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Adolescent alcohol and tobacco use: onset, persistence and trajectories of use across two samples

Kristina M. Jackson, **Kenneth J. Sher**, **M. Lynne Cooper**, and **Phillip K. Wood**
University of Missouri-Columbia, Columbia, MO, USA

Abstract

Aims—We examined the alcohol-tobacco relationship using two prospective, ethnically diverse samples. Trajectories of alcohol and tobacco use are portrayed overall and by sex and ethnicity. Using prospective analyses, we examine directional influences between alcohol and tobacco use, and we characterize initiation versus persistence of drinking and smoking as a function of use of the other substance.

Design, setting—Data were from the National Longitudinal Study of Adolescent Health (AddHealth) and the Adolescent Health Risk Study (AHRS). Follow-up intervals for AddHealth and AHRS were 1 and 5 years, respectively.

Participants—AddHealth respondents (n = 4831) were on average 14.8 years old (48% male, 23% black, 61% white) and AHRS respondents (n = 1814) were on average 16.7 years old (47% male, 44% black, 49% white).

Measurements—Two alcohol consumption variables and two smoking variables were used: drinking frequency and heavy drinking frequency, and regular (current) smoking and daily number of cigarettes.

Findings—Alcohol and tobacco use exhibited monotonic increases over adolescence and young adulthood. Men and white respondents reported more use than women and black respondents. Alcohol and tobacco were moderately associated at both times. Analyses revealed that prior alcohol use predicted tobacco use more strongly than the converse. Initiation of smoking was a function of prior drinking; to a lesser extent, initiation of drinking was a function of prior smoking. Persistence of smoking was a function of prior drinking and persistence of drinking was a function of prior smoking.

Conclusions—Provisional support exists for the claim that alcohol use predicts tobacco use more strongly than the converse. For both drinking and smoking, onset and persistence are predicted by prior use of the other substance, and these associations were robust across sex and ethnicity.

Keywords

Adolescence; alcohol; onset; persistence; prospective; smoking

INTRODUCTION

Research consistently documents an association between alcohol consumption and tobacco use. Social drinkers are more likely to smoke than non-drinkers (Istvan & Matarazzo 1984;

Correspondence to: Kristina M. Jackson, Psychology Department, University of Missouri, 200 S. 7th Street, Columbia, MO 65211-0001, USA, Tel: + 1 573 884 1485, Fax: + 1 573 884 5588, JacksonKM@missouri.edu.

Rohde *et al.* 1995) and alcoholics are more likely to smoke than non-alcoholics (Gulliver *et al.* 1995). Similarly, smokers are more likely to drink than non-smokers (Bien & Burge 1990; DiFranza & Guerrera 1990). The covariation between alcohol and tobacco use transcends sex, age and culture (Istvan & Matarazzo 1984).

Four general classes of theories have been proposed to account for co-occurring alcohol and tobacco use: (1) alcohol use causes tobacco use; (2) tobacco use causes alcohol use; (3) alcohol use and tobacco use influence each other reciprocally, and (4) alcohol and tobacco use are a function of common third variables. Laboratory studies manipulating the use of one substance have demonstrated greater use of the other, particularly increased smoking when alcohol consumption is manipulated (e.g. Mitchell *et al.* 1995; Glautier *et al.* 1996). Additionally, nicotine administration is associated with increased ethanol consumption in animals (Blomqvist *et al.* 1996). Although this evidence supports directional influences between alcohol and tobacco use (and support for both directions suggests a reciprocal, bi-directional model), the extant literature also supports a common vulnerability model. That is, alcohol and tobacco use share important third-variable precursors and hence are likely to co-occur. Several factors have been identified as important predictors of both alcohol and tobacco involvement (although often in separate examinations of each substance), including family history of alcoholism, temperament and personality (e.g. behavioral undercontrol and negative affectivity), socialization factors (e.g. parent and peer modeling), life stress and substance use outcome expectancies (see Jackson *et al.* 2000 for a review of these factors).

Most research describing the alcohol–tobacco relationship has been cross-sectional in nature, and is consequently subject to recall bias. The few existing prospective studies have yielded conflicting results, with some studies showing directional influences (Fleming *et al.* 1989; Simon *et al.* 1995) and others finding no prospective association (Gordon & Doyle 1986; Northwehr *et al.* 1995).

Early drinking onset is a robust predictor of lifetime drinking and the development of alcohol problems and alcohol use disorders (Grant 1998). Similarly, early smoking onset is associated with daily smoking, daily quantity of cigarettes and long smoking careers (Grant 1998; Lando *et al.* 1999), as well as excessive alcohol consumption and alcohol use disorders (Grant 1998). As alcohol and tobacco are generally the first drugs with which adolescents experiment (Shiffman & Balabanis 1995), it is likely that initiation of drinking among non-drinkers will be highly associated with prior smoking (and initiation of smoking among non-smokers will be highly associated with prior drinking).

It seems likely that use of one substance leads to initiation of use of the other substance. However, an alternate research question is whether use of one substance increases the likelihood of maintaining use of the other substance. Different risk factors have been documented for initiation versus persistence in both drinking (e.g. Kilbey *et al.* 1998) and smoking (e.g. Carvajal *et al.* 2000). In addition, multidimensional scaling and behavior genetic analyses suggest that drinking initiation and alcohol (quantity/frequency) consumption are only loosely coupled constructs (e.g. Heath *et al.* 1991a, 1991b). Correspondingly, behavior genetic data suggest that smoking initiation and persistence are largely genetically and environmentally distinct (although they do share some common genetic and environmental variance) (e.g. True *et al.* 1997; Koopmans *et al.* 1999). Given the different mechanisms that underlie initiation and maintenance of alcohol and tobacco use, it is important that both processes be investigated.

In addition, few studies characterizing the nature of alcohol and tobacco comorbidity sample from the general population, and there is an over-reliance on clinical (e.g. Mintz *et al.* 1985; York & Hirsch 1995) and college student populations (e.g. Sher *et al.* 1996), which typically differ from the general population in their base rates of substance use. Also, these non-

representative samples under-represent minority/low socioeconomic status (SES) groups (Kazdin 1993; National Center for Education Statistics 1997) and fail to characterize the alcohol–tobacco relationship early enough in the course of substance use initiation.

Overview

The present study examines alcohol and tobacco use prospectively to explore alternate theories of comorbidity. Data were taken from two studies of adolescents, capturing a time during which initiation of substance use often occurs. The first sample was a nationally representative dataset, the National Longitudinal Study of Adolescent Health (AddHealth). Analyses were replicated using data from the Adolescent Health Risk Study (AHRS). Although both datasets examine roughly the same time period and both oversample minority respondents, AddHealth and AHRS each provide a unique perspective on alcohol–tobacco comorbidity, with different follow-up intervals, populations and age spans. Using both datasets, mean trajectories of alcohol and tobacco use are portrayed for the full sample and are described by sex and ethnicity. In addition, cross-sectional and prospective relationships between alcohol and tobacco use are examined, and, in order to understand the role of one substance upon the other, initiation versus maintenance of behavior (i.e. onset versus regular use) are examined. Finally, the extent to which the association between alcohol and tobacco remained in the presence of etiologically relevant third variables was explored.

METHOD

National Longitudinal Study of Adolescent Health

Data and procedures—Data were taken from the public use subset of the National Longitudinal Study of Adolescent Health (AddHealth; Lang *et al.* 1997) (n = 6504; n = 4831 at year 2). AddHealth focused on the influence of the social environment on adolescent health (e.g. diet and nutrition, depression, exercise, substance use).

