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A Twin Study of Drinking and Smoking Onset and Latencies from First Use to Regular Use

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The early onset of alcohol and tobacco use has been associated with increased risk for later substance abuse and dependence problems. This study investigated genetic and environmental influences on age at onset of alcohol and tobacco use by examining twin resemblance for several retrospectively reported onset milestones including age at first use, age at first alcohol intoxication experience, and age at regular use. In addition, we also examined the latency between age at first use and age at regular use of tobacco and alcohol. The subjects were a volunteer sample of older adult twins 50 to 96 years of age. MZ twin correlations for age at first alcohol use and age at first tobacco use were .57 and .44, respectively, compared to .45 and .37 for DZ same-sex twins. MZ twins correlated .30 and .26 for the latencies between first use and regular use of alcohol and of tobacco, while DZ correlations were −.01 and .05, respectively. Biometrical model-fitting results confirmed that familial resemblance for age at first use for both alcohol and tobacco was largely the result of shared environmental factors, while the latencies between first use and regular patterns of use were more genetically influenced. These findings add to a growing body of literature suggesting that initiation of substance use is influenced primarily by environmental rather than genetic factors.

KEY WORDS: Twin study; drinking onset; smoking onset; latency from first to regular use; substance abuse; genetics.

INTRODUCTION

A number of studies of normal population samples have suggested that early experimentation with alcohol and/or tobacco is associated with greater severity and persistence of subsequent substance use (Barnes *et al.,* 1992; Chou and Pickering, 1992; Clapper *et al.,* 1995; Fergusson *et al.,* 1994; Gonzalez, 1989; Grant and Dawson, 1997; Hawkins *et al.,* 1992, 1997; Hays *et al.,* 1986; 1987; Jessor and Jessor, 1975; 1977; Kandel, 1975; Kandel and Faust; 1975, Kandel *et al.,* 1992; Newcomb and Bentler, 1988; Pedersen and Skrondal, 1998; Robins and Przybeck, 1985; Welte and Barnes, 1985; Yamaguchi and Kandel, 1984a, b; Yu and Williford, 1992), and prevention efforts have been designed specifically to prevent or delay initiation of alcohol and tobacco use (Kandel *et al.,* 1992; Williams *et al.,* 1995). However, there is also evidence that substance use normally peaks in late adolescence and early adulthood (Jessor *et al.,* 1991; Kandel and Raveis, 1989; Labouvie, 1996) and then decreases without significant negative consequences for most youth (Jessor *et al.,* 1991; Labouvie *et al.,* 1997; Newcomb and Bentler, 1988). Thus, some early substance experimentation is probably normative and research efforts have begun to focus more on identifying risk factors that may help to distinguish between adolescent-limited and life course-persistent patterns of substance involvement.

The fact that age at onset, per se, appears to be only weakly related to patterns of use and problem substance use in later adulthood may be partly explained

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by evidence from behavioral genetic studies suggesting that environmental factors play an important role in abstinence versus alcohol use (Han *et al.,* 1999; Heath and Martin, 1988; Heath *et al.,* 1989, 1991a, b; Koopmans and Boomsma, 1996; Prescott *et al.,* 1994a; Rose *et al.,* 1999; but see also Heath, 1999; Kaprio *et al.,* 1987; Partanen *et al.,* 1966). In contrast, there is substantial evidence that genetic factors may make a greater contribution to familial resemblance for alcohol consumption patterns such as frequency and quantity of use (Carmelli *et al.,* 1990; Clifford *et al:,* 1984; Gabrielli and Plomin, 1985; Kaprio *et al.,* 1987; Partanen *et al.,* 1966; Prescott *et al.,* 1994a) and for problem drinking and alcoholism (Cloninger *et al.,* 1981; 1988; Heath *et al.,* 1997a, b; Kaij, 1960; Kaprio *et al.,* 1987; Kendler *et al.,* 1992; McGue, 1993; McGue *et al.,* 1992; Pickens *et al.,* 1991; Prescott *et al.,* 1994b).

A similar pattern of findings can be noted in the tobacco use literature, although genetic influences on the initiation versus abstinence of tobacco use may be more substantial than has been reported for alcohol use. For example, several twin studies have suggested that the heritability for smoking initiation is substantial, with estimates ranging between 46 and 84% (Carmelli *et al.,* 1992; Heath and Madden, 1995; Heath and Martin, 1993; True *et al.,* 1997). In terms of regular smoking, the data are very consistent with the alcohol consumption data in indicating that genetic factors appear to play a more important role than environmental influences in predicting which individuals who become smokers progress to long-term persistent smoking (Heath and Madden, 1995; Heath *et al.,* 1998; True *et al.,* 1997). Substantial genetic influences underlying twin resemblance for nicotine dependence have also been reported (True *et al.,* 1999).

