Published in final edited form as:

Drug Alcohol Depend. 2008 January 11; 93(1-2): 111–120. doi:10.1016/j.drugalcdep.2007.09.004.

Cigarette Smoking and the Lifetime Alcohol Involvement Continuum

Christopher W. Kahler¹, David R. Strong², George D. Papandonatos³, Suzanne M. Colby¹, Melissa A. Clark⁴, Julie Boergers⁵, Raymond Niaura², David B. Abrams⁶, and Stephen L. Buka⁷

¹Center for Alcohol and Addiction Studies, Brown University, Providence, RI, 02912

²Butler Hospital and the Warren Alpert Medical School of Brown University, Providence, RI, 02906

³Center for Statistical Sciences, Brown University, Providence, RI 02912

⁴Center for Gerontology & Health Care Research, Brown University, Providence, RI, 02912

⁵Rhode Island Hospital and the Warren Alpert Medical School of Brown University, Providence, RI, 02903

⁶Office of Behavioral and Social Sciences Research, National Institutes of Health, Bethesda, MD. 20892

⁷Department of Community Health, Brown University, Providence, RI, 02912

Abstract

Greater understanding of how alcohol use relates to the initiation, progression, and persistence of cigarette smoking is of great significance for efforts to prevent and treat smoking and excessive drinking and their substantial combined iatrogenic effects on health. Studies investigating the relationship between levels of alcohol involvement and smoking have typically been limited by analytic approaches that treat drinking behavior and alcohol use disorder diagnoses as separate phenomena rather than as indicators of a single latent alcohol involvement dimension. The purposes of the present study were (a) to create a lifetime index of alcohol involvement that integrates information about alcohol consumption and alcohol problems into a single measure and (b) to relate this index to initiation of smoking, progression from initiation to daily smoking, progression from initiation to dependence, and persistence of smoking. Rasch model analyses of data from 1,508 middle-aged (34–44 years) adults showed that creating an additive index of lifetime alcohol involvement was psychometrically supported. Significant quadratic effects of alcohol involvement on initiation, progression, and persistence of smoking demonstrated that there were specific regions of the alcohol involvement continuum that were particularly strongly related to increased smoking. These results provide the most comprehensive depiction to date of the nature of the relationship between lifetime alcohol involvement and lifetime cigarette smoking and suggest potential avenues for research on the etiology and maintenance of smoking and tobacco dependence.

Keywords

Alcohol; alcohol use disorders; smoking; tobacco, item response modeling; Rasch model

CORRESPONDENCE TO: Christopher W. Kahler, Ph.D., Center for Alcohol and Addiction Studies, Brown University, Box G-S121-5, Providence, RI 02912, USA, E-mail: Christopher_Kahler@brown.edu, Phone: (401) 863-6651 Fax: (401) 863-6697.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

1. Introduction

A number of epidemiologic studies have documented the significant positive association between smoking and both alcohol use and alcohol use disorders (Anthony and Echeagaray-Wagner, 2000; Chioleri et al., 2006; Dawson, 2000; Falk et al., 2006; Friedman et al., 1991; Grant, 1998). These associations are of great public health significance not only because heavy drinking and tobacco use have additive and even synergistic iatrogenic health effects (Pelucchi et al., 2006), but also because greater understanding of their association ultimately may inform efforts to prevent and treat excessive drinking and smoking.

Most epidemiologic studies to date that have examined alcohol-smoking relations have relied on one of two analytic approaches: (a) correlating current levels of alcohol use and levels of smoking or (b) comparing rates of heavy drinking or alcohol use disorders between smokers, former smokers, and nonsmokers. Such approaches, while informative, do not allow for more detailed depiction of the alcohol-smoking relationship for three reasons. First, they necessarily reduce information through dichotomization of either alcohol use disorder (AUD) history (e.g., dependent or not) or drinking history (e.g., heavy drinking or not). Second, they typically focus entirely on only one type of alcohol involvement indicator at a time, namely either an index of current drinking or an index of current or past AUD. Item response modeling has shown that indicators of involvement with alcohol generally fall along a single latent dimension that ranges from minimal drinking to heavy drinking to mild alcohol problems to severe dependence (Kahler et al., 2005; Kahler et al., 2003; Krueger et al., 2004; Saha et al., 2007). These findings indicate that it is possible to place individuals along a single continuum of lifetime alcohol involvement based on lifetime indices of drinking and alcohol-related problems. Such a continuum integrates information about drinking history and alcohol use disorder symptoms and can yield a richer and more integrated examination of how an individual's history of alcohol involvement relates to key milestones in the lifetime smoking trajectory. For example, using an ordinal index of lifetime alcohol involvement may reveal whether the relationships between lifetime alcohol involvement and smoking outcomes are linear or take a curvilinear form in which there are regions along the alcohol involvement continuum where the odds of a given smoking outcome increase particularly rapidly with increasing alcohol involvement.

The third limitation of most prior studies of smoking and alcohol involvement is that they have collapsed lifetime smoking outcomes into categories that do not allow for a maximally informative examination of how alcohol involvement and smoking relate. Specifically, defining participants as either never, former, or current smokers, precludes a fine-grained examination of how alcohol involvement relates independently to initiation of smoking and to progression of smoking; those who never initiated smoking are typically combined in analyses with those who initiated smoking but did not progress to daily smoking. Examining initiation, progression, and persistence of smoking separately may allow for a greater understanding of how alcohol involvement relates to smoking. For example, studies have shown that prior alcohol use is associated with greater risk for initiating smoking (Jackson et al., 2002; Wetzels et al., 2003). However, it is not clear that increasingly high levels of alcohol involvement would be associated with increasing risk of smoking initiation. Initiation of smoking is likely to occur at an age in which alcohol use and problems have not approached their peak. Furthermore, smoking initiation is so common that rates may approach an asymptote at near 100% even at relatively moderate levels of alcohol involvement, leaving little variance in initiation to explain.

By contrast, among those who have initiated smoking, the relation between alcohol involvement and progression from initiation of smoking to daily smoking and tobacco dependence may be especially strong at higher levels of alcohol involvement. Alcohol use can potentiate the rewarding effects of smoking (Glautier et al., 1996; Rose et al., 2002, 2004), which could increase motivation to smoke. Thus, greater levels of alcohol involvement may

be associated with a consistent linear increase in the odds of progressing to daily smoking. However, there also may be common mechanisms that contribute to both dependence on alcohol and dependence on tobacco, such as specific genetic vulnerabilities (Enoch et al., 2006; Liu et al., 2005; Schinka et al., 2002). For example, the rates of current nicotine dependence among those with current alcohol dependence are markedly higher than the rates of nicotine dependence among those with alcohol abuse (Falk et al., 2006). Likewise, among adolescents and young adults, current smokers have a significantly higher odds of an alcohol use disorder compared to never smokers who drink equivalent quantities (Gruza and Bierut, 2006).