The sample was stratified by region, urbanicity, school type and school ethnic mix and size; representative clustered sampling techniques were utilized. In 1994/1995, 20 745 students in grades 7–12 completed a 45–60 minute school interview and a 60–120 minute in-home interview. Of these, 14 736 students were re-interviewed in 1996. The current study used data from 4831 adolescents who comprised the public use data and who provided data at both waves.

Participants—At baseline, 52% of the respondents were female, with 23% black, 61% white, 12% Hispanic and 4% categorized as ‘other’. Mean age was 14.8 (SD = 1.7; range 12–18). At baseline, 53% endorsed having had more than 2–3 drinks in their lifetime and 54% endorsed ever smoking.

Measures—Alcohol and tobacco use items were assessed at both years 1 and 2. Frequency of alcohol consumption was assessed with an item recording weekly frequency of alcohol use in the past 12 months. In addition, frequency of past-year heavy (five or more drinks) drinking was assessed. For both, response options ranged from 0 (never) to 6 (every day). Respondents who reported that they had never drunk more than 2–3 drinks in their lifetime were given values of zero for all drinking frequency variables.

Cigarette quantity was assessed with a single item: calculating the number of cigarettes smoked per day. Whether the respondent considered himself/herself to be a ‘regular smoker’ was assessed with a binary item (1 = yes, 0 = no). Respondents who reported that they had never smoked were given values of zero for cigarette quantity.

Sex, ethnicity and age were assessed at year 1. Ethnicity was dummy-coded (two codes were created: black and Hispanic, with white as the reference group) in the analyses. Additional covariates that were examined included history of alcoholism and cigarette smoking in the biological parents, depression assessed by 18 items from the Center for Epidemiological Studies Depression Scale (Radloff 1977) ($\alpha = 0.86$), which ranged from 0 (never or rarely) to 3 (most or all of the time), and behavioral undercontrol assessed at baseline with 15 past-year delinquency items ($\alpha = 0.83$), which ranged from 0 (not at all) to 3 (five or more times).

Adolescent Health Risk Study

Data and procedures—The goal of the Adolescent Health Risk Study (AHRS) was to identify the processes that drive the relationship between alcohol use and risky sexual behavior (Cooper & Orcutt 2000; Dermen & Cooper 2000). In 1989, 2052 randomly selected black and white adolescents from the Buffalo (NY) region aged 13–19 completed a 2 hour interview and self-administered questionnaire. Approximately 5 years later, 1814 respondents were re-interviewed.

Participants—At baseline, 957 (53%) of the respondents were female; 44% were black, 49% were white and 8% were categorized as 'other'. Mean age was 16.7 (SD = 2.02; range 13–20). At baseline, 65% had ever had a drink of alcohol in their lifetime and 20% currently smoked (at year 5, 62% reported having ever smoked; this information was unavailable at year 1).

Measures—Frequency of drinking and heavy drinking were based on the past 6 months. Heavy drinking was operationalized as the mean of two items: (1) frequency of five or more drinks per day, and (2) frequency of drinking to intoxication. These ordinal items, adapted from the National Health and Leisure Time Survey (Wilsnack *et al.* 1984), ranged from 0 (not at all) to 8 [every day (for drinking frequency) or five times a week or more (for heavy drinking)].

Whether the respondent is a current smoker (year 1) or smoked in the past 6 months (a proxy for current smoker; year 5) and daily quantity of cigarettes were assessed. Current smoking was considered to be a proxy for the regular smoking variable in AddHealth.

Additional covariates included sex, ethnicity, age, history of alcoholism in the caregiver and biological history of cigarette smoking, depression (or past month psychological distress), measured by the 51-item Brief Symptom Inventory (Derogatis & Melisaratos 1983) ($\alpha = 0.93$) and behavioral undercontrol, a count of 16 items assessing frequency of delinquent behavior in the past 6 months ($\alpha = 0.82$).

A number of other drinking and smoking variables were assessed in both AHRS and AddHealth (e.g. ever drank, frequency of drinking to intoxication, drinking quantity, ever smoked, smoking frequency). Ancillary analyses based on these variables yielded results nearly identical to those reported herre, with only a few non-remarkable exceptions. Given the complexity of the analyses reported in this paper, we focused on two drinking variables and two smoking variables per dataset: a 'use' variable (drinking frequency, regular/current smoking) and a 'heavy use' variable (heavy drinking, smoking half pack/day). Ever drank and ever smoked were used for descriptive analyses (in the earlier description of participants) and for a subset of analyses (i.e. onset and persistence). Although non-cigarette tobacco use was assessed in AddHealth, it exhibited low correlations with the other tobacco variables and is not considered further.

Complex sampling analysis—AddHealth (but not AHRS) utilized a complex sampling design, including clustering of observations, stratification and unequal probability of inclusion for demographic strata. To obtain unbiased variance estimates, we adjusted for complex sampling and non-response in all analyses (exceptions noted below) using WesVar Complex

Samples (Westat 1998), employing a repeated replication procedure using the AddHealth final post-stratified sampling weights.

Attrition analysis

AddHealth: Only 4831 (74%) of the 6504 respondents were reassessed at year 2. Overall, attrition rates for those lost at year 2 tended to be similar to those successfully followed, with the following exceptions: non-completers were more likely to be male [$p < 0.01$; Cohen's (1977) $h = 0.08$] and to use alcohol and tobacco [all p values < 0.001; $h = 0.12–0.25$]. In addition, non-completers were more likely to be older participants [$p < 0.001$; Cohen's (1977) $d = 0.65$] and (unexpectedly) to exhibit lower delinquency scores ($p < 0.01$; $d = 0.08$). These findings suggest that our longitudinal analyses represent conservative estimates of alcohol and tobacco use, which could lead to lower prevalence estimates of substance use and of the association between alcohol, tobacco and other variables of interest. In addition, because younger adolescents were more likely to be retained, caution should be exercised in generalizing to older adolescent populations.

AHRS: Those who were re-interviewed at year 5 ($n = 1814$; 88%) were significantly younger at year 1 ($p < 0.001$; $d = 0.25$) than non-completers ($n = 238$), and more likely to be female ($p < 0.001$; $h = 0.29$) and less highly educated ($p < 0.001$; $d = 0.24$). In addition, completers were more depressed and anxious ($p < 0.001$; $d = 0.23$; $d = 0.25$) and less delinquent ($p < 0.01$; $d = 0.24$). Unlike AddHealth, these data do not suggest biases in alcohol and tobacco use, although care must be exercised in generalizing to more educated, older and male populations.

RESULTS

Firstly, trends in drinking and smoking over time are described, capitalizing on the multiple-cohort design of each dataset. Bivariate associations of alcohol and tobacco use are then reported for cross-sectional as well as prospective data. Also, the alcohol–tobacco association is examined for processes of both initiation and persistence. Finally, the influence of common third variables upon the alcohol–tobacco relationship is reported.

Age trends in drinking and smoking

Mean trends in drinking and smoking were examined over time by plotting the mean trend for times 1 and 2 by age cohort, controlling for sex and ethnicity. The extent to which growth was linear and/or quadratic was tested using repeated measures analysis of variance (ANOVA) tests, and interactions between growth and sex and between growth and ethnicity were tested using interaction contrasts.

AddHealth—As indicated in Fig. 1, by age 12 there was already some use of alcohol and tobacco. Both alcohol consumption and cigarette use monotonically increased from ages 12 to 19. Although frequency of drinking and heavy drinking did not asymptote, endorsement of regular smoker status and cigarette quantity increased until age 17 or 18, at which point they leveled off. All variables exhibited significant linear growth (minimum $F_{1,3898} = 35.27$, $p < 0.001$).