Although there is a considerable body of literature on smoking and drinking patterns in adults, less is known about the determinants of age at onset and how quickly one moves from first use to heavier use or problem use. Studies of substance abuse risk suggest that early problem use, which implies a short latency between first exposure and regular use, is an important risk factor for later adult alcoholism and illicit substance abuse. Further, one might speculate that sensitivity to the rewarding properties—and/or insensitivity to the negative aspects—of tobacco and alcohol might be related to how quickly one makes the transition from initial exposure to regular use, and physiological sensitivity to alcohol has been proposed as a potential mediator of the genetic influence on alcoholism risk (Schuckit, 1980, 1985, 1994; Schuckit and Smith, 1997; Pollock, 1992).

In the present study we used retrospective reports from adult twins to examine the genetic and environmental determinants of age at onset for first alcohol and tobacco use, as well as the latency in years between first use and regular use of these substances. We hypothesized that environmental influences would be shown to be more important in determining twin resemblance for age at first use, while genetic factors might make a greater contribution to twin resemblance for the latency between first use and regular use.

METHOD

Subjects

The sample for this study was a subset of twins from a volunteer population-based sample ascertained for research on tobacco and alcohol use. The original sample pool was restricted to Caucasian twins by design (for details on sample ascertainment see Heath *et al.,* 1993). Subjects were adult twins 50 to 96 years of age, originally recruited between 1985 and 1989 through a newsletter published nationally by the American Association of Retired Persons (AARP). Questionnaires were mailed to 12,118 individuals and returned by 9476 subjects, a response rate of about 78%. If either twin responded to this original recruitment, both pair members were mailed a follow-up questionnaire between late 1990 and early 1991. Former responders who were known to be deceased, who requested to be removed from the mailing list, or for whom previous mailings were returned without a forwarding address were excluded from the second mailing, resulting in a potential subject pool of 7622 individuals.

Follow-up questionnaires were returned by 4956 subjects representing a response rate of 65% (approximately 41% of the originally ascertained sample). From this sample, 835 subjects younger than age 50 and 2 with invalid questionnaires were excluded, resulting in a final sample of 4119 individuals. Data for this study came from this follow-up sample. The sample was 74% female (3049 women and 1070 men) with a mean age of 66.8 (SD=7.9) years. Males were slightly older (M=67.3, SD=7.9) than females (M=66.5, SD=7.9) on average, but there was substantial variation in age within gender groups. Males, and particularly male DZ twins, were underrepresented in this sample, which is common in volunteer twin samples. The preponderance of females probably reflects both the earlier mortality of men and the higher tendency of females to participate in volunteer studies.

Demographic data were obtained from the initial assessment and are available for 4040 individuals (98%) in the final sample. At the time of original ascertainment, 72% of the sample was married or cohabitating, with males more likely to be married or cohabitating (88%) than females (67%). In addition, respondents were relatively well educated, with 52% of the females and 65% of the males having some education beyond high school. The sample was generally of middle-class SES, with approximately 43% reporting an annual income of at least $35,000 (for a more detailed description of this sample see Prescott *et al.,* 1994a).

Attrition analyses, done previously (Prescott *et al.,* 1994a), compared responders to the first assessment only to subjects who completed both questionnaires. Results indicated that the final sample tended to have higher educational attainment, higher family income levels, and among men, were more likely to be retired. The final sample was not shown to be further selected on current alcohol consumption, but individuals with a history of alcohol abuse were probably underrepresented.

Twin Sample

From the full sample ($N = 4119$), there were 1287 twin pairs (2,574 individuals) for whom sufficient information was available to make zygosity judgments. Zygosity determination was made through questionnaire procedures assessing the degree of physical similarity among the twins. The classification scheme was conservative, with misclassifications most likely to be monozygotic (MZ) twins incorrectly assigned as dizygotic (DZ) twin pairs. Therefore, any biasing effects would be in the direction of overestimating the similarity of DZ twins, which would lead to the underestimation of the importance of genetic influences in our analyses. The classification of same-sex twins based on similar questionnaire items has demonstrated approximately 95% accuracy compared to blood typing (Cederlöf *et al.,* 1961; Kasriel and Eaves, 1976). For more details regarding zygosity determination in this sample, see Prescott *et al.* (1994a).

Genetic analyses used only twin pairs with complete data. Twin pairs where *either* twin had never tried alcohol or tobacco (complete abstainers)—or for whom age at onset was not reported—were omitted from the relevant genetic analyses. Further, individuals who never reported *regular use* of alcohol or tobacco (weekly use of alcohol and daily use of tobacco) would not have transition latency data (latency in years between age at first use and age at regular use). Thus, twin pairs where one or both of the twins never progressed to regular use were necessarily excluded from the relevant transition latency analyses. Analyses were performed on the alcohol and tobacco use measures separately, however, use of both substances was not required. The numbers of twin pairs utilized for our genetic analyses of the substance use milestones and transition latencies are shown in Tables III and IV, respectively.