Finally, the relationship of lifetime alcohol involvement to persistence of smoking may be quite different than its relationship to progression to daily smoking and tobacco dependence. In general, current alcohol use is associated with a reduced odds of smoking cessation (Carmelli et al., 1993; Hymowitz et al., 1997; Osler et al., 1999; Sobell et al., 1995; Sorlie and Kannel, 1990; Zimmerman et al., 1990) with frequency of heavy alcohol use, rather than overall level of drinking, appearing to be most strongly associated with continued smoking (Dawson, 2000; Murray et al., 1995; Vander Ark et al., 1997). However, although cessation rates are lower for individuals with a history of AUD compared to those without an AUD history (Dawson, 2000), having a past history of alcohol problems does not appear to reduce the odds of successful smoking cessation on a given attempt (Hughes and Kalman, 2006). These varying results indicate the continued need for a detailed analysis of the relationship between lifetime alcohol involvement and persistence of smoking.

In the present study, we examined the relationship between lifetime alcohol involvement and four key lifetime smoking outcomes (initiation of smoking, progression from initiation to daily smoking, progression from initiation to tobacco dependence, and persistence of smoking in middle adulthood) among 1,508 adult participants in the Transdisciplinary Tobacco Use Research Center: New England Family Study (TTURC: NEFS). To define an alcohol involvement continuum, we relied upon item response analyses based on the Rasch model (Rasch, 1960). These Rasch analyses were used to select indicators of lifetime alcohol use and AUD symptoms that could be combined in a single additive scale that was unbiased across demographic groups. We then graphically depicted the rates of smoking initiation, progression, and persistence at each level of alcohol involvement. Finally, logistic regression models were used to examine whether the association between alcohol involvement and the log odds of smoking initiation, progression, and persistence was generally linear or showed significant curvilinear effects. We expected that alcohol involvement would relate most strongly to smoking initiation in the lower regions of the continuum and would relate to progression to daily smoking and tobacco dependence most strongly in the upper regions of the continuum. Based on equivocal evidence about the importance of a history of alcohol dependence in predicting smoking cessation outcomes, we expected a relatively weak positive linear trend between alcohol involvement and the odds of currently smoking at the time of the interview.

2. Method

2.1. Participants

Participants were offspring of pregnant women enrolled in the National Collaborative Perinatal Project (NCPP) between 1959 and 1966 (Broman, 1984; Niswander and Gordon, 1972). Mothers were enrolled during pregnancy, and their offspring were followed periodically through age 7. The TTURC: NEFS was established in 1999 to locate and interview a subsample of the adult NCPP offspring at the Providence, Rhode Island, and Boston, Massachusetts sites.

Participants in the current study were selected as part of the TTURC: NEFS using a multi-stage sampling procedure that oversampled families in which multiple siblings participated. For this

project, screening questionnaires were mailed to 4,614 of the 16,080 Boston and Providence TTURC:NEFS offspring who survived until age 7. Of the 3,121 questionnaires returned (67.6%), 3,062 were eligible for participation in the current study. Of these, we attempted to interview 2,549 individuals, and at the conclusion of the study enrolled 1,674 TTURC:NEFS offspring (65.7%). Data from 49 individuals were excluded from the final sample because they received a pilot version of the survey ($n = 11$) or because of problems with the interview administration ($n = 38$). This yielded a sample of 1,625 completed adult assessments. This sample was 59.2% female, and 61.1% were married. The mean age was 39.1 years ($SD = 1.9$; range = 34 - 44). The racial/ethnic composition of the sample was as follows: 83.5% non-Hispanic White, 9.6% Black/African American, 1.1% Hispanic/Latino, and 5.9% of other backgrounds. Six percent of participants completed less than high school education, 19.1% completed high school or GED only, 46.4% completed some post-secondary education, 18.9% completed college, and 9.6% completed a graduate degree. The sample included 923 singletons, 280 sibling pairs, 39 sibling trios, 5 sibling quartets, and 1 sibling quintet.

2.2. Measures

2.2.1. Tobacco use and dependence—Smoking histories were obtained by the Lifetime Interview of Smoking Trajectories and the Quitting Methods Questionnaire, developed by the Methods and Measurement core of the TTURC: NEFS. These instruments obtain detailed information on participants' experiences with tobacco smoking beginning from experimentation, progression to regular smoking, levels of consumption, and patterns of quit attempts. In addition, tobacco dependence according to DSM-IV criteria (American Psychiatric Association, 1994) was assessed using a modified version of the Composite International Diagnostic Interview (CIDI) (World Health Organization, 1990). This modified diagnostic module is described in detail in Dierker et al. (2007).

Data were available to classify 1,506 participants (99.9% of the sample) according to whether they had ever initiated smoking, defined as ever having a puff of a cigarette. Among those who had initiated smoking ($n = 1,350$), 1,340 (99.3%) could be classified according to whether they had ever progressed to daily smoking, and 1,331 (98.6%) could be classified according to whether that had ever progressed to tobacco dependence. Finally, of the 834 participants who had ever smoked daily, 830 (99.5%) were classified according to whether they had persisted in smoking, defined as smoking at least once a week at the time of the interview.

2.2.2. Alcohol use and disorders—Participants' lifetime alcohol use was assessed as part of a mental health screener that determined eligibility for completing full psychiatric diagnostic modules. Participants responded whether they had ever consumed a total of 12 drinks in a given year. Participants who had never drunk at least 12 drinks in one year were not asked further details about their drinking. Those who had drunk 12 or more drinks in a year reported on whether they had ever drunk 4+ drinks (for women) or 6+ drinks (for men) on a given day at least two times in one year (i.e., repeated heavy drinking). In addition, they reported on their quantity and frequency of drinking in the past 12 months and in the 12 months of their life in which they were drinking most heavily. From these questions, we were able to determine whether participants had ever drunk on a weekly basis (i.e., regular drinking) and had ever regularly exceeded recommended weekly limits for moderate drinking over the course of a year; these limits are 8 or more drinks per week for women and 15 or more drinks per week for men (National Institute on Alcohol Abuse and Alcoholism, 1995). Therefore, for each individual, we had 4 indices of lifetime drinking: ever 12+ drinks in one year, ever repeated heavy drinking, ever weekly drinking, and ever excessive weekly drinking.

The lifetime occurrence of the four DSM-IV symptoms of alcohol abuse and the seven symptoms of alcohol dependence were assessed with a slightly modified version of the alcohol

use disorder module of the CIDI. Participants completed the module if they reported ever having drunk heavily repeatedly or ever having drunk monthly with an average of 3 or more drinks per occasion, or reported that they or other people ever thought that they had a problem with drinking. The module differed from the CIDI in the following ways: (1) dependence symptoms were assessed regardless of responses to abuse symptoms, (2) withdrawal symptoms were assessed individually, and (3) withdrawal was only coded as present if at least two symptoms were endorsed, consistent with DSM-IV. Due to slight changes in the screening module over time and interviewer errors in using skip patterns, there were some missing data for all items considered. We chose to include only those participants who had valid data on at least two-thirds of the items examined, leaving a sample of 1,508.