These trends were also examined by sex and by ethnicity (due to the few respondents in other ethnic groups, these analyses were limited to blacks and whites). The trends were similar, with some exceptions. Specifically, men drank more alcohol than women, particularly after age 15. Although men showed rapid linear growth in alcohol use, women followed a quadratic pattern with growth at age 13 and relative stability after age 16. The extent to which the drinking (and smoking) linear and quadratic trends differed across sex was tested: for heavy drinking only, men had a significantly steeper linear trend than women ($F_{1,3864} = 8.55$, $p < 0.05$).

Overall, blacks drank less than whites. Although drinking by whites increased linearly over ages 12–19, drinking by blacks grew more slowly, with an increase beginning at age 15. Blacks also smoked less than whites, with a moderate linear increase for whites but low levels for blacks until age 16, at which point smoking began to increase. Interestingly, this growth for blacks was primarily reflected in the number of regular smokers, rather than cigarette quantity. These linear trends were significantly steeper for white than for black adolescents for all drinking and smoking variables (minimum $F_{1, 3864} = 3.92$, $p < 0.05$). No significant three-way interactions were observed between sex, ethnicity and linear (or quadratic) effects.

AHRS—Low but non-zero mean levels of drinking were reported by 13-year-old respondents, and alcohol consumption exhibited a linear increase from age 13 to about age 18, at which point it leveled off (see Fig. 2). Conversely, respondents reported virtually no smoking at age 13, although cigarette use increased linearly from age 13 to about age 19, at which point it leveled off. All variables exhibited significant linear growth (minimum $F_{1,1633} = 22.29$, $p < 0.001$).

Men showed higher mean drinking levels and steeper growth than women. For heavy drinking (but not drinking frequency), linear growth interacted with sex ($F_{1,1631} = 4.84$, $p < 0.05$) such that men had steeper growth than women. Mean smoking levels and growth of smoking over time was relatively similar across gender.

Again, blacks drank less than whites. Whites showed a steep increase in drinking up to about age 19, followed by a gradual decrease. Alternately, blacks exhibited constant linear growth, such that mean levels of consumption were roughly comparable for blacks and whites by about age 23. Linear growth interacted with ethnicity ($F_{1,1631} = 4.40$, $p < 0.05$) for heavy drinking. Blacks also smoked less than whites. Although smoking rates for whites increased linearly until age 19 and then leveled off, rates increased steadily for blacks until age 24. No three-way interactions were observed.

Characterization of the alcohol-tobacco relationship

Given that developmental trends for alcohol and tobacco use were relatively similar, cross-sectional and prospective relations between alcohol and tobacco use were examined using product-moment correlation, logistic regression and ordinary least squares (OLS) regression analyses, controlling for prior (time 1) use in the prospective analyses.

AddHealth—Product-moment correlations were computed to describe cross-sectional associations among alcohol and tobacco use items at years 1 and 2. Alcohol and tobacco use were moderately associated (year 1 r values range between 0.27 and 0.38, median $r = 0.335$; year 2 r values range between 0.27 and 0.35, median $r = 0.315$).

Year 1 drinking frequency predicted both regular smoking (standardized $\beta = 0.27$; OR = 1.40, 95% CI = 1.30, 1.52) and number of cigarettes (standardized $\beta = 0.16$; $\beta = 0.64$, $p < 0.001$) at year 2, controlling for year 1 tobacco use. Correspondingly, year 1 heavy drinking frequency predicted year 2 regular smoking (standardized $\beta = 0.21$; OR = 1.34, 95% CI = 1.22, 1.47) and number of cigarettes (standardized $\beta = 0.13$; $\beta = 0.59$, $p < 0.001$). Analogously, year 1 regular smoking predicted year 2 alcohol use, controlling for year 1 alcohol use, predicting both occasional drinking (standardized $\beta = 0.12$; $\beta = 0.45$, $p < 0.001$) and heavy drinking (standardized $\beta = 0.17$; $\beta = 0.60$, $p < 0.001$). Number of cigarettes did not predict year 2 drinking prospectively.

AHRS—Cross-sectional product-moment correlations at years 1 and 5 revealed that alcohol and tobacco use were moderately associated (year 1 r values range between 0.39 and 0.44, median $r = 0.415$; year 5 r values range between 0.30 and 0.40, median $r = 0.36$).

Year 1 drinking frequency predicted year 5 tobacco use (controlling for year 1 tobacco use) for both current smoking (standardized β = 0.18; OR = 1.18, 95% CI = 1.11, 1.25) and number of cigarettes (standardized β = 0.09; β = 0.33, $p < 0.001$). Likewise, year 1 heavy drinking frequency predicted year 5 current smoking (standardized β = 0.15; OR = 1.16, 95% CI = 1.08, 1.24) and number of cigarettes (standardized β = 0.08; β = 0.32, $p < 0.001$). Year 1 current smoking predicted year 5 alcohol use (controlling for year 1 alcohol use) for both occasional drinking (standardized β = 0.05; β = 0.25, $p < 0.05$) and heavy drinking (standardized β = 0.07; β = 0.37, $p < 0.01$). Again, number of cigarettes did not predict year 5 drinking prospectively.

Onset and persistence of smoking and drinking

Prior to discussion of onset/persistence analyses, prevalences of alcohol and tobacco use (versus no use) over time are reviewed. For these data and for the onset/ persistence analyses that follow, drinking and smoking variables were dichotomized in order to characterize drinking and smoking as discrete behaviors (yes versus no) rather than as continuous gradients of substance use. Alcohol use was categorized into occasional and heavy drinking. Occasional drinking was coded if the respondent drank at least 3–12 times/year for AddHealth and 12 times/year for AHRS. Heavy drinking was coded if the respondent drank 5+ drinks at least 3–12 times/ year for AddHealth and drank heavily (mean of 5+ drinks and drink to intoxication) at least 12 times/year for AHRS. Smoking was categorized into regular smoking (AddHealth) or current smoking (AHRS) and half pack/day (i.e. at least 10 cigarettes/day) smoking.

Figures 3 and 4 display the proportion of respondents who drink and smoke, only drink, only smoke and abstainers. Prevalences are presented for both regular and heavy use; hence, there are four graphs (per dataset): (1) occasional drinking/regular smoking; (2) occasional drinking/ heavy smoking; (3) heavy drinking/regular smoking, and (4) heavy drinking/heavy smoking. When plotting prevalence rates for a given age, both time 1 and time 2 data were accounted for by averaging the prevalence rates (e.g. if age 14 prevalence for occasionally drinking is 0.45 at time 1 and age 13 prevalence is 0.55 at time 2, these numbers were averaged to 0.50).

AddHealth—The majority of respondents did not use alcohol or tobacco (see Fig. 3). As expected, fewer respondents exhibited heavy use than regular use. Although (nonsmoking) drinking was more prevalent than (non-drinking) smoking, smoking and drinking appeared to be equally, if not more, prevalent than (non-drinking) smoking.

In a series of logistic regressions, we examined the extent to which initiation of alcohol use (i.e. year 2 alcohol use by year 1 non-drinkers) was predicted by year 1 tobacco use, and the extent to which initiation of tobacco use was predicted by year 1 alcohol use. Only respondents who reported never having consumed alcohol ($n = 2288$) (or having smoked a cigarette, $n = 2228$) at year 1 were included in the analysis. Sex, ethnic group and baseline age were controlled. Drinking onset was associated with prior smoking and smoking onset was associated with prior drinking (see Table 1, top left panel).