There is no "correct" method for dealing with missing data in our analyses, and the omission of subjects who have not obtained particular alcohol or tobacco use milestones can be problematic, particularly in younger samples where censoring is likely. However, given the age range of our subjects (50 to 96 years of age), a strength of our sample is that most of our subjects have passed through the age of risk for obtaining these use milestones. That is, it is unlikely that many of our participants who have not yet begun using alcohol or tobacco regularly will do so at some later point in their lives. The omission of complete abstainers (i.e., subjects who had never tried alcohol or tobacco even once) is problematic if abstinence is simply the extreme end of a single liability dimension for alcohol and tobacco consumption. However, existing evidence does not provide strong support for including abstainers in a single liability dimension for alcohol consumption (Heath *et al.,* 1991a,b) or cigarette consumption (Meyer *et al.,* 1992).

Measures

Alcohol Use. Alcohol use milestones were assessed using the following questionnaire items embedded within the 16-page follow-up (1990–1991) mail-out survey concerning health, habits, and opinions: (1) At what *age* did you first drink alcohol—first time ever? (2) At what *age* did you first become intoxicated? and (3) At what *age* did you start drinking alcohol at least once a week? Two transition latency indices were computed by subtracting (1) age at first intoxication experience from age at first use ever and (2) age at weekly alcohol use from age at first use ever. The latency between age at first intoxication and age at weekly use was not computed because some subjects reported that their first intoxication experience occurred after the age at which they reported weekly use (i.e., this was not a "normative" transition for many subjects).

Tobacco Use. Similar tobacco use milestones were assessed using the following questionnaire items:(1) At what *age* did you smoke your first ciga-

rette? (2) At what *age* did you start smoking at least one cigarette a day? and (3) At what *age* did you start smoking at least a half-pack of cigarettes (10 cigarettes) a day? Two latency scores were computed by subtracting age of regular use (at least one cigarette a day and half a pack or more per day) from age at first use. The latency between smoking at least one cigarette a day and smoking half a pack per day was not computed because age at heavier use sometimes preceded age at daily use of at least one cigarette. This occurred because some subjects responded to the questionnaire items consistent with their decreased daily use over the lifecourse (i.e., patterns of heavy use sometimes preceded later patterns of reduced use). Since this transition was not normative for all subjects, it was not utilized. Further, since our questionnaire survey specifically referred to cigarette use only, individuals using other tobacco products could not be assessed.

Data Transformation. Age at onset for the various drinking and smoking milestones was positively correlated with age at assessment in our sample (correlations ranged between .11 and .16). That is, there was a modest trend for older individuals to estimate later ages at onset than younger individuals. This may represent real age cohort differences, but there is also evidence from prospective longitudinal data to suggest that there is a systematic tendency for most individuals to shift their estimates of age at onset upward as they get older (Labouvie *et al.,* 1996). Age at onset also correlated negatively with gender (correlations ranged between −.15 and −.27), with men reporting earlier ages of onset for all of our smoking and drinking milestones than women. Unfortunately, our samples sizes were insufficient to examine age and gender effects in our genetic analyses, so these effects were partialed from all of our measures prior to genetic analysis. That is, our genetic analyses were performed on residual scores for each of the onset milestones and latency measures, partialing out the effects of gender, linear and quadratric age effects, and the interactions between these effects using standard regression procedures. In addition, our age at onset and latency measures all showed significant positive skewness, so all scores were further transformed to approximate normality using a rank normalizing procedure (Blom, 1958).

Statistical Analyses

We used standard univariate biometrical twin models (Neale and Cardon, 1992) to estimate the genetic and environmental contributions to population variance in the age at onset for three alcohol use milestones (age at first use ever, age at first intoxication experience, and age at which regular weekly alcohol use began), three tobacco use milestones (age at first use ever, age at which daily smoking of at least one cigarette per day began, and age at which daily smoking of half a pack or more per day began), and the latency in years between these various use milestones. As noted above, two of the six possible transition latencies were not examined in detail because they were not normative transitions (i.e., the ordering of the events was not uniform enough to make use of the data as representing a hard transition from one milestone to the next).