Those excluded from the sample due to missing alcohol data ($n = 117$; 7.2%) did not differ significantly from those included on age, gender, race/ethnicity, education or likelihood of having initiated smoking. They were significantly less likely to have ever been a daily smoker (45.7% vs. 55.8%), to have ever been tobacco dependent (27.2% vs. 39.9%), and to be a current smoker (21.4% vs. 31.4%) compared to those included, $ps < .05$.

2.3. Data Analysis Plan

2.3.1. Rasch model analyses—We conducted Rasch model analyses of the four lifetime drinking variables, four abuse symptoms, and seven dependence symptoms using the BIGSTEPS software package (Linacre and Wright, 1998). The Rasch model is a single parameter logistic item response in which the odds of an individual endorsing a given item is modeled as a function of the individual's overall level of problem severity and the severity of that item (Wright and Masters, 1982). In this model, item severity is defined by the point on a latent severity continuum at which the item has a 50% likelihood of being endorsed. The Rasch model constrains items to have equal discrimination. An advantage of this constraint is that if items fit a Rasch model well, the scale can be assumed to be additive across the full range of the continuum such that endorsing one additional item indicates a relatively equal increase in severity regardless of which other items are endorsed. Because of this model property, raw scores can be directly linked to the probability of endorsing each item. Therefore, when certain regions of the alcohol involvement continuum show particularly strong associations with smoking, we can characterize the nature of the alcohol involvement items that are likely to be expressed along that region.

The first assumption of the Rasch model is that item responses are a function of individual variation along a *single* underlying dimension, an assumption we tested using principal components analysis. The second assumption is that responses to a given item are independent from responses to other items (i.e., locally independent). Local independence is satisfied if after the removal of the contribution of a single latent dimension to item responses, the item residuals are not correlated with each other (Wright, 1996). This assumption was tested through principal components analysis of these residuals (Smith and Miao, 1994). Factors from residual analysis accounting for >1.5 units of variance are considered significant (Linacre, 1998; Smith and Miao, 1994), and the absence of significant factors in the residuals supports the assumption of local independence.

To determine how well the data fit the Rasch model, we examined infit and outfit statistics for each item (Wright and Masters, 1982). When data fit the Rasch model well, fit statistics will fall within an acceptable range of 0.6-1.4 (Linacre and Wright, 1994). Because infit statistics are weighted locally, they are less susceptible to outlier influences than outfit statistics and generally preferred (Bond and Fox, 2001). Therefore, we eliminated those items whose infit statistics fell outside of the acceptable range but retained those items with acceptable infit but poor outfit statistics.

After conducting Rasch model analyses in the full sample, we examined differential item functioning (DIF) across demographic subgroups to determine whether the alcohol involvement items showed any bias by demographic group. The estimation of DIF involves comparing analyses conducted separately within each demographic group (Holland and Wainer, 1993). If items behave similarly across groups, then severity parameters estimated independently in different samples will fall within an acceptable range of agreement (e.g. 95% confidence interval). In this study, we compared (a) women to men and (b) non-Hispanic Whites to other racial/ethnic groups. Given the relatively large sample sizes involved, differences in severity estimates between groups can be significant statistically but not be of sufficient magnitude to affect the interpretation of scores across groups. Following our previous work (Kahler et al., 2004), we considered differences greater than 1.5 logits to be clinically meaningful, equal to about 1/2 of the sample standard deviation of item severities, i.e., a medium effect size. We eliminated items showing significant DIF of this magnitude and refit the model using those items without meaningful DIF.

2.3.2. Relating alcohol involvement to smoking—After deriving a summative index of lifetime alcohol involvement, we related this index to initiation, progression, and persistence of smoking using Generalized Estimating Equations (GEE), an analytic method that extends logistic regression to account for the nonindependence in our data due to the inclusion of siblings. Within-sibling correlation was modeled using an exchangeable correlation matrix. The odds of each smoking outcomes was modeled as a function of both a linear and a quadratic effect of lifetime alcohol involvement. Involvement was median-centered prior to analysis to minimize correlation between the linear and quadratic involvement terms. In these GEE models, we also controlled for the effects of age, gender and race/ethnicity because we considered such demographic characteristics to be potential confounding variables that might contribute to either smoking, alcohol use, or both.

3. Results

Table 1 shows the 15 lifetime alcohol involvement items arranged in descending order by their frequency of endorsement. All indices of lifetime drinking were more commonly endorsed than the AUD symptoms. There was a broad range of frequency of endorsement across items from 88.6% (ever drank 12+ drinks in one year) to 5.1% (reduced/gave up important or pleasurable activities due to drinking). Principal components analysis of the tetrachoric correlations among the 15 alcohol involvement items provided very strong support for a dominant single component accounting for variability in the item set. The first component had an eigenvalue of 10.8 and accounted for 77.0% of the variance in the items. The second component had an eigenvalue of only 0.87 and accounted for only 6.2% of item variance. The lowest loading on the first factor was .75 for repeated alcohol-related arrests. All other items had loadings between .80 and .96.

3.1. Rasch Model of The Lifetime Alcohol Involvement Continuum

We fit the Rasch model using all alcohol involvement items other than item 1, which assessed drinking 12+ drinks in one year. This item could not produce meaningful Rasch model estimates because all other items were entirely dependent on it. Specifically, individuals who did not endorse this item were assigned a value of 0 for all other items. Therefore, it would not be meaningful to model the odds of endorsing other items conditional on having endorsed or not endorsed the 12+ drinks item.

The initial Rasch model produced an unacceptably high infit value of 1.56 for item 14 regarding repeated alcohol-related arrests. This result suggests that an overly large proportion of individuals endorsed items of similar severity without endorsing this item. This item was

therefore dropped, and the model was fit again. The 13 remaining items fit a Rasch model well with infit values ranging from 0.76 to 1.21 well within the target range of .60 to 1.40 (Linacre and Wright, 1994). Principal components analysis of residual variance after fitting the Rasch model to the 13 items supported the unidimensionality and local independence of this set with the first two residual factors accounting for only 1.18 and 1.12 units of variance, well below significance. The outfit value for weekly drinking was very high (item 2, outfit = 9.90), but the infit value was acceptable (1.21). Examination of response patterns for those endorsing vs. not endorsing this item indicated that the high outfit value may have been due to the fact that those who did not endorse weekly drinking also did not endorse heavy weekly drinking (item 4; 0%) but did endorse other items of similar severity such as drinking larger amounts or longer than intended (item 5; 7.6%). We considered these response patterns of limited significance and therefore retained item 2 given that the more preferred infit statistic was acceptable.

We next compared severity estimates obtained in men to those obtained in women to determine whether items functioned similarly across genders. There were five items that showed significant gender DIF (i.e., logit differences > 95% CI). Repeated heavy drinking, exceeding moderate drinking criteria, and the larger/longer dependence criterion (items 3, 4, and 5) were significantly more severe symptoms for men compared to women with logit values for men being .94, .64, and .79 greater than those for women. On the other hand, severity estimate differences for withdrawal (item 10; logit difference = -.64) and reducing/giving up activities (item 15; -1.94) indicated that these symptoms were significantly less severe for men than for women. However, only the DIF for item 15 exceeded our a priori criterion of $1/2 SD$ of the item severity estimates. Therefore this item was eliminated and the others retained. None of the items showed significant DIF in the comparison between Whites and non-Whites.

