The data in Table 1 show a strong association between being diagnosed with a lifetime AUD at the Wave 7 follow-up and the number of diagnoses before the Wave 6 index assessment among those with no lifetime AUD at the Wave 6 index assessment, $\chi^2(5) = 34.36$, Cramer's $V = 0.36$, $p < .01$. Having one or more diagnoses before the index assessment (i.e., during Waves 1–5) was associated with increased odds (odds ratio = 5.34, 95% CI [2.28, 12.50]) of having a lifetime AUD at the Wave 7 follow-up among those with no lifetime diagnosis at the Wave 6 index assessment. In addition, Table 1 shows that, among those without a lifetime AUD at both Waves 6 and 7, 20% met the criteria for lifetime AUD at one or more waves before Wave 6. These findings from the prospective approach indicate that false negatives may be underestimated when using the retrospective approach.

Combined approach

A combined approach was used to integrate both retrospective age-at-onset data and pre-baseline data at follow-up. However, given that DSM-IV data were only available at Waves 6 and 7, the combined approach, which used data at all seven waves, was limited to DSM-III data only. As noted

Table 1. Number of previous lifetime alcohol use disorder (L-AUD) diagnoses at Waves 1–5 among those who did not meet criteria for an L-AUD at Wave 6 (n = 292)

	Total number of previous DSM-III L-AUDs at Waves 1–5						
Variable	0	1	2	3	4	5	Total
No L-AUD at W6 and W7 (n = 244)	197 (80%)	17 (7%)	17 (7%)	4 (2%)	4 (2%)	5 (2%)	244
No L-AUD at W6 and positive L-AUD at W7 (n = 25)	11 (44%)	2 (8%)	3 (12%)	1 (4%)	4 (16%)	4 (16%)	25
No L-AUD at W6 and not assessed at W7 (n = 23)	15 (65%)	3 (13%)	1 (4%)	2 (9%)	0 (0%)	2 (9%)	23
Total	223	22	21	7	8	11	292

Notes: DSM-III = *Diagnostic and Statistical Manual of Mental Disorders, Third Edition;* W6 = Wave 6; W7 = Wave 7.

earlier, 269 individuals had data at the Wave 6 index assessment and the Wave 7 follow-up. Among these individuals, 25 (9%) met the criteria for DSM-III lifetime AUD at follow-up, superficially suggesting new onset of the disorder. Of the 25 ostensible new onsets at follow-up, 4 (16%) were deemed to be false negatives based on the retrospective approach alone, 5 (20%) appeared to be false negatives based on the prospective approach alone, and 9 (36%) appeared to be false negatives according to both the retrospective and prospective approaches, resulting in 18 (72%) total individuals who appeared to be false negatives at baseline (12 abuse diagnoses, 6 dependence diagnoses). This suggests that only seven individuals were "true" new onsets. These seven individuals did not meet criteria for DSM-III lifetime AUD before the index assessment and retrospectively reported at follow-up meeting syndromal criteria before their age at the index assessment.

Discussion

Despite the fact that AUD false negatives at follow-up have been a focus of prior research (so-called negative prevalence), the possibility that false negatives could occur at baseline has not been previously considered. This is somewhat surprising because if there is inherent unreliability of lifetime assessment occasioned by relatively low sensitivity, false negatives at baseline should be at least as problematic as false negatives at follow-up. Indeed, the issue is likely even more problematic because some large, costly studies assume that baseline assessments are sufficiently reliable, and once obtained, follow-up assessments need only cover the time since the prior assessment. Our findings suggest that such an approach is potentially problematic and call into question acceptance of this practice; further, they suggest the value of repeated, full lifetime assessments. Researchers should recognize the problem of low sensitivity of lifetime assessments and not rely on single assessments of lifetime AUDs from a single reporter if possible. When feasible, multiple reporters and repeated assessments over time should be obtained.