Under the basic twin model, individual differences in the age at which different use milestones occur, and in the latencies between different use milestones, are assumed to arise from four potential latent sources: additive genetic effects (A); nonadditive genetic effects (D); shared environmental effects (C), which include all nongenetic influences that increase the similarity between the members of a twin pair; and nonshared environmental effects (E), which include all other nongenetic effects (including measurement error) that contribute to within-pair differences among twins. MZ twins are genetically identical, thus they share, or correlate perfectly for, all additive and nonadditive genetic effects. On average, DZ twins share half the additive effects of their genes and have a one-in-four chance of sharing nonadditive genetic effects at any given locus. Thus, the additive and nonadditive genetic effects influencing the members of DZ twin pairs are expected to correlate .5 and .25, respectively. Given equivalent environmental influences on both types of twins, shared environmental influences are expected to correlate 1.0 across the members of a twin pair for both zygosity groups. Other implicit assumptions of the basic twin model are that genetic and environmental latent effects are uncorrelated and combine additively and that mating is random in the parent generation of the twins.

Given these theoretical expectations derived from quantitative genetic theory (Falconer, 1989), a model for the expected twin covariance structure can be specified and compared to the observed twin covariance structure using standard structural equation modeling procedures. Univariate models were fit directly to the observed 2×2 twin covariance matrices for each of our measures and model parameters were estimated using maximum-likelihood procedures operationalized in the statistical package Mx (Neale, 1998). The fit of alternative nested models was evaluated using standard χ^2 difference tests (for details see Neale and Cardon, 1992).

Note that with twins reared together in the same household, as is the case in our sample, not all of the latent sources of variance described above can be estimated simultaneously. Shared environmental influences (C) are confounded with nonadditive genetic influences (D) in such data. Thus, the influence of one source cannot be estimated without assuming the absence of the other. Nevertheless, the plausibility of obtained parameter estimates and the fit of alternative models to the observed data (though no formal statistical basis for preferring one model over another is available when comparing nonnested models) can provide guidelines for model selection. To evaluate the relative fit of nonnested models we used Akaike's (1970) information criterion (AIC).

RESULTS

Descriptive Analyses

Table I shows the mean ages at onset for the untransformed alcohol and smoking use milestones for the full sample and for males and females separately. Untransformed data are shown here for descriptive purposes only. All statistical analyses were performed on transformed scores.

Almost 90% of our subjects had at least tried alcohol and about half had tried cigarettes (i.e., reported an age at onset for first use ever). There were no substantial gender differences in the proportions of our male and female subsamples who had used alcohol at least once (93% of the men and 88% of the women), while greater differences are evident regarding cigarette use (69% of the males reported at least trying cigarettes, compared to only 46% of the females). Nearly twice as many men as women reported regular use of alcohol and tobacco, and age at onset for all of the drinking and smoking milestones occurred earlier for men than women. For both men and women first use and regular use of cigarettes occurred earlier, on average, than first use and regular use of alcohol.

Table I also shows mean age-at-onset milestones for weekly versus nonweekly alcohol users and for daily versus nondaily cigarette users. Age at onset for first alcohol use in subjects reporting weekly use precedes age at first use in nonweekly alcohol users by about 2 years. In contrast, when comparing daily cigarette smokers (defined in the bottom portion of Table I as smoking at least 10 cigarettes per day) and nondaily cigarette users (all others who had tried cigarettes at least once), age at first use in daily smokers preceded that in nondaily smokers by less than a year. Age at first use was essentially equivalent between the groups when daily smoking was defined as smoking at least one cigarette per day, so it is not shown. In fact, only 17% (N=366) of our subjects who had ever tried cigarettes (N=2135) did not progress to smoking at least one cigarette daily at some point in their lives, and only 33% (N=697) did not progress to smoking a half pack or more. These data indicate a relatively high level of regular smoking in our older age cohort, particularly for males. In comparison, over half (57%) of our subjects who ever tried alcohol did not progress to weekly alcohol use.

Table II shows the untransformed mean transition latencies between the different drinking and smoking milestones. Mean latencies (defined as the difference in years between milestones) are shown for the full sample and for males and females separately. Note that, on average, about 6 years elapsed between first alcohol use and first intoxication experience, and about 10 years between age at first use and age at which weekly alcohol use began for those progressing to weekly use. Latencies for males are about 2 years shorter, on average, than for females. For tobacco users who progressed to smoking at least one cigarette per day, the mean latency was about 2 years between age at first use and age at daily smoking. About 4 years, on average, elapsed between age at first use and age at smoking a half-pack or more daily for those progressing to that level of smoking. In contrast to the gender differences in latencies between alcohol use milestones, latencies between smoking milestones were about equal for men and women.