Having eliminated item 15, we re-ran a final Rasch model. Table 1 presents each retained item's severity estimate and standard error along with infit and outfit statistics from this final model. The severity estimates in a Rasch analysis are expressed in equal interval logit (i.e., log odds) units, and indicate the region along the latent alcohol involvement continuum where that individual item is making discriminations. The estimates of each item's severity is standardized so that the average severity of the items is 0. Items covered a broad range of severity ranging from -6.52 to 2.67 ($M = 0.0$; $SD = 2.86$). There was one particularly notable gap in severity estimates between adjacent items. This gap occurred between repeated heavy drinking (severity = -4.83) and excessive weekly drinking (severity = -1.22). The gap indicates that this region of the continuum is mapped relatively less precisely than the higher regions of the continuum where inter-item distances tend to be much smaller. There are also an overabundance of items with relatively high and very similar severity estimates, particularly items 10-13.

3.1.1. Rasch score and item severity—Based on the Rasch model analysis results, we created a total alcohol involvement score by summing the 12 items that fit the model well along with item 1 (12+ drinks in one year) resulting in a 0 to 13 scale. For those with some missing data ($n = 183$; 12.1%), we took the mean of the available items, multiplied by 13, and rounded to the nearest whole number. This set of items was internally consistent (Cronbach's $\alpha = .89$) with no items detracting from alpha. In this scale, a score of 0 indicates never drinking 12+ drinks in a year and a score of 1 indicates drinking 12+ drinks but never endorsing any other alcohol involvement item. Scores greater than 1 indicate progressively greater levels of alcohol involvement. The frequencies and cumulative frequencies of each raw score are presented in Table 2 along with their estimated severity based on Rasch model analyses. The distance between a raw score's severity estimate and an item severity estimate indicates the likelihood of a given item being endorsed at a given raw score. For example, a raw score of 3 (severity estimate = -3.73) is more severe than item 3 (repeated heavy drinking) by 1.10 logits units. At a score of 3, participants have a 75% probability of endorsing item 3. By contrast, the probability endorsing drinking despite social problems (item 6) is only 4.3%.

3.2. Relation of Lifetime Alcohol Involvement to Smoking

Given the over-representation of severe alcohol involvement items, the distribution of the alcohol involvement continuum variable had a long right tail (i.e., there were 4 possible scores below the median and mode of 4, and 9 possible scores above the median), with increasingly sparse population of values beyond 7. To reduce skewness and create larger cell sizes for each level of severity when examining proportions¹, we created a 0 to 10 scale by collapsing scores of 8 and 9 into a score of 8 ($n = 101$) and scores of 10, 11, and 12 into a 9 ($n = 89$). There were 48 participants with a score of 13 which we recoded as a value of 10; we did not collapse this category with any other because these participants endorsed all possible items and thus represent the maximal possible severity that we were capable of measuring. Rates of lifetime alcohol dependence were 0% in the score ranges of 0 to 5, 3.4% at a score of 6, 14.6% at 7, 41.6% at 8, 84.3% at 9, and 100% at 10. Thus, a score of 8 indicates possible alcohol dependence, 9 indicates very likely alcohol dependence, and 10 indicates severe alcohol dependence.

Figure 1 presents for each score on the 0 to 10 alcohol involvement scale (a) the percentage of participants in the sample who had ever initiated smoking, (b) the percentage of those participants initiating smoking who ever progressed to daily smoking, (c) the percentage of those participants initiating smoking who ever progressed to tobacco dependence, and (d) the percentage of ever daily smokers who persisted in smoking at the time of the interview.

3.2.1. Initiation—The percentage of those who had ever puffed increased rapidly as alcohol involvement increased from 0 to 4 and then stabilized at around 95% or higher across the upper half of the continuum. We ran a GEE analysis with ever puffed as the dependent variable and the linear effect of alcohol involvement (median centered by subtracting 4 from the total) and alcohol involvement squared as independent variables. Age, gender and race (white vs. non-white) were included as covariates. As expected, there was a significant positive linear effect for alcohol involvement (odds ratio [OR] = 1.40, 95% CI = 1.30-1.50, $p < .0001$) and a significantly negative quadratic effect (i.e., $OR < 1.0$; $OR = 0.97$, 95% $CI = 0.95-0.99$, $p = .006$).

3.2.2. Progression to daily smoking and dependence—Among those who had ever initiated smoking, the percentage of those who ever progressed to daily smoking increased steadily across the entire involvement continuum, but showed an apparent increase in slope between a score of 9 and 10. GEE analysis controlling for age, gender and race revealed a significant positive linear effect for alcohol involvement ($OR = 1.18$, 95% $CI = 1.13-1.24$, $p < .0001$) and a significantly positive quadratic trend ($OR = 1.02$, 95% $CI = 1.01-1.04$, $p = .007$), suggesting that the odds of daily smoking accelerated significantly across the upper ranges of the continuum. Tobacco dependence among those who had puffed ($n = 1,329$) showed a highly similar association with alcohol involvement for both linear ($OR = 1.21$, 95% $CI = 1.15-1.27$, $p < .0001$) and quadratic ($OR = 1.02$, 95% $CI = 1.01-1.04$, $p = .002$) effects.

3.2.3. Persistence of smoking—Finally, among those who had ever smoked daily ($n = 830$), the proportion who were still smoking at the time of the interview showed a less clear relationship to alcohol involvement with the highest proportions at the lowest and the highest

¹When categories were not collapsed, the skewness and kurtosis of the quadratic term of the 0-13 scale became high at 2.03 and 3.55, respectively. By contrast, these values were 1.36 and 1.43 when the scale was reduced to range from 0-10. GEE analyses using the 0-13 scale yielded similar results as those using the 0-10 scale, but the positive skewness of the 0-13 scale led to stronger negative quadratic and weaker positive quadratic effects. Furthermore, the estimates for proportions of each smoking outcome at each scale score had relatively low precision when rare categories were not combined. For example, the 95% margin of error with a cell size of 100 is below $\pm 10\%$ for any given proportion. This increases to $\pm 14\%$ for cell sizes of 50 and $\pm 20\%$ for cell sizes of 25. Combining scores to produce larger cell sizes allows for proportions to be estimated with more adequate precision.

scores on the continuum. GEE analysis controlling for age, gender and race revealed a nonsignificant negative linear effect for alcohol involvement ($OR = .95$, 95% $CI = 0.89-1.01$, $p = .08$) and a significant positive quadratic trend ($OR = 1.02$, 95% $CI = 1.01-1.04$, $p = .009$). Thus, the odds of continued smoking did not significantly increase across the alcohol involvement continuum as a whole. Rather, it decreased across the low ranges of the continuum, increased across the highest ranges, and was essentially flat across most of the continuum.