We note that the retrospective approach enabled by a single, follow-up assessment could reflect unreliable reports for age at onset rather than false negatives at the index assessment. Researchers may be tempted to distrust the approach using retrospectively assessed age-at-onset data from follow-up assessments to investigate false negatives at an index assessment; however, there is no a priori reason to give more credence to an interview at one age than another (e.g., ≈ 29 years rather than age ≈ 34 years in this study). Moreover, these individuals, on average, reported experiencing the onset of an AUD 10.26 years before the age 29 assessment, with an average age at onset of 19.32 years (consistent with the peak prevalence of lifetime AUDs; e.g., Sher et al., 2005). Given this large discrepancy in years, it is seems highly unlikely that most of the false negatives at the index assessment were incorrectly recalling their age at onset at the age ≈ 34 follow-up.

Consequently, these findings and others (Copeland et al., 2011; Haeny et al., 2014; Moffitt et al., 2010) suggest that epidemiological studies that measure lifetime estimates of AUDs at one time point are very likely underestimating the true rate of the disorder in the population as operationalized by study instruments. Importantly, major studies like the NESARC (Grant et al., 2003) that only assess alcohol problems in the interval since the last assessment (Grant and Dawson, 2006) are likely to not only underestimate the full lifetime prevalence of a disorder but also to overestimate new onsets of disorders. That is, someone who was negative for a lifetime AUD at baseline could report meeting criteria in the recent past at follow-up. However, without reassessing lifetime drinking fully, it is impossible to determine if this is really a new onset or a recurrence. We strongly suggest that longitudinal researchers reassess lifetime diagnoses at every follow-up period, if practical.

Given that the present study also investigated ostensible new onsets at an age when the onset rate tends to be low, these findings suggest that the incidence of AUDs may actually be much lower than expected (at least between ages ≈ 29 and ≈ 34 years). The present study provides further evidence that researchers should be aware of the limitations of retrospective assessments of lifetime AUDs and be cautious when drawing strong conclusions when using such data. To the extent possible, institutional records and infor-

mant reports as well as participant reassessments should all be taken into consideration. Indeed, incorporating as much of Spitzer's (1983) LEAD standard (longitudinal, expert, all data) as possible can only help to improve the current state of practice.

Interestingly, all three methods indicated that the rate of false negatives was much higher for abuse diagnoses. These results are consistent with previous research suggesting that reliability was much lower for alcohol abuse diagnoses compared with alcohol dependence diagnoses (Easton et al., 1997; Hasin et al., 1997). However, there are major differences in the abuse/dependence distinction between DSM-III and DSM-IV; therefore, although our DSM-III findings are nominally consistent in suggesting that the problem is "worse" for abuse, changes in DSM versions should be kept in mind. The majority of individuals with any AUD diagnosis are threshold cases, and previous research (e.g., Haeny et al., 2014; Vandiver and Sher, 1991) has indicated that these individuals are less likely to be re-diagnosed over time. Although the DSM-5 (American Psychiatric Association, 2013) removed the abuse/dependence distinction, we predicted, based on previous research (Haeny et al., 2014), that individuals on the mild end of the AUD continuum (endorsing 2 of 11 symptoms) would be more likely to be unreliably diagnosed over time (see also Martin et al., 2011, regarding potential problems with the low diagnostic threshold in DSM-5). This probably will be most pronounced in general population and community samples, where the majority of cases are likely to be threshold, but less of a problem in clinical samples, where symptom counts are likely to be much higher.

Although we believe these findings draw attention to a potentially serious problem and are consistent with other types of data highlighting the unreliability of lifetime diagnoses, the generalizability of these findings across types of diagnostic interviews, training and skill of interviewers, assessment context, and study populations is unknown. Also, although the focus here has been on AUDs, existing data suggest that the situation is far worse for internalizing disorders such as mood and anxiety disorders (e.g., Hasin et al., 2005; Kessler et al., 2005; Moffitt et al., 2010; Vandiver and Sher, 1991). Therefore, the concerns about false negatives at baseline would appear to apply to these other types of assessments as well. The problems with lifetime assessment have been recognized for half a century (Gruenberg, 1963), but it is surprising how little attention this issue gets from practicing researchers.

Acknowledgments

Portions of the work reported here were completed as part of the first author's master's thesis. The authors would like to thank Drs. Phillip K. Wood and Victoria Osborne for their assistance with this project.