Twin Correlations

Age-at-Onset Milestones. Twin correlations and covariance matrices utilized in our genetic analyses were computed from the transformed measures. Because MZ twins are genetically identical, while DZ twins, on average, share only about half of their segregating genes, significantly higher MZ than DZ correlations would indicate genetic influences underlying twin resemblance. Equivalent MZ/DZ correlations would connote shared environmental mechanisms. Comparisons of the MZ and DZ twin correlations in Table III suggest that there are substantial shared environmental influences on the age at onset, or age at first use ever, for both alcohol and tobacco. MZ twin correlations also were not substantially higher than DZ correlations for age at first alcohol intoxication experience or age at daily tobacco use (at least one cigarette

Table I. Age at Onset for Drinking and Smoking Milestones

	Full sample (N = 4119)			Males (N = 1070)			Females (N = 3049)		
Use milestone	%[a]	M	SD	%	M	SD	%	M	SD
Alcohol									
Age at first use ever	89	18.9	6.5	93	17.2	6.0	88	19.6	6.5
Age at first intoxication	46	23.1	7.9	69	21.2	6.1	38	24.4	8.7
Age at weekly use[b]	37	27.9	10.9	53	24.2	8.7	32	30.3	11.5
Among non-weekly users only									
Age at first use ever	100	19.8	7.0	100	18.3	7.2	100	20.2	6.9
Age at first intoxication	34	23.0	7.4	55	21.9	5.7	28	23.5	7.5
Among weekly users only									
Age at first use ever	100	17.6	5.4	100	16.3	4.7	100	18.4	5.6
Age at first intoxication	78	23.2	8.2	89	20.8	5.7	72	25.0	9.3
Tobacco									
Age at first use ever	52	17.5	5.7	69	15.6	4.7	46	18.5	5.9
Age at daily use (1 cig/day)[c]	43	19.9	5.9	60	18.4	4.2	37	20.7	6.5
Age at daily use (10 cigs/day)[d]	35	21.5	6.0	53	19.8	4.1	29	22.6	6.8
Among nondaily users only									
Age at first use ever	100	17.2	5.9	100	15.2	5.9	100	18.0	5.8
Among daily (1 cig/day) users									
Age at first use ever	100	17.5	5.6	100	15.6	4.5	100	18.6	5.9
Age at assessment		66.8	7.9		67.3	7.9		66.5	7.9
Median age at assessment		66			67			66	

Note. M, mean age at use milestone; SD, standard deviation.
[a] Percentage of sample reaching the use milestone.
[b] Weekly alcohol use—drink alcohol *at least* once per week.
[c] Smoke *at least* one cigarette per day.
[d] Smoke *at least* half a pack (10 cigarettes) per day.

Table II. Mean Ages for Drinking and Smoking Transition Latency Measures

	Full sample		Males		Females	
Transition latency measure[a]	M	SD	M	SD	M	SD
Alcohol						
Age at first use ever to age at first intoxication	6.1	7.4	5.0	5.9	6.9	8.2
Age at first use ever to age at weekly use [b]	10.2	9.9	7.9	8.3	11.6	10.6
Tobacco						
Age at first use ever to age at daily use (1 cig/day)[c]	2.4	4.5	2.7	4.0	2.2	4.7
Age at first use ever to age at daily use (10 cigs/day)[d]	4.4	5.1	4.3	4.5	4.4	5.4

Note. M, mean latency (years); SD, standard deviation.
[a] Latency—difference in years between age at onset for use milestones.
[b] Weekly alcohol use—drink alcohol *at least* once per week.
[c] Smoke *at least* one cigarette per day.
[d] Smoke *at least* half a pack (10 cigarettes) per day.

per day), and the MZ correlation for age at heavier regular cigarette use (half a pack per day or more) was estimated to be lower than the corresponding DZ twin correlation. In general, our pattern of twin correlations is consistent with substantial shared environmental contributions to the timing of these substance use milestones. Only age at weekly alcohol use appears to be substantially influenced by genetic effects.

Transition Latencies. Table IV shows the MZ and DZ twin correlations for the latency (difference in years) between first use of alcohol and tobacco and later use milestones for these substances. Comparison of the

Table III. MZ and DZ Twin Correlations for Age-at-Onset Milestones

Onset milestone	r_{MZ}	$(N)^a$	r_{DZ}	$(N)^a$
Alcohol				
Age at first use ever	.57 ± .04	(563)	.45 ± .07	(240)
Age at first intoxication	.43 ± .07	(222)	.41 ± .10	(96)
Age at weekly use[b]	.40 ± .08	(158)	.18 ± .13	(62)
Tobacco				
Age at first use ever	.44 ± .06	(312)	.37 ± .08	(136)
Age at daily use (1 cig/day)[c]	.41 ± .07	(240)	.34 ± .10	(97)
Age at daily use (10 cigs/day)[d]	.35 ± .08	(174)	.50 ± .12	(69)

Note. r_{MZ} = MZ twin (product–moment) correlation ± standard error of estimate; r_{DZ} = DZ twin (product–moment) correlation ± standard error of estimate.
[a] N = number of twin pairs with complete data.
[b] Weekly alcohol use—drink alcohol *at least* once per week.
[c] Smoke *at least one* cigarette per day.
[d] Smoke *at least* half a pack (10 cigarettes) per day.