4. Discussion

Results of this study indicate that a measure of lifetime alcohol involvement with adequate psychometric properties can be constructed by combining indices of lifetime drinking and symptoms of alcohol abuse and dependence. These results extend those of Krueger et al. (2004) and Saha et al. (2007) by showing that, in addition to episodic heavy drinking, both weekly drinking and regularly drinking more than the NIAAA recommended weekly limit can be used to map the less severe regions of the alcohol involvement continuum. In this sample, the chosen indices of alcohol use all were less severe than diagnostic symptoms of alcohol abuse and dependence. Three of the abuse symptoms (hazardous use, interference with work or home, and social problems) appeared to be effective in making discriminations among the mild to moderate range of alcohol problem severity in this sample. However, as has been shown in other item response analyses of DSM-IV AUD criteria (Kahler and Strong, 2006; Krueger et al., 2004; Langenbucher et al., 2004; Proudfoot et al., 2006), abuse symptoms were not consistently less severe than dependence symptoms. Among the dependence symptoms, the larger/longer criterion appeared to be a particularly effective item for discriminating between those with heavy drinking and no AUD symptoms and those with at least some AUD symptoms. This is consistent with population-based studies in the U.S. (Saha et al., 2007) and Australia (Proudfoot et al., 2006).

The measure of lifetime alcohol involvement we constructed showed significant relationships with smoking initiation, progression to daily smoking, progression to dependence, and persistence of smoking in middle adulthood. Given the wealth of studies demonstrating significant alcohol-smoking associations, these results are not surprising. However, the continuous measure of lifetime alcohol involvement enabled us to depict associations between alcohol involvement and smoking in ways that traditional analytic techniques can not accomplish. For example, results indicated that there were regions of the alcohol involvement continuum which were associated more strongly with increased prevalence of specific smoking outcomes and other regions in which greater alcohol involvement showed a weak association with increased smoking. These findings are reviewed below.

4.1. Smoking initiation

As we hypothesized, the odds of ever trying cigarettes increased significantly with greater alcohol involvement, but this association was most notable in the lower regions of the alcohol involvement continuum. Increases in the odds of smoking initiation were essentially absent beyond an alcohol involvement score of 4 on our 0 to 10 scale, at which point the proportion smoking reached an asymptote of just over 95%. At a score of 4, individuals were very likely to have been weekly drinkers and to have drunk heavily repeatedly and may have exceeded moderate drinking levels for a year or more, but they were very unlikely to have shown more than one symptom of alcohol abuse or dependence. The steepest rise in rates of smoking initiation was observed between scores of 0 and 2, the region in the continuum which reflects going from never drinking, to drinking 12+ drinks in a year, to drinking on a weekly basis.

These results suggest there are factors that contribute strongly both to smoking initiation and to initiation of occasional and more regular, moderate drinking. Given how common puffing a cigarette and drinking on a weekly basis were in this sample, it is likely that these results

reflect protective factors operating to insulate some individuals from initial use of either substance. These protective factors likely operate during adolescence and young adulthood when initiation of use of both alcohol and tobacco is most common. Certain environmental influences during adolescence such as peers, parents, and religious beliefs, may limit both exposure to alcohol and exposure to tobacco during this time in life (Cohen et al., 1994; Flay et al., 1994; Flay et al., 1999). However, longitudinal studies also have shown that there is a bidirectional influence between smoking and alcohol initiation in adolescence that appears to be independent of common potential third factors (Jackson et al., 2002; Wetzels et al., 2003).

4.2. Progression to daily smoking and dependence

The associations between alcohol involvement and both daily smoking and tobacco dependence among those who had ever tried smoking were remarkably similar. Even in the absence of significant symptoms of alcohol dependence, there were very strong positive linear relationships between greater alcohol involvement and increased odds of progression to daily smoking and tobacco dependence. As shown in Figure 1, the prevalence of daily smoking and tobacco dependence increased steadily from an alcohol involvement score of 0 to a score of 7 where most symptoms of alcohol abuse and dependence would still be rare, but at least some would be expressed. However, there also was a positive quadratic effect indicating that the odds of daily smoking increased more rapidly with higher levels of alcohol involvement. In the top 10% of the sample (alcohol involvement scores of 9-10), where most individuals would be moderately or severely dependent on alcohol, daily smoking and tobacco dependence increased especially rapidly. For those 48 participants who endorsed all alcohol involvement items, the proportion who ever were daily smokers or tobacco dependent was very high at 97.9% and 89.6%, respectively.

The association between heavy alcohol use and abuse and daily smoking and tobacco dependence may reflect, in part, environmental or dispositional factors that contribute to both. Also, given that alcohol appears to enhance the rewarding effects of nicotine (Glautier et al., 1996; Rose et al., 2002, 2004) and nicotine appears to enhance the rewarding effects of alcohol (Kouri et al., 2004; Perkins et al., 1995), the association between increasing levels of tobacco and alcohol use may reflect bidirectional influences in which the use of one substance makes use of the other substance more rewarding and therefore more likely to progress to greater use. Prior longitudinal research with adolescents and young adults has found that greater alcohol use prospectively predicts greater tobacco use and vice versa (Jackson et al, 2002).

The marked increase in daily smoking and tobacco dependence among those at the highest levels of alcohol involvement is of potential importance. Population studies have found elevated rates of smoking in those with current alcohol dependence (Dawson, 2000; Grant, 1998; Lasser et al., 2000), even when compared to those with current alcohol abuse (Falk et al., 2006). However, even higher rates of smoking have been reported in individuals seeking treatment for alcohol dependence (DiFranza and Guerrero, 1990; Joseph et al., 1990). In the present study, our use of an additive alcohol involvement index allowed for discrimination between levels of alcohol involvement among those who had ever been alcohol dependent. Our results suggest that the especially high rates of smoking found in substance abuse treatment centers is likely to be due to the very high levels of alcohol involvement seen in these populations. Indeed, even among those in alcohol dependence treatment, level of alcohol dependence has been correlated significantly with greater nicotine dependence (Monti et al., 1995). It appears that those who are vulnerable to progressing to levels of alcohol involvement where severe symptoms of alcohol dependence are observed, also are especially vulnerable to smoking and tobacco dependence. Such vulnerability may reflect, for example, common genetic contributions to dependence on both substances (Enoch et al., 2006; Liu et al., 2005; Schinka et al., 2002).

4.3. Persistence of smoking

In contrast to results obtained for smoking initiation and progression, the odds of smoking persistence neither increased nor decreased reliably with greater alcohol involvement, which may explain some of the inconsistency observed in prior studies relating alcohol dependence history to smoking cessation (Hughes and Kalman, 2006). Our results suggest that this inconsistency may be due to a curvilinear relationship between alcohol involvement and persistence of smoking. A significant quadratic effect suggested those at either ends of the alcohol involvement continuum were at greater risk of persisting in smoking. Thus, in any particular study, the relationship observed between a history of alcohol dependence and smoking cessation may depend, in part, on the proportion of participants who have never had significant alcohol involvement relative to those who have ever been alcohol dependent.