The extent to which drinking persistence (i.e. year 2 drinking by year 1 drinkers) was predicted by year 1 tobacco use and the extent to which smoking persistence was predicted by year 1 alcohol use were estimated (see Table 1, top right panel). Only respondents who reported having consumed alcohol ($n = 2578$) (or having smoked a cigarette, $n = 2632$) at year 1 were included; sex, ethnicity and baseline age were again controlled. Smoking persistence was associated with prior drinking and drinking persistence was associated with prior smoking. Odds ratios for persistence were smaller than those for onset, suggesting that onset is better predicted by prior use of the other substance than is persistence. For the most part, findings from the onset and persistence analyses were similar across sex, ethnicity and age; when parameters differed as a function of subgroup (tested using interactions), they are presented separately by subgroup.

AHRS—Although the majority of respondents did not use tobacco (see Fig. 4), occasional alcohol use increased over time such that by age 19, more respondents reported occasional alcohol use than not (see top left and top right panels). Interestingly, by age 21 more respondents were comorbid occasional drinkers and smokers than (nonsmoking) occasional drinkers, (non-drinking) regular smokers or even abstainers (see top left panel). There were few heavy drinkers who were not regular smokers (see bottom left panel).

Initiation and persistence of alcohol and tobacco use as a function of prior use of the other substance were again examined, controlling for sex, ethnicity and baseline age. Initiation of drinking was somewhat associated with prior regular smoking, but not half pack/day smoking (in fact, the association between half pack/day smoking and heavy drinking was not estimable because none of the nine individuals who smoked half pack/day at year 1 reported heavy drinking at year 5). Initiation of smoking was associated with prior drinking (see Table 1, top left panel). Drinking persistence was associated with prior smoking; half pack/day smoking persistence (but not regular smoking persistence) was associated with prior drinking (see Table 1, bottom right panel). Again, parameters that significantly differed by subgroup are presented separately.

Common third-variable explanation for comorbidity

Analyses were consistent with a bi-directional causal model of comorbidity between alcohol and tobacco use. However, it is possible that comorbidity is also due to a set of common third variables. To address this issue, we examined the extent to which the alcohol–tobacco association was attenuated or eliminated in the presence of etiologically important third variables (i.e. age, gender, race, family history of smoking and alcohol use, delinquency and depression).

We examined cross-sectional correlations between alcohol use and tobacco use (at times 1 and 2), partial-ling out age, gender, race, family history of alcohol and tobacco use, delinquency and depression. In the AddHealth dataset, Pearson correlations were reduced from a range of 0.27–0.38 to a range of 0.15–0.26 and from a range of 0.30–0.44 to a range of 0.19–0.32 in AHRS. These represent decreases ranging from 26 to 44% (AddHealth) and 20 to 49% (AHRS). Next, logistic and OLS regression parameters were tested, controlling for these same third variables. Standardized β values decreased from a range of 0.07–0.27 to a range of 0.04–0.18 in AddHealth, and from a range of 0.01–0.18 to a range of −0.02–0.13 in AHRS. These represent decreases ranging from 18 to 46% (AddHealth) and from 11 to 69% (AHRS). Finally, logistic and OLS regressions examining onset and persistence of alcohol/tobacco use were estimated with the addition of family history, delinquency and depression (recall that original onset and persistence analyses already controlled for age, gender, and race). Standardized β values decreased from a range of 0.09–0.30 to a range of 0.06–0.21 in AddHealth, and from a range of 0.01–0.19 to a range of −0.03–0.15 in AHRS. These represent decreases ranging from 5 to 53% (AddHealth) and from 0 to 100% (AHRS) (due to the addition of family history, delinquency and depression).

Although these third variables account for non-trivial shared association between alcohol and tobacco use, cross-sectional correlations between alcohol and tobacco use remained significant and the majority of significant prospective bi-directional effects (12 of 16, 75%) and onset/persistence effects (20 of 31, 65%) were still evident in the presence of the third variables.

DISCUSSION

Using two datsets, trends in alcohol and tobacco use were examined throughout adolescence and young adulthood, and sex and ethnic differences in these trends were described. Cross-

sectional and prospective associations between alcohol and tobacco use were examined for both initiation and continued use (as a function of prior use of the other substance).

General trends in drinking and smoking

Consistent with other research (e.g. Chassin *et al.* 1996; Bachman *et al.* 1997), alcohol and tobacco use onset by age 12 or 13 escalated through the adolescent years, and reached an asymptote by mid young adulthood. As expected, men had higher mean levels and greater increases in drinking, yet women's smoking appeared very similar to men's, a finding that has only recently been supported in the literature (Johnston *et al.* 1998; Duncan & Duncan 1994). In addition, whites showed steeper mean smoking trajectories than blacks. Whereas whites generally mature out of heavy drinking after age 21 or 22, heavy drinking among blacks peaks later and persists longer into adulthood (Caetano & Kaskutas 1995; Caetano & Clark 1998). Black and white adolescents may have identical but time-delayed trajectories of alcohol consumption (e.g. consumption for blacks at age 25 is identical to that for whites at age 20).

Much research in addictive behavior (e.g. Grant 1998; Anthony *et al.* 1994) describes prevalence rates using coarse groupings (e.g. ages 15–24, 25–34) that are less informative for describing developmental trends in substance use. In the current study, mean drinking and smoking levels are described over an 8 (or 13) year period of adolescence/young adulthood using only two waves of data. The close proximity of points at a given age (e.g. time 1 mean cigarette use for a 15-year-old is nearly identical to time 2 mean cigarette use for a 14-year-old; see Fig. 1) suggest few cohort effects that might obscure the true pattern of growth.

Alcohol–tobacco comorbidity

Alcohol and tobacco use were moderately associated at both time 1 and time 2 in both samples. Prospective analyses, controlling for baseline levels, revealed that prior alcohol use predicted tobacco use and that prior tobacco use predicted alcohol use – although with smaller magnitude and only for current/regular smoking status (versus quantity of cigarettes smoked). In addition, our graphical displays of drinking and smoking showed that smokers are more likely to drink than not – prevalence of comorbid use was greater than (non-drinking) smoking for both regular and heavy use.

Year 1 smokers were more likely than non-smokers to initiate drinking either 1 year (AddHealth) or 5 years (AHRS) later; this is consistent with Fleming *et al.* (1989), who found that time 2 onset of beer and liquor consumption was higher for time 1 adolescent smokers than for non-smokers. Also, smoking onset was shown to be higher for prior drinkers. The disin-hibiting effects of alcohol might reduce resistance to smoking and lead to initiation of use (Shiffman & Balabanis 1995).

Prescott & Kendler (1995) found that shared (genetic) variation between alcohol and tobacco use was much reduced when abstainers were removed; they suggest that much of the genetic covariation between alcohol and tobacco use may be due to the large group of abstainers. If the robust association between drinking and smoking is actually due to the variance associated with abstention, then we would expect to see a null association among non-abstainers. That is, persistent smoking among smokers would not be associated with prior alcohol use and persistent drinking among drinkers would not be associated with prior tobacco use. Yet smoking persistence varied as a function of prior drinking and drinking persistence varied as a function of prior smoking, indicating that the alcohol-tobacco relationship is not merely an artefact of abstention of both alcohol and tobacco.