Table IV. MZ and DZ Twin Correlations for Transition Latency Variables

Transition latency variable[a]	r_{MZ}	$(N)^b$	r_{DZ}	$(N)^b$
Alcohol				
Age at first use ever to age at first intoxication	.32 ± .07	(212)	.26 ± .11	(89)
Age at first use ever age at weekly use	.30 ± .08	(151)	−.01 ± .14	(58)
Tobacco				
Age at first use ever to age at daily use (1 cig/day)	.26 ± .07	(235)	.05 ± .10	(97)
Age at first use ever to age at daily use (10 cigs/day)	.37 ± .08	(170)	.12 ± .12	(69)

r_{MZ} = MZ twin (product–moment) correlation ± standard error of estimate; r_{DZ} = DZ twin (product–moment) correlation ± standard error of estimate.
[a] Latency—difference in years between age at onset for use milestones.
[b] N = number of twin pairs with complete data.

pattern of MZ and DZ twin correlations suggests that the latency between age at first alcohol use and age at which weekly alcohol use began appears to be more strongly influenced by genetic factors than the timing or age at which these use milestones occurred (shown in Table III). A similar pattern is also evident for the smoking latencies as well. Note that the DZ correlations for two of the latency measures are lower than might be expected given additive genetic influences, suggesting that nonadditive genetic factors may play a role. However, special MZ environmental factors (i.e., a greater correlation between the environments experienced by MZ twin pairs compared to DZ twins) cannot be ruled out as an alternative explanation. Note that because there are fewer DZ twins than MZ twins, the standard errors around the DZ twin correlations are larger than for the MZ correlations. Thus, the relatively low magnitudes of the DZ correlations may simply reflect chance sampling fluctuations.

Special MZ Environment. To examine the possibility that the relevant environments shared by MZ and DZ might not be equivalent, we examined twin similarity for twins in frequent contact with each other (at least weekly) versus those in less frequent contact. If the environmental influences on the transition latencies are more highly correlated among MZ pairs than DZ pairs, one might expect that MZ twins in more frequent contact with one another would show greater similarity than those in less frequent contact. Our questionnaire data included two items assessing current (at time of assessment) frequency of contact (1) How often do you and your twin see each other? and (2) If you live apart, how frequently do you and your twin contact each other by telephone or letter? Responses were coded by a 6-point Likert scale for the first item (1=we live together; 2=almost everyday; 3=at least once a week; 4=once or twice a year; 5=a few times a year; 6=once a year or less) and a 5-point scale for the sec-

ond item (omitting the first response category). Because of our relatively small samples, we were unable to compute meaningful twin correlations separately for each category, so the categories were collapsed into two groups: (1) at least weekly contact and (2) less frequent contact.

We did observe MZ/DZ differences in contact for these groups. Most twins were now living apart. Only about 5% of MZ twin pairs and 4% of the same-sex DZ pairs reported still living together at the time of assessment. Approximately 32% of MZ twins reported physically seeing their cotwin at least weekly, compared to only 20% of DZ same-sex twin pairs. In terms of contact by phone or letter for those living apart, 53% of MZ twins reported at least weekly contact, compared to 33% of the DZ twins. However, comparing twin correlations for the two frequency of contact groups did not show a general pattern of greater similarity for twins in more frequent contact. For the transition latency between age at first alcohol use and age at weekly use, MZ twins who saw their cotwin at least weekly (N=44 pairs) correlated .33, compared to .29 for MZ twins seeing their cotwin less frequently (N=107 pairs). However, there was a significant difference when examining frequency of contact by phone or letter. Here MZ twins in weekly contact (N=64 pairs) correlated .44 for the transition latency, compared to only .10 for MZ twins in less frequent contact (N=71 pairs). A mixed pattern was also found for the two smoking transition latency measures. For the transition between first tobacco use and smoking at least one cigarette per day, the MZ correlations were .15 and .34 (N = 78 and 156 pairs) and .26 and .25 (N = 115 and 103) for the high- and low-contact groups for frequency of seeing each other and for contact by phone or letter, respectively. Regarding the transition from first use to smoking at least half a pack of cigarettes or more per day, the same MZ twin correlations were .36 and .37 (N = 52 and 118 pairs) and .33 and .42 (N = 84 and 74 pairs). In general, the patterns among the MZ twins are inconclusive regarding the potential for special MZ environment effects. DZ twin sample sizes for the frequency of contact groups were too small for adequate comparisons but also showed a mixed pattern. It is important to point out, however, that our frequency of contact data referred to patterns of cotwin contact at the time of assessment (median age=66), not during the developmental periods when the majority of our twins were initiating substance use. Because patterns of contact are likely to change over the life course, frequency of contact during adolescence and early adulthood would have provided a more relevant test for special MZ environment effects.