Those who have had only minimal alcohol involvement are much less likely to try smoking and if they do try it, they are less likely to progress to daily smoking. It may be that those who progress to daily smoking despite the protective factors associated with low alcohol involvement may be those who find smoking especially rewarding in their initial experiences. That is, despite potentially being more isolated from environments and other behaviors that facilitate smoking, these individuals went on to smoke regularly and continued to do so. At the high ends of alcohol involvement there also appeared to be an increase in current smoking, although this increase was not entirely consistent (for example, those with a lifetime alcohol involvement score of 10 showed a nonsignificantly lower rate of current smoking than those with a score of 9). That a high proportion of participants at the highest levels of alcohol involvement initiated smoking, progressed to daily smoking, and persisted in smoking speaks to the importance of further study of factors that contribute to both smoking and alcohol dependence.

4.4. Limitations

The results of this study must be understood in light of some limitations in sample and methodology. The size of the sample was relatively large and certainly adequate for conducting item response analyses and testing linear and quadratic effects in logistic GEE models. However, when we divided the sample into discrete scores representing levels of lifetime alcohol involvement, the sample sizes became overly small for certain scores, and we had to collapse some categories. This problem was compounded when we restricted our sample further for certain analyses, such as examining current smoking only among those who had smoked daily.

The sample also was limited to persons aged 34-44 from one region of the country. The relative homogeneity of ages at the time of the interview was useful given our focus on lifetime smoking and alcohol involvement and given that participants were likely to have passed through the ages in life in which risk for use and dependence on these substances is most elevated. Although we controlled for possible effects of age, we can not rule out the possibility that certain associations we found might be different in other age cohorts in which prevailing social attitudes around both drinking and smoking may have been quite different. Finally, for analyses of persistence of smoking, the age of study participants precluded examination of individuals who might have stopped smoking later in life (i.e., after the age of 44).

The measure we constructed to assess the lifetime alcohol involvement continuum allowed us to demonstrate relationships between smoking and lifetime alcohol involvement along the region of the continuum in which symptoms of AUDs were very unlikely to be endorsed. At the same time, our measure of lifetime alcohol involvement was imperfect. First, some items were dependent on others. Specifically, only individuals who drank at least 12+ drinks in a year were queried about other alcohol items. Second, there was a large gap in coverage between

repeated heavy drinking and exceeding moderate drinking criteria. An index that includes additional measures of low severity alcohol consequences, such as hangovers and minor social embarrassments (Kahler et al., 2005), may be able to map more precisely the bottom half of the lifetime alcohol involvement continuum where item coverage was sparse. At the same time, rates of initiation and progression of smoking showed consistent, linear increases across the bottom half of the alcohol involvement continuum, and it is not clear that more precise mapping of this region would produce different results or added insights.

4.5. Conclusions

This study provides, to our knowledge, the most detailed depiction to date of the relationship between lifetime alcohol involvement and smoking in a community sample, as well as a methodology for examining these relationships in future studies. Using this methodology to examine the alcohol-smoking relationship in countries other than the United States would be highly valuable. Although studies have shown that alcohol use and smoking also frequently co-occur in countries outside of the US (e.g. Burger et al., 2004; Chiolerio et al., 2006; Clausen et al., 2006), detailed examination of these relationships is still needed. National and cultural differences in social practices, norms, and policies regarding alcohol use and smoking may play a significant role in the way that the use of these two substances relate. For example, whereas alcohol use predicted tobacco use more strongly than the converse in a US sample (Jackson et al., 2002), tobacco use predicted alcohol use more strongly than the converse in a number of European countries (Wetzels et al., 2003). Methods similar to those used here could be highly informative regarding how alcohol-smoking relations differ across national and cultural contexts.

A necessary next step in this research is to examine variables that may account for the association between alcohol involvement and initiation, progression, and persistence of smoking. Our results suggest that it may be most productive to focus on specific regions of the alcohol involvement continuum depending on the particular aspect of smoking that is examined. For example, certain genes, epigenetic factors or gene-environment interactions may predispose individuals to severe alcohol dependence. These factors might show a gradual rise in prevalence over the alcohol involvement continuum but a sharper rise at the high ends. Investigation of risk factors that show such nonlinear associations with an ordinal alcohol involvement index could be identified using quadratic cumulative odds regression models (Scharfstein et al., 2001). Such factors might account for the marked increase in tobacco dependence that emerges in the highest regions of the alcohol involvement continuum. Logistic regression models, such as the ones used in this study, ultimately may be used to test what factors mediate the relationship between alcohol involvement and smoking within specific regions of the alcohol involvement continuum. These mediators, in turn, may prove useful as targets for prevention and treatment efforts to address the high costs associated with combined smoking and alcohol use.

References

- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. Vol. 4th ed.. Author; Washington, DC: 1994.
- Anthony JC, Echeagaray-Wagner F. Epidemiologic analysis of alcohol and tobacco use. *Alcohol Res Health* 2000;24:201–208. [PubMed: 15986714]
- Bond, TG.; Fox, CM. Applying the Rasch Model: Fundamental measurement in the human sciences. Erlbaum; Mahway, NJ: 2001.
- Broman, S. The Collaborative Perinatal Project: an Overview. In: Mednick, SA.; Harway, M.; Finello, KM., editors. *Handbook of Longitudinal Research*. Praeger; New York: 1984. p. 185-215.
- Burger M, Mensink G, Bronstrup A, Thierfelder W, Pietrzik K. Alcohol consumption and its relation to cardiovascular risk factors in Germany. *Eur J Clin Nutr* 2004;58:605–614. [PubMed: 15042128]