References

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Buu, A., Wang, W., Schroder, S. A., Kalaida, N. L., Puttler, L. I., & Zucker, R. A. (2012). Developmental emergence of alcohol use disorder symptoms and their potential as early indicators for progression to alcohol dependence in a high risk sample: A longitudinal study from childhood to early adulthood. *Journal of Abnormal Psychology, 121,* 897–908.

Chilcoat, H. D., & Breslau, N. (1996). Alcohol disorders in young adulthood: Effects of transitions into adult roles. *Journal of Health and Social Behavior, 37,* 339–349.

Copeland, W., Shanahan, L., Costello, E. J., & Angold, A. (2011). Cumulative prevalence of psychiatric disorders by young adulthood: A prospective cohort analysis from the Great Smoky Mountains Study. *Journal of the American Academy of Child and Adolescent Psychiatry, 50,* 252–261.

Easton, C., Meza, E., Mager, D., Ulüg, B., Kilic, C., Göğüş, A., & Babor, T. F. (1997). Test-retest reliability of the alcohol and drug use disorder sections of the Schedules for Clinical Assessment in Neuropsychiatry (SCAN). *Drug and Alcohol Dependence, 47,* 187–194.

Edlund, M. J., Booth, B. M., & Han, X. (2012). Who seeks care where? Utilization of mental health and substance use disorder treatment in two national samples of individuals with alcohol use disorders. *Journal of Studies on Alcohol and Drugs, 73,* 635–646.

Goodwin, D. W. (1971). Is alcoholism hereditary? A review and critique. *Archives of General Psychiatry, 25,* 545–549.

Grant, B. F., & Dawson, D. A. (2006). Introduction to the National Epidemiologic Survey on Alcohol and Related Conditions. *Alcohol Research & Health, 29,* 74–78. Retrieved from http://pubs.niaaa.nih.gov/publications/arh29-2/74-78.pdf

Grant, B. F., & Harford, T. C. (1995). Comorbidity between DSM-IV alcohol use disorders and major depression: Results of a national survey. *Drug and Alcohol Dependence, 39,* 197–206.

Grant, B. F., Moore, T. C., & Kaplan, K. D. (2003). *Source and accuracy statement: Wave 1 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC)*. Bethesda, MD: National Institute on Alcohol Abuse and Alcoholism.

Gruenberg, E. M. (1963). A review of mental health in the metropolis. The Midtown Manhattan Study. *Millbank Memorial Fund Quarterly, 41,* 77–94.

Haeny, A. M., Littlefield, A. K., & Sher, K. J. (2014). Repeated diagnoses of lifetime alcohol use disorders in a prospective study: Insights into the extent and nature of the reliability and validity problem. *Alcoholism: Clinical and Experimental Research, 38,* 489–500.

Hasin, D., Carpenter, K. M., McCloud, S., Smith, M., & Grant, B. F. (1997). The Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS): Reliability of alcohol and drug modules in a clinical sample. *Drug and Alcohol Dependence, 44,* 133–141.

Hasin, D. S., Goodwin, R. D., Stinson, F. S., & Grant, B. F. (2005). Epidemiology of major depressive disorder: Results from the National Epidemiologic Survey on Alcoholism and Related Conditions. *Archives of General Psychiatry, 62,* 1097–1106.

Hasin, D. S., Stinson, F. S., Ogburn, E., & Grant, B. F. (2007). Prevalence, correlates, disability, and comorbidity of DSM-IV alcohol abuse and dependence in the United States: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry, 64,* 830–842.

Jackson, K. M., O'Neill, S. E., & Sher, K. J. (2006). Characterizing alcohol dependence: transitions during young and middle adulthood. *Experimental and Clinical Psychopharmacology, 14,* 228–244.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62,* 593–602.

Macdonald, D. I., & Blume, S. B. (1986). Children of alcoholics. *American Journal of Diseases of Children, 140,* 750–754.

Martin, C. S., Steinley, D. L., Vergés, A., & Sher, K. J. (2011). Letter to the Editor: The proposed 2/11 symptom algorithm for DSM-5 Substance Use Disorders is too lenient. *Psychological Medicine, 41,* 2008–2010.