These prospective associations between (initiation and maintenance of) alcohol and tobacco were observed even when controlling for sex, ethnicity, age, family history of smoking and

alcohol use, delinquency and depression. This suggests that the relationship between alcohol and tobacco use cannot be explained entirely by these common third variables. Yet the extent to which these third variables account for the alcohol-tobacco relationship remains to be explored.

Although prediction of initiation and maintenance was expected to differ as a function of the intensity of the outcome variable (e.g. occasional versus heavy drinking), this was not the case. However, heavy drinking appeared to be more predictive of heavy smoking than regular/current smoking (across both datasets). The converse was not true: heavy smoking was not more predictive of heavy drinking than occasional drinking, perhaps because there are far fewer heavy smokers than heavy drinkers at year 1. Although certainly not conclusive evidence, this finding – as well as the prospective finding that tobacco use predicted alcohol use to a lesser extent than the converse – is consistent with the notion that drinking causes smoking more strongly than the converse, as noted by Shiffman & Balabanis (1995). Animal and human laboratories demonstrate that alcohol administration promotes increased smoking (e.g. Mitchell *et al.* 1995; Glautier *et al.* 1996). Alternately, individuals may use nicotine to counteract alcohol's debilitating effects on cognitive skills (Kerr *et al.* 1991; Madden *et al.* 1995). Relatedly, there is evidence that alcohol may act as a cue for tobacco use in both laboratory (Gulliver *et al.* 1995) and field (Shiffman *et al.* 1994) settings. Wise (1988) suggests that nicotine and ethanol stimulate the same dopaminergic pathways and therefore might stimulate cravings for one another. There might also be a learned association between smoking and drinking (e.g. both often occur in similar social situations). Clearly, the datasets in the current study were not designed to test competing theories of alcohol and tobacco comorbidity, but our findings are consistent with the view that alcohol use has a causal role in subsequent tobacco use and (less strongly) that tobacco use has a causal role in subsequent alcohol use. In addition, results of the onset and persistence analyses were relatively robust across sex, ethnicity and age: the few effects that differed by subgroup were not consistent patterns and all differences were deviations in magnitude, rather than differential relations between drinking and smoking.

AddHealth versus AHRS

Except when noted, all of our findings were replicated across both datasets, increasing our confidence in their robustness. However, one important distinction between the two datasets is that the AddHealth follow-up fell over a 1 year interval, whereas the AHRS follow-up occurred over a 5 year interval. The effect of the differential time lag across datasets is particularly apparent in the graphs depicting mean trends in drinking and smoking by age. AHRS clearly shows a leveling-off or decline in use that occurs around age 19. Additionally, AHRS data revealed that among young adults (age 21 and above), comorbid occasional drinking and smoking was more prevalent than use of any single substance or even abstinence. These observations were not apparent from AddHealth data, which are truncated at age 19 (year 2 for the 18-year-old cohort). Nevertheless, the 1 year follow-up interval of AddHealth allows for a more refined depiction of the growth process. In AddHealth, one can observe annual fluctuations in use that are assumed to be linear growth in AHRS. For example, in AddHealth, growth in alcohol use appears to level off at ages 15–17 but is followed again by positive growth in use, presumably at the time of college enrollment, for a substantial number of respondents. Also, a decrease over the time 1 to time 2 timespan within certain age cohorts (e.g. heavy drinking for age group 13 in the AddHealth dataset) is apparent: this might reflect an ascertainment or retention bias in these cohorts.

Associations between alcohol and tobacco use were stronger in AddHealth data, particularly for onset analyses, consistent with its shorter follow-up interval. However, greater prospective associations for AHRS (relative to AddHealth) than expected were observed, given that a first-

order, autoregressive process would predict that a 5 year association would be diluted greatly from a 1 year association. The similarity of parameters over a 1 year follow-up and a 5 year follow-up lend support to the concept of alcohol and tobacco use as trait-like factors (shown for alcohol and tobacco use disorders in Jackson *et al.* 2000), as opposed to a more time-bound level of association that is reflective of a true autoregressive process.

Strengths and limitations

Alcohol and tobacco use and the covariation between the two substances were described using prospective data, which allowed us to establish directionality. In addition, the prospective nature of the study allowed us to examine substance use onset without relying on retrospective estimations. Given that the size of both datasets was quite large, there was sufficient power to test all of our effects within a multivariate framework; in particular, samples were partitioned into non-users and users in order to examine the onset and persistence of substance use. Also, there were ethnically diverse samples with oversampling of blacks and (in the case of AddHealth) Hispanics. This enabled us to compare parameters not only across sex but also across ethnicity, with sufficient power to test these differences. In addition, alcohol and tobacco use was described from a young age (12 or 13), a developmental period that appears to be crucial in terms of substance use initiation. Finally, the majority of our findings were replicated across independent datasets, greatly increasing our confidence in the validity of the results. In particular, the two datasets used different operationalizations of substance use (e.g. the measure of regular smoking in AddHealth differed from the measure of current smoking in AHRS; assessments of drinking frequency across datasets were based on different scales). The extent to which the different measures yielded similar findings suggests that our findings are not simply artefacts of a particular operationalization of the construct(s).

However, this study was limited by the reliance upon self-report measures, as there were no biological measures with which to validate reported alcohol consumption or tobacco use. In addition, not only were measures of alcohol and tobacco use different across datasets, but the conceptualizations of ‘regular’ and ‘heavy’ use were different for drinking and smoking and hence are not directly comparable. Moreover, it is unlikely that measurement error is equivalent across substances; this has implications for drawing conclusions about directionality.

Another limitation of our work is that we could examine the onset of substance use only among those who had not already initiated substance use – that is, those adolescents with late onset. These individuals may be different to those who were already drinking (or smoking) by time 1. Early onset drinking is associated with positive family history of alcoholism (Dawson 2000); this association might be due to genetic factors (Tarter & Vanyukov 1994) such as dopaminergic and serotonergic systems, which are associated with novelty-seeking and impulsivity (Howard *et al.* 1997; Masse & Tremblay 1997). Alternately, this association might be due to environmental factors such as poor parental monitoring or access to alcohol in the home (Dawson 2000). In addition, early onset of cigarette use is associated with high novelty seeking (Masse & Tremblay 1997) and parental smoking (Jackson & Henriksen 1997). Certainly, early onset of drinking (and smoking) may show different relationships with prior tobacco (alcohol) use than were observed in the current study.

In addition, although the findings provide evidence for directional effects, as mentioned earlier, it is not possible to distinguish among the theories that underlie directional effects (e.g. reciprocal antagonism, cross tolerance, cueing). Also, both datasets were limited to two waves, which hinders our ability to distinguish between alternate models of comorbidity (e.g. a trait model of comorbidity versus an autoregressive, stochastic process).

CONCLUSIONS

Both directional and common third-variable theories have been proposed to explain alcohol–tobacco comorbidity. Previous research has provided evidence for each of these classes of theories but has been limited in terms of sample size, homogeneous populations, limited measures of alcohol and tobacco involvement and the ability to distinguish between theories because of the cross-sectional nature of the data. Using two large, prospective, ethnically diverse samples, the current study provided support for a bi-directional association between alcohol and tobacco use, revealing a particularly strong association between smoking and subsequent alcohol use. These associations were robust across sample, subgroup (sex, ethnicity and, to an extent, age group) and measure of alcohol and tobacco use ('regular' versus 'heavy' use). In addition, the current study provided preliminary support for a common third-variable explanation of comorbidity that operates in addition to the directional influences described.