- Carmelli D, Swan GE, Robinette D. The relationship between quitting smoking and changes in drinking in World War II veteran twins. *J Subst Abuse* 1993;5:103–116. [PubMed: 8400834]
- Chiolero A, Wietlisbach V, Ruffieux C, Paccaud F, Cornuz J. Clustering of risk behaviors with cigarette consumption: A population-based survey. *Prev Med* 2006;42:348–353. [PubMed: 16504277]
- Clausen T, Charlton KE, Holmboe-Ottesen G. Nutritional status, tobacco use and alcohol consumption of older persons in Botswana. *The journal of nutrition, health & aging* 2006;10:104–110.
- Cohen DA, Richardson J, LaBree L. Parenting behaviors and the onset of smoking and alcohol use: A longitudinal study. *Pediatrics* 1994;94:368–375. [PubMed: 8065865]
- Dawson DA. Drinking as a risk factor for sustained smoking. *Drug Alcohol Depend* 2000;59:235–249. [PubMed: 10812284]
- Dierker LC, Donny E, Tiffany S, Colby SM, Perrine N, Clayton RR. The association between cigarette smoking and DSM-IV nicotine dependence among first year college students. *Drug Alcohol Depend* 2007;86:106–114. [PubMed: 16837142]
- DiFranza JR, Guerrera MP. Alcoholism and smoking. *Journal of Studies on Alcohol* 1990;51:130–135. [PubMed: 2308350]
- Enoch MA, Waheed JF, Harris CR, Albaugh B, Goldman D. Sex differences in the influence of COMT Val158Met on alcoholism and smoking in plains American Indians. *Alcohol Clin Exp Res* 2006;30:399–406. [PubMed: 16499480]
- Falk DE, Yi HY, Hiller-Sturmhofel S. An epidemiologic analysis of co-occurring alcohol and tobacco use and disorders: findings from the National Epidemiologic Survey on Alcohol and Related Conditions. *Alcohol Res Health* 2006;29:162–171. [PubMed: 17373404]
- Flay BR, Hu FB, Siddiqui O, Day LE, Hedeker D, Petraitis J, Richardson J, Sussman S. Differential influence of parental smoking and friends' smoking on adolescent initiation and escalation of smoking. *Journal of Health and Social Behavior* 1994;35:248–265. [PubMed: 7983337]
- Flay BR, Petraitis J, Hu FB. Psychosocial risk and protective factors for adolescent tobacco use. *Nicotine Tob Res* 1999;1(Suppl 1):S59–65. [PubMed: 11072406]
- Friedman GD, Tekawa I, Klatsky AL, Sidney S, Armstrong MA. Alcohol drinking and cigarette smoking: An exploration of the association in middle-aged men and women. *Drug Alcohol Depend* 1991;27:283–290. [PubMed: 1884670]
- Glaetzer S, Clements K, White JA, Taylor C, Stolerman IP. Alcohol and the reward value of cigarette smoking. *Behav Pharmacol* 1996;7:144–154. [PubMed: 11224406]
- Grant BF. Age at smoking onset and its association with alcohol consumption and DSM-IV alcohol abuse and dependence: Results from the national longitudinal alcohol epidemiologic survey. *J Subst Abuse* 1998;10:59–73. [PubMed: 9720007]
- Gruzca RA, Bierut LJ. Cigarette smoking and the risk for alcohol use disorders among adolescent drinkers. *Alcohol Clin Exp Res* 2006;30:2046–2054. [PubMed: 17117970]
- Holland, PW.; Wainer, H. *Differential Item Functioning*. Lawrence Erlbaum; Hillsdale, NJ: 1993.
- Hughes JR, Kalman D. Do smokers with alcohol problems have more difficulty quitting? *Drug Alcohol Depend* 2006;82:91–102. [PubMed: 16188401]
- Hymowitz N, Cummings KM, Hyland A, Lynn WR, Pechacek TF, Hartwell TD. Predictors of smoking cessation in a cohort of adult smokers followed for five years. *Tob Control* 1997;6:S57–62. [PubMed: 9583654]
- Jackson KM, Sher KJ, Cooper ML, Wood PK. Adolescent alcohol and tobacco use: onset, persistence and trajectories of use across two samples. *Addiction* 2002;97:517–531. [PubMed: 12033653]
- Joseph AM, Nichol KL, Willenbring ML, Korn JE, Lysaght LS. Beneficial effects of treatment of nicotine dependence during an inpatient substance abuse treatment program. *Jama* 1990;263:3043–3046. [PubMed: 2342215]
- Kahler CW, Strong DR. A Rasch model analysis of DSM-IV alcohol abuse and dependence items in the National Epidemiological Survey on Alcohol and Related Conditions. *Alcohol Clin Exp Res* 2006;30:1165–1175. [PubMed: 16792564]
- Kahler CW, Strong DR, Read JP. Toward efficient and comprehensive measurement of the alcohol problems continuum in college students: the brief young adult alcohol consequences questionnaire. *Alcohol Clin Exp Res* 2005;29:1180–1189. [PubMed: 16046873]

- Kahler CW, Strong DR, Read JP, Wood MD, Palfai T. Mapping the continuum of alcohol problems in college students: A Rasch model analysis. *Psychol Addict Behav* 2004;18:322–333. [PubMed: 15631604]
- Kahler CW, Strong DR, Stuart GL, Moore TM, Ramsey SE. Item functioning of the alcohol dependence scale in a high-risk sample. *Drug Alcohol Depend* 2003;72:183–192. [PubMed: 14636973]
- Kouri EM, McCarthy EM, Faust AH, Lukas SE. Pretreatment with transdermal nicotine enhances some of ethanol's acute effects in men. *Drug Alcohol Depend* 2004;75:55–65. [PubMed: 15225889]
- Krueger RF, Nichol PE, Hicks BM, Markon KE, Patrick CJ, Lacono WG, McGue M. Using latent trait modeling to conceptualize an alcohol problems continuum. *Psychol Assess* 2004;16:107–119. [PubMed: 15222807]
- Langenbucher JW, Labouvie E, Martin CS, Sanjuan PM, Bavly L, Kirisci L, Chung T. An application of item response theory analysis to alcohol, cannabis, and cocaine criteria in DSM-IV. *J Abnorm Psychol* 2004;113:72–80. [PubMed: 14992659]
- Lasser K, Boyd JW, Woolhandler S, Himmelstein DU, McCormick D, Bor DH. Smoking and mental illness: A population-based prevalence study. *Jama* 2000;284:2606–2610. [PubMed: 11086367]
- Linacre JM. Detecting multidimensionality: Which residual data-type works best. *Journal of Outcome Measurement* 1998;2:266–283. [PubMed: 9711024]
- Linacre JM, Wright BD. Reasonable mean-square fit values. *Rasch Measurement Transactions* 1994;8:370.
- Linacre, JM.; Wright, BD. A User's Guide to BIGSTEPS: A Rasch-Model Computer Program. MESA Press; Chicago, ILL: 1998.
- Liu Y, Yoshimura K, Hanaoka T, Ohnami S, Kohno T, Yoshida T, Sakamoto H, Sobue T, Tsugane S. Association of habitual smoking and drinking with single nucleotide polymorphism (SNP) in 40 candidate genes: data from random population-based Japanese samples. *J Hum Genet* 2005;50:62–68. [PubMed: 15654505]
- Monti, PM.; Rohsenow, DJ.; Colby, SM.; Abrams, DB. Smoking among alcoholics during and after treatment: Implications for models, strategies, and policy. In: Fertig, JB.; Allen, JP., editors. *Alcohol and Tobacco: From Basic Science to Clinical Practice*. National Institutes of Health; Bethesda, MD: 1995. p. 187-206.
- Murray RP, Istvan JA, Voelker HT, Rigdon MA, Wallace MD. Level of involvement with alcohol and success at smoking cessation in the lung health study. *J Stud Alcohol* 1995;56:74–82. [PubMed: 7752637]
- National Institute on Alcohol Abuse and Alcoholism. The physicians' guide to helping patients with alcohol problems. National Institutes of Health; 1995.
- Niswander, KR.; Gordon, M. The women and their pregnancies: the Collaborative Perinatal Study of the National Institute of Neurological Diseases and Stroke. National Institutes of Health; Washington: 1972.
- Osler M, Prescott E, Godtfredsen N, Hein HO, Schnohr P. Gender and determinants of smoking cessation: A longitudinal study. *Prev Med* 1999;29:57–62. [PubMed: 10419801]
- Pelucchi C, Gallus S, Garavello W, Bosetti C, La Vecchia C. Cancer risk associated with alcohol and tobacco use: focus on upper aero-digestive tract and liver. *Alcohol Res Health* 2006;29:193–198. [PubMed: 17373408]
- Perkins KA, Sexton JE, DiMarco A, Grobe JE, Scierka A, Stiller RL. Subjective and cardiovascular responses to nicotine combined with alcohol in male and female smokers. *Psychopharmacology (Berl)* 1995;119:205–212. [PubMed: 7659768]
- Proudfoot H, Baillie AJ, Teesson M. The structure of alcohol dependence in the community. *Drug Alcohol Depend* 2006;81:21–26. [PubMed: 16005578]
- Rasch, G. Probabilistic models for some intelligence and attainment test. Denmark's Paedagogiske Institut; Copenhagen: 1960.
- Rose JE, Brauer LH, Behm FM, Cramblett M, Calkins K, Lawhon D. Potentiation of nicotine reward by alcohol. *Alcohol Clin Exp Res* 2002;26:1930–1931. [PubMed: 12500124]
- Rose JE, Brauer LH, Behm FM, Cramblett M, Calkins K, Lawhon D. Psychopharmacological interactions between nicotine and ethanol. *Nicotine Tob Res* 2004;6:133–144. [PubMed: 14982697]