Model-Fitting Results

Onset Milestones. Table V shows the results of fitting univariate biometrical twin models to each of the drinking and smoking milestones. The best-fitting models by the AIC are shown in boldface type. Our model-fitting analyses confirm the importance of shared environmental influences on the age at which these substance use milestones occurred. For all of the milestones except age at first alcohol use and age at weekly alcohol use, dropping additive genetic effects from the full ACE model did not result in a significant decrement in model fit by χ^2 difference test. Thus, for four of the six substance use milestones, twin resemblance could be adequately explained by a model including only shared environmental influences. Although additive genetic effects did contribute to twin resemblance for age at first use of alcohol, the estimate of shared environmental effects wals also substantial—accounting for approximately 35% of the phenotypic variance in age at first use, compared to 21% attributable to additive genetic effects.

No model provided a satisfactory fit to the age at weekly alcohol use data by χ^2 test, but the best-fitting model by AIC suggested the importance of genetic factors and absence of shared environmental influences in explaining twin resemblance for the onset of weekly alcohol use. Examination of the expected and observed MZ and DZ covariance matrices for this milestone indicated twin 1 and twin 2 differences in variance, and greater DZ than MZ variances were responsible for the poor model fit. However, since the DZ sample was relatively small (62 pairs) and this pattern was not found for any of the other use milestones, alternative ad hoc models were not explored.

Transition Latencies. Univariate model-fitting results for each of the four transition latencies are shown in Table VI. In contrast to the findings presented in Table V regarding the timing or age at which the different substance use milestones occurred, the latency in years between age at first alcohol use—or first tobacco use—and the age at which regular weekly drinking or daily smoking occurred appears to be more greatly influenced by genetic factors. A simple additive genetic model was adequate to explain twin resemblance for three of the four transition latencies. Shared environmental effects made a contribution to the latency between first alcohol use and first intoxi-

Table V. Model-Fitting Results: Alcohol and Tobacco Use Milestones

Model	h^2	c^2	e^2	χ^2	df	p	AIC
Alcohol							
Age at first use							
ACE[a]	**.21**	**.35**	**.43**	**0.35**	**3**	**.95**	**−5.65**
(CI_{95})	(.02–.44)	(.14–.53)	(.38–.49)				
AE	.58	—	.42	10.32	4	.04	2.32
CE	—	.54	.46	4.91	4	.30	−3.09
Age at first intoxication							
ACE	.09	.35	.56	1.65	3	.65	−4.36
AE	.46	—	.54	5.31	4	.26	−2.69
CE[a]	—	**.43**	**.57**	**1.90**	**4**	**.76**	**−6.10**
(CI_{95})		(.33–.51)	(.49–.67)				
Age at weekly use							
ACE	.43	.00	.57	13.36	3	<.01	7.36
AE[a]	**.43**	—	**.57**	**13.36**	**4**	**.01**	**5.36**
(CI_{95})	(.29–.55)		(.44–.71)				
CE	—	.31	.69	19.78	4	<.01	11.78
Tobacco							
Age at first use							
ACE	.14	.30	.56	1.19	3	.76	−4.81
AE	.46	—	.54	4.74	4	.32	−3.26
CE[a]	—	**.42**	**.58**	**1.90**	**4**	**.75**	**−6.10**
(CI_{95})		(.34–.49)	(.51–.66)				
Age at daily use (1 cig/day)							
ACE	.15	.26	.59	2.43	3	.49	−3.57
AE	.43	—	.57	4.23	4	.38	−3.77
CE[a]	—	**.39**	**.61**	**3.07**	**4**	**.55**	**−4.93**
(CI_{95})		(.30–.48)	(.52–.70)				
Age at daily use (10 cigs/day)							
ACE	.00	.40	.60	3.02	3	.39	−2.98
AE	.41	—	.59	9.94	4	.04	1.94
CE[a]	—	**.40**	**.60**	**3.02**	**4**	**.55**	**−4.98**
(CI_{95})		(.29–.50)	(.50–.71)				

Note. (CI_{95}) = 95% confidence intervals for parameter estimates; A = additive genetic effects; C = shared environmental effects; E = nonshared environmental effects; h^2 = proportion of variance explained by additive genetic effects; c^2 = proportion of variance explained by shared environmental effects; e^2 = proportion of variance explained by nonshared environmental effects.
[a] Best-fitting model by AIC criteria.

cation experience only. Again, the relatively low DZ twin correlations for two of the transition latencies may indicate the possibility of nonadditive genetic effects or special MZ environment effects, but given our relatively small DZ twin samples, there was insufficient power to detect these influences.