Moffitt, T. E., Caspi, A., Taylor, A., Kokaua, J., Milne, B. J., Polanczyk, G., & Poulton, R. (2010). How common are common mental disorders? Evidence that lifetime prevalence rates are doubled by prospective versus retrospective ascertainment. *Psychological Medicine, 40,* 899–909.

Robins, L. N. (1985). Epidemiology: Reflections on testing the validity of psychiatric interviews. *Archives of General Psychiatry, 42,* 918–924.

Robins, L. N., Cottler, L., Bucholz, K. K., & Compton, W. (1997). *Diagnostic Interview Schedule for DSM-IV (DIS-IV).* St. Louis, MO: Washington University.

Robins, L. N., Helzer, J. E., Cottler, L., & Goldring, E. (1989). *The National Institute of Mental Health (NIMH) Diagnostic Interview Schedule III-rev.* St. Louis, MO: Washington University.

Robins, L. N., Helzer, J. E., Croughan, J., & Ratcliff, K. S. (1981). National Institute of Mental Health Diagnostic Interview Schedule. Its history, characteristics, and validity. *Archives of General Psychiatry, 38,* 381–389.

Robins, L. N., Helzer, J. E., Croughan, J., Williams, J. B. W., & Spitzer, R. L. (1985). *The National Institute of Mental Health Diagnostic Interview Schedule, Version III-A* (DHHS Publication No. ADDM-T-42-3). Washington, DC: U.S. Public Health Service.

Robins, L. N., Helzer, J. E., Ratcliff, K. S., & Seyfried, W. (1982). Validity of the Diagnostic Interview Schedule, version II: DSM-III diagnoses. *Psychological Medicine, 12,* 855–870.

Schuckit, M. A., Tsuang, J. W., Anthenelli, R. M., Tipp, J. E., & Nurnberger, J. I., Jr. (1996). Alcohol challenges in young men from alcoholic pedigrees and control families: A report from the COGA project. *Journal of Studies on Alcohol, 57,* 368–377.

Sher, K. J. (1991). *Children of alcoholics: A critical appraisal of theory and research.* Chicago, IL: University of Chicago Press.

Sher, K. J., Grekin, E. R., & Williams, N. A. (2005). The development of alcohol use disorders. *Annual Review of Clinical Psychology, 1,* 493–523.

Sher, K. J., Walitzer, K. S., Wood, P. K., & Brent, E. E. (1991). Characteristics of children of alcoholics: Putative risk factors, substance use and abuse, and psychopathology. *Journal of Abnormal Psychology, 100,* 427–448.

Shrout, P. E., Bolger, N., Stadler, G. S., Jackson, G. L., Lane, S. P. (2011, June). *Are there attenuation effects in repeated reports of college drinking patterns?* Poster session presented at the annual meeting for the Research Society on Alcoholism, Atlanta, GA.

Spitzer, R. L. (1983). Psychiatric diagnosis: are clinicians still necessary? *Comprehensive Psychiatry, 24,* 399–411.

Vandiver, T., & Sher, K. J. (1991). Temporal stability of the Diagnostic Interview Schedule. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 3,* 277–281.

Vergés, A., Jackson, K. M., Bucholz, K. K., Grant, J. D., Trull, T. J., Wood, P. K., & Sher, K. J. (2012). Deconstructing the age-prevalence curve of alcohol dependence: Why "maturing out" is only a small piece of the puzzle. *Journal of Abnormal Psychology, 121,* 511–523.

Wang, K.-S., Liu, X., Aragam, N., Mullersman, J. E., Jian, X., Pan, Y., & Liu, Y. (2012). Polymorphisms in ABLIM1 are associated with personality traits and alcohol dependence. *Journal of Molecular Neuroscience, 46,* 265–271.

Wells, K., Klap, R., Koike, A., & Sherbourne, C. (2001). Ethnic disparities in unmet need for alcoholism, drug abuse, and mental health care. *American Journal of Psychiatry, 158,* 2027–2032.