- Saha TD, Stinson FS, Grant BF. The role of alcohol consumption in future classifications of alcohol use disorders. *Drug Alcohol Depend* 2007;89:82–92. [PubMed: 17240085]
- Scharfstein, DO.; Liang, KY.; Eaton, W.; Chen, LS. Biostatistics. Vol. 2. Oxford; England: 2001. The quadratic cumulative odds regression model for scored ordinal outcomes: application to alcohol dependence; p. 473–483.
- Schinka JA, Town T, Abdullah L, Crawford FC, Ordorica PI, Francis E, Hughes P, Graves AB, Mortimer JA, Mullan M. A functional polymorphism within the muopioid receptor gene and risk for abuse of alcohol and other substances. *Mol Psychiatry* 2002;7:224–228. [PubMed: 11840318]
- Smith, RM.; Miao, CY. Assessing unidimensionality for the Rasch measurement. In: Wilson, M., editor. *Objective Measurement: Theory and Practice*. Ablex; Norwood, NJ: 1994. p. 316–327.
- Sobell, MB.; Sobell, LC.; Kozlowski, LT. Dual recoveries from alcohol and smoking problems. In: Fertig, JB.; Allen, JP., editors. *Alcohol and Tobacco: From Basic Science to Clinical Practice*. National Institutes of Health; Bethesda, MD: 1995. p. 207–224.
- Sorlie PD, Kannel WB. A description of cigarette smoking cessation and resumption in the Framingham Study. *Prev Med* 1990;19:335–345. [PubMed: 2377595]
- Vander Ark W, DiNardo LJ, Oliver DS. Factors affecting smoking cessation in patients with head and neck cancer. *The Laryngoscope* 1997;107:888–892. [PubMed: 9217125]
- Wetzels JJ, Kremers SP, Vitoria PD, de Vries H. The alcohol-tobacco relationship: a prospective study among adolescents in six European countries. *Addiction* 2003;98:1755–1763. [PubMed: 14651508]
- World Health Organization. Composite International Diagnostic Interview (CIDI), version 1.0. World Health Organization; Geneva: 1990.
- Wright BD. Local dependency, correlations and principal components. *Rasch Measurement Transactions* 1996;10:509–511.
- Wright, BD.; Masters, GN. *Rating scale analysis*. MESA; Chicago: 1982.
- Zimmerman RS, Warheit GJ, Ulbrich PM, Auth JB. The relationship between alcohol use and attempts and success at smoking cessation. *Addict Behav* 1990;15:197–207. [PubMed: 2378280]

Figure 1.

Percentage of participants who initiated smoking, progressed to daily smoking from initiation, progressed to tobacco dependence from initiation, and persisted in smoking. Initiation data were available for 1,506 participants. The percentages of those who progressed to daily smoking and those who progressed to tobacco dependence are based on those participants who had ever initiated smoking and who had valid data, $n_s = 1,340$ and 1,331 respectively. The percentage of those persisting in smoking at the time of the interview is based on those who had ever smoked daily and whose current smoking status was known ($n = 830$).

Table 1
Item endorsement frequency, parameter estimates, fit statistics and item bias estimates for the alcohol involvement items

		%	Severity	SE	Infit	Outfit
1.	Drank 12+ drinks in one year ^d	88.6	---	---	---	---
2.	Drank on a weekly basis for 12 months	75.9	-6.52	.14	1.21	9.90
3.	Drank heavily 2+ times in one year	67.4	-4.83	.11	0.76	1.40
4.	Exceeded moderate weekly drinking criteria	41.2	-1.22	.08	1.07	1.01
5.	Drank larger or longer than intended	38.1	-0.92	.08	0.96	0.86
6.	Drank when physically hazardous	34.9	-0.62	.08	1.13	1.74
7.	Drinking interfered with work or home	16.8	1.39	.09	1.03	0.87
8.	Drank despite social problems	16.3	1.46	.09	0.81	0.43
9.	Tolerance	15.0	1.66	.10	1.12	0.70
10.	2+ alcohol withdrawal symptoms	11.7	2.25	.11	0.88	1.03
11.	Failed attempts to cut down/quit	11.4	2.32	.11	0.84	0.44
12.	Spent great deal of time drinking/recovering	11.2	2.35	.11	0.97	0.98
13.	Drank despite physical/emotional problems	9.8	2.67	.12	0.93	0.37
14.	2+ alcohol-related arrests ^b	7.1	---	---	---	---
15.	Reduced/gave up activities ^c	5.1	---	---	---	---

Note.

^aItem can not be included in the Rasch model analyses as participants not endorsing the item were not asked the remaining items and therefore estimates of its relation to other items are not meaningful.

^bItem showed poor fit in an initial Rasch model analysis and was therefore dropped from the model.

^cItem showed differential item functioning by gender and was therefore dropped from the model.

Table 2

Raw Total Scores on the Sum of the 13 Selected Items, Estimated Severity of the Scores in Logit Units, and Frequencies and Cumulative Frequencies of Scores in the Study Sample

Raw Score	Severity Estimate	S.E.	Frequency (n)	% of sample	Cumulative Frequency (n)	Cumulative % of sample
0 ^a	---	---	172	11.4	172	11.4
1 ^a	---	---	143	9.5	315	20.9
2	-6.23	1.53	168	11.1	483	32.0
3	-3.73	1.63	188	12.5	671	44.5
4	-1.86	1.12	236	15.6	907	60.2
5	-0.80	0.96	148	9.8	1055	70.0
6	0.04	0.89	119	7.9	1174	77.8
7	0.78	0.82	96	6.4	1270	84.2
8	1.41	0.77	50	3.3	1320	87.5
9	1.98	0.75	51	3.4	1371	90.9
10	2.56	0.77	23	1.5	1394	92.4
11	3.20	0.85	31	2.1	1425	94.5
12	4.10	1.09	35	2.3	1460	96.8
13	4.88 (estimated)	1.47	48	3.2	1508	100.00

^aSeverity estimates are not calculated for these raw scores as they could not be modeled in Rasch analyses given that there is no variability in response patterns for either score. All participants with a score of 1 endorse having drunk 12 drinks in one year and no other alcohol involvement items.