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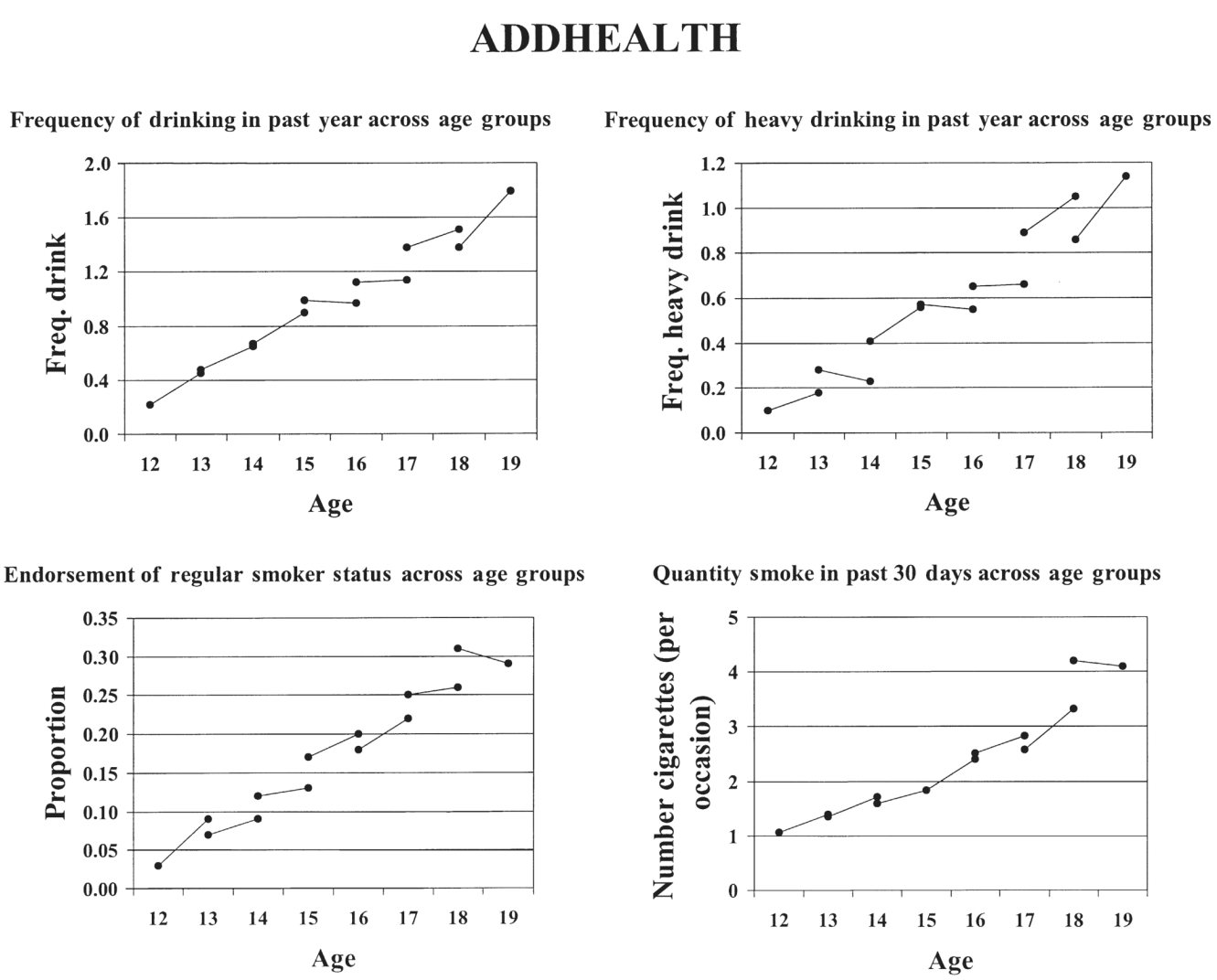

Figure 1.

Mean trajectories of times 1 and 2 alcohol and tobacco use over ages 12–19 for the National Longitudinal Study of Adolescent Health (AddHealth). Adjusted for sex and ethnicity. Freq. drink refers to frequency of drinks, expressed in days per week. Freq. heavy drink refers to frequency of five or more drinks per occasion, expressed in days per week

AHRS

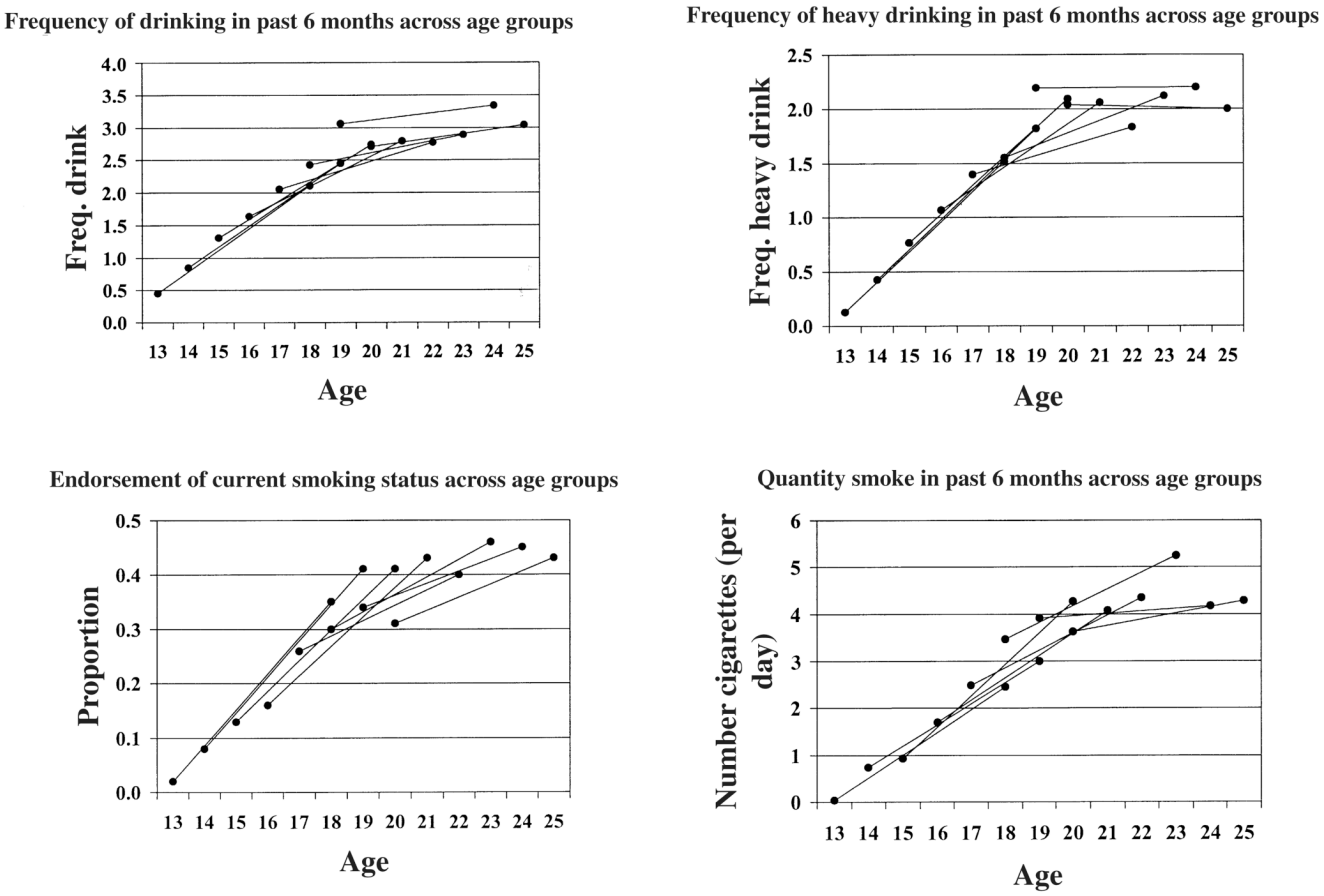

Figure 2.

Mean trajectories of times 1 and 2 alcohol and tobacco use over ages 13–25 for the Adolescent Health Risk Study (AHRS). Adjusted for sex and ethnicity. Freq. drink refers to frequency of drinks. Freq. heavy drink is a mean of frequency of five or more drinks per day and frequency of drinking to intoxication

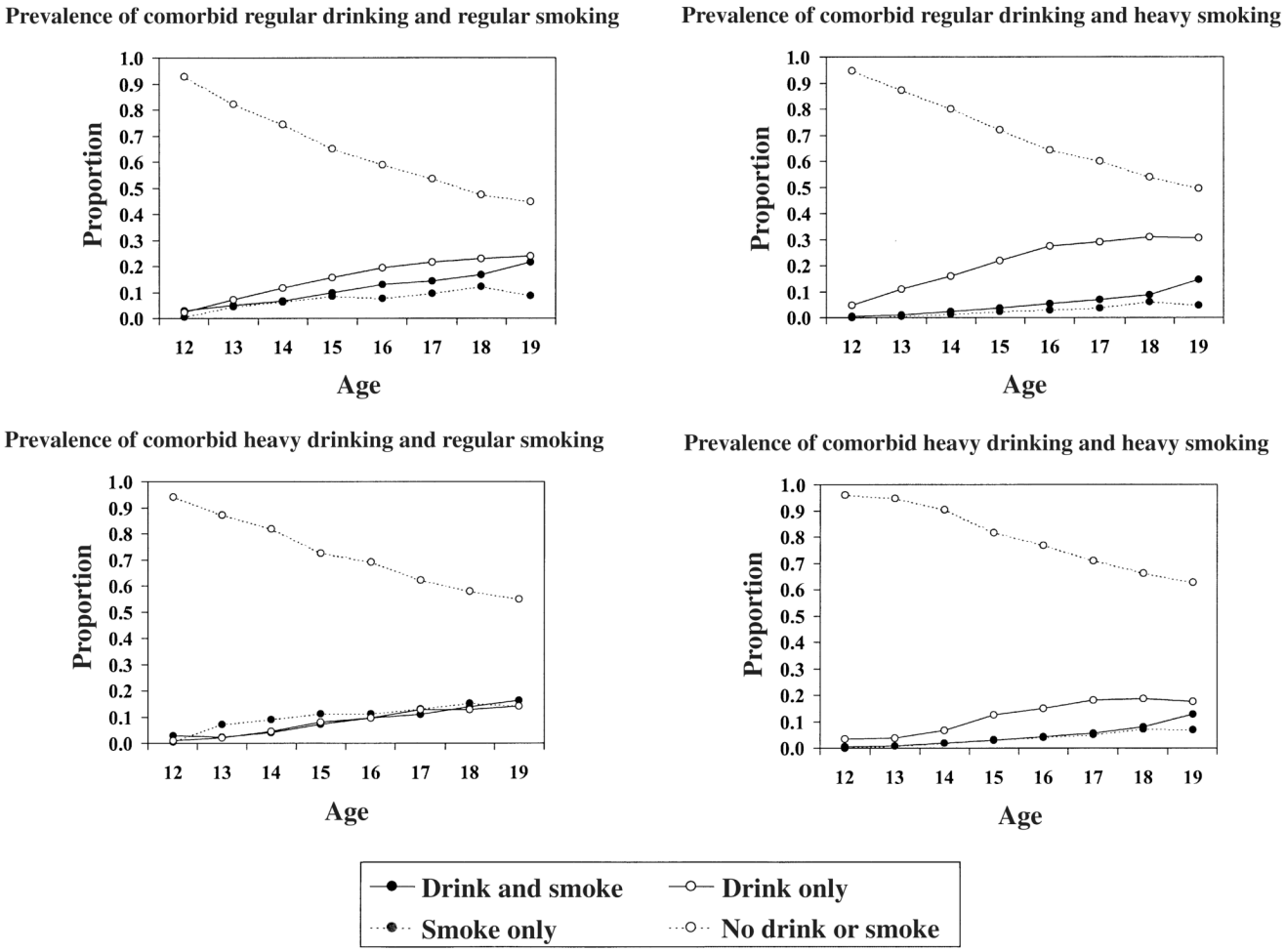

Figure 3.
Mean trajectories of comorbidity, alcohol use only, tobacco use only and abstinence over ages 12–19 for AddHealth

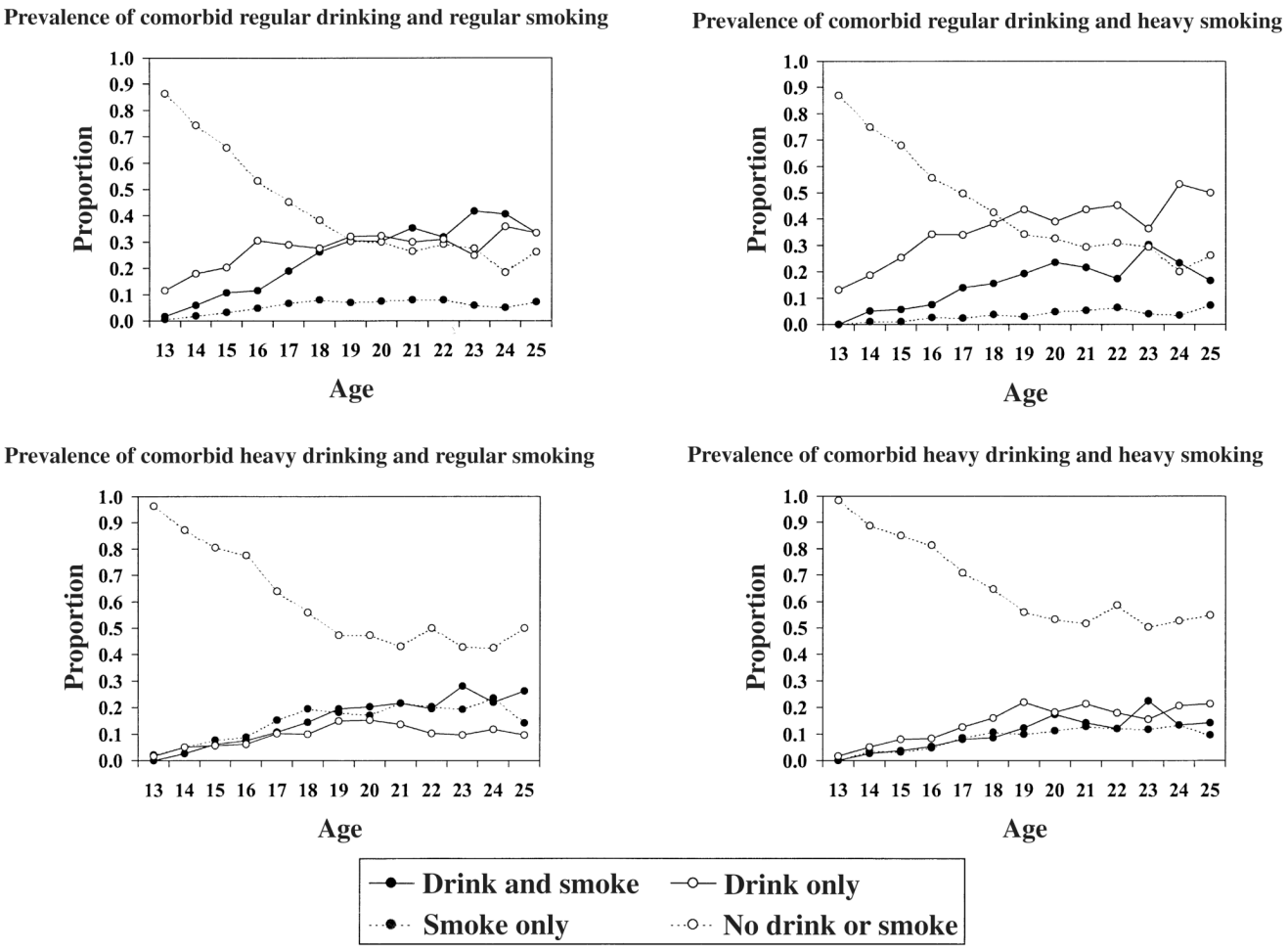

Figure 4.
Mean trajectories of comorbidity, alcohol use only, tobacco use only and abstinence over ages 13–25 for AHRS

Table 1

Odds ratios (adjusted for sex, ethnicity and age) showing prediction of time 2 drinking from time 1 smoking and time 2 smoking from time 1 drinking for AddHealth and AHRS data.

AddHealth

	ONSET of year 2 drinking among year 1 non-drinkers (n = 2288)				**PERSISTENCE of year 2 drinking among year 1 drinkers (n = 2578)**			
	Year 1	**Prevalence**	**OR**	**95% CI**	**Year 1**	**Prevalence**	**OR**	**95% CI**
Year 2	Regular smoke		4.14	(2.3, 7.5)	Regular smoke		1.62 (White)	(1.2, 2.1)
Occ. drink							1.48 (Black)	(0.8, 2.9)
							4.18 (Hisp)	(2.0, 8.6)
	yes	0.30			yes	0.62		
	no	0.09			no	0.45		
	½ pack/day		2.72 (White)	(0.9, 8.4)	½ pack/day		1.65	(1.15, 2.37)
			12.68 (Black)	(0.8, 199.5)				
			28.79 (Hisp)	_ *a*				
	yes	0.29			yes	0.65		
	no	0.09			no	0.48		
Heavy drink	Regular smoke		5.87	(3.00, 11.6)	Regular smoke		2.25 (White)	(1.7, 2.94)
							2.23 (Black)	(1.0, 5.2)
							4.44 (Hisp)	(2.2, 8.8)
	yes	0.21			yes	0.48		
	no	0.04			no	0.26		
	½ pack/day		5.87	(2.0, 16.8)	½ pack/day		2.32	(1.6, 3.4)
	yes	0.25			yes	0.56		
	no	0.04			no	0.30		
	ONSET of year 2 smoking among year 1 non-smokers (n = 2288)				PERSISTENCE of year 2 smoking among year 1 smokers (n = 2632)			
	Year 1	Prevalence	OR	95% CI	Year 1	Prevalence	OR	95% CI
Year 2	Occasional drink		5.00	(2.6, 9.5)	Occasional drink		3.41 (young)	(2.5, 4.6)
Reg. smoke							2.15 (old)	(1.6, 2.8)
	yes	0.13			yes	0.51		

AddHealth

	ONSET of year 2 drinking among year 1 non-drinkers (n = 2288)				PERSISTENCE of year 2 drinking among year 1 drinkers (n = 2578)			
	Year 1	**Prevalence**	**OR**	**95% CI**	**Year 1**	**Prevalence**	**OR**	**95% CI**
	no	0.03			no	0.27		
	Heavy drink		5.87	(2.9, 11.8)	Heavy drink		3.80 (young)	(2.6, 5.5)
							2.51 (old)	(1.9, 3.3)
	yes	0.17			yes	0.57		
	no	0.03			no	0.30		
½ pack/day	Occasional drink		6.96	(2.8, 17.4)	Occasional drink		3.26 (young)	(2.2, 4.8)
							1.88 (old)	(1.4, 2.5)
	yes	0.05			yes	0.26		
	no	0.01			no	0.12		
	Heavy drink		10.18	(3.5, 29.7)	Heavy drink		2.48	(1.9, 3.3)
	yes	0.09			yes	0.31		
	no	0.01			no	0.13		

AHRS

	ONSET of year 2 drinking among year 1 non-drinkers (n = 632)				PERSISTENCE of year 2 drinking among year 1 drinkers (n = 1179)			
	Year 1	Prevalence	OR	95% CI	Year 1	Prevalence	OR	95% CI
Year 5	Regular smoke		12.4 (men)	(1.6, 99.7)	Regular smoke		1.46	(1.0, 2.0)
Occ. drink			1.08 (women)	(0.3, 3.9)				
	yes	0.65			yes	0.81		
	no	0.42			no	0.73		
	½ pack/day		1.16	(0.3, 4.6)	½ pack/day		1.12	(0.8, 1.6)
	yes	0.44			yes	0.80		
	no	0.43			no	0.74		
Heavy drink	Regular smoke		3.03	(1.2, 7.8)	Regular smoke		1.94	(1.5, 2.6)
	yes	0.35			yes	0.51		
	no	0.17			no	0.37		
	½ pack/day		_[b]	–	½ pack/day		1.56	(1.1, 2.1)
	yes	0.00			yes	0.49		

AddHealth

	ONSET of year 2 drinking among year 1 non-drinkers (n = 2288)				PERSISTENCE of year 2 drinking among year 1 drinkers (n = 2578)			
	Year 1	**Prevalence**	**OR**	**95% CI**	**Year 1**	**Prevalence**	**OR**	**95% CI**
	no	0.18			no	0.39		
	ONSET of year 2 smoking among year 1 non-smokers (n = 1450)				PERSISTENCE of year 2 somking among year 1 smokers (n = 361)			
	Year 1	Prevalence	OR	95% CI	Year 1	Prevalence	OR	95% CI
Year 5	Occasional drink		1.59 (men)	(1.1, 2.3)	Occasional drink		2.14	(0.9, 4.8)
Reg. smoke			2.33 (women)	(1.6, 3.4)				
	yes	0.38			yes	0.94		
	no	0.27			no	0.86		
	Heavy drink		1.49	(1.0, 2.2)	Heavy drink		1.23	(0.5, 2.8)
	yes	0.39			yes	0.93		
	no	0.30			no	0.91		
½ pack/day	Occasional drink		1.56	(1.1, 2.2)	Occasional drink		2.29	(1.3, 4.0)
	yes	0.17			yes	0.77		
	no	0.12			no	0.57		
	Heavy drink		1.93	(1.2, 3.0)	Heavy drink		1.84	(1.1, 3.1)
	yes	0.22			yes	0.81		
	no	0.12			no	0.65		

Reg. smoke = regular smoke; ½ pack/day = smoke half pack/day or more; Occ. drink = drink occasionally.

Hisp = Hispanic; Young ≤ 15 years; old ≥ 16 years, Adjusted for sex, ethnicity, and age.

[a]These parameters are inestimable. The standard error was more than twice the magnitude of the logistic regression coefficient, and as a result the upper confidence interval was over 47 million. The large standard error is in part a function of the small sample size in this subgroup (only 245 Hispanic respondents reported no drinking at baseline, 16 of whom reported occasional drinking at year 2)

[b]Due to a cell that contained no data, this analysis was inestimable.