DISCUSSION

Numerous behavioral genetic studies have investigated the extent to which genetic and environmental factors influence tobacco and alcohol abstinence and smoking and drinking consumption patterns in adulthood (for a review see Heath, 1999). Much less, however, is known about the determinants of age at onset and how quickly one makes the transition from initial use to regular or problem use. In this study we used retrospective reports from adult twins to estimate the magnitude of the genetic and environmental sources of variance in age at onset for first tobacco and alcohol use and for the transition latency in years between age at first use and age at regular use. We hypothesized that twin resemblance for age at onset would be more strongly influenced by shared environmental factors, while twin resemblance for the latency between first use and regular use might be influenced more by genetic factors.

Our data generally support both hypotheses. For age at first cigarette use estimates of the proportion of variance explained by additive genetic effects (h^2),

Table VI. Model-Fitting Results: Latencies from First Use to Regular Alcohol and Tobacco Use

Transition model	h^2	c^2	d^2	e^2	χ^2	df	p	AIC
Alcohol								
Age at first use to age at first intoxication								
ACE	.11	.21	—	.68	0.84	3	.84	−5.16
AE	.33	—	—	.67	1.83	4	.77	−6.18
CE[a]	—	**.30**	—	**.70**	**1.07**	**4**	**.90**	**−6.93**
(CI_{95})		(.19–.40)		(.60–.81)				
Age at first use to age at weekly use								
ADE	.00	—	.31	.69	5.65	3	13	−.35
AE[a]	**.28**	—	—	**.72**	**7.17**	**4**	**.13**	**−.83**
(CI_{95})	(.13–.43)			(.57–.87)				
Tobacco								
Age at first use to age at daily use (1/day)								
ADE	.00	—	.25	.75	4.05	3	.26	−1.95
AE[a]	**.24**	—	—	**.76**	**4.60**	**4**	**.33**	**−3.40**
(CI_{95})	(.12–.35)			(.65–.88)				
Age at first use to age at daily use (10/day)								
ACE	.37	.00	—	.63	4.28	3	.23	−1.72
AE[a]	**.37**	—	—	**.63**	**4.27**	**4**	**.37**	**−3.72**
(CI_{95})	(.23–.49)			(.51–.77)				
CE	—	.30	—	.70	8.25	4	.08	.25

Note:(CI_{95}) = 95% confidence intervals for parameter estimates; A = additive genetic effects; C = shared environmental effects; D = nonadditive genetic effects; E = nonshared environmental effects; h^2 = proportion of variance explained by additive genetic effects; c^2 = proportion of variance explained by shared environmental effects; d^2 = proportion of variance explained by nonadditive genetic effects; e^2 = proportion of variance explained by nonshared environmental effects.

[a] Best-fitting model by AIC criteria.

shared environmental effects (c^2), and nonshared environmental effects (e^2) obtained from the full ACE model were 14, 30, and 56%, respectively. Dropping additive genetic effects resulted in a minimal and nonsignificant decrement in model fit (χ^2 *change* = .17, p = .40). Dropping shared environmental effects yielded a poorer fit to the data—though the χ^2 difference was also not significant, at p = .05 (χ^2 *change* = 3.55, p = .06). The best-fitting model by AIC required only shared environmental effects to explain twin resemblance for age at onset of first cigarette use. For age at first alcohol use, the full ACE model was necessary to obtain an acceptable fit to the data. Shared environmental influences were estimated at 35% and the contribution of additive genetic influences on twin resemblance for age at first alcohol use was estimated as 21%. Our findings for onset of alcohol use are similar to those reported by Prescott and Kendler (1999), though they report somewhat stronger evidence for genetic effects, particularly in females (h^2 = .36 and c^2 = .16 in women, h^2 = .22 and c^2 = .23 in men).

In general, our results suggest that shared environmental factors make a substantial contribution to twin resemblance for age at onset for first tobacco and first alcohol use. Further, this general pattern was found for five of the six substance use milestones we examined. That is, not only did shared environment explain a sizable proportion of the variance in age at onset of use, but also our data suggest that similar environmental processes underlie twin similarity for the timing of the first alcohol intoxication experience, as well as when daily smoking occurs. Only resemblance for age at regular drinking, defined as at least weekly alcohol use, appeared to be substantially influenced by genetic factors. These findings indicate that shared environmental factors may explain most of the differences between families in the timing of many substance use milestones. They also suggest that early experimentation and even the age at onset for regular tobacco use are likely to be better indicators of familial cultural influences than of biological vulnerability or risk for persistent substance use.

In contrast, three of the four transition latencies we examined showed little evidence for shared environmental influences on twin resemblance. Only the transition from first alcohol use to first intoxication experience appeared to be substantially influenced by environmental factors. This may reflect, in part, the fact

that for many subjects (20% of those who had used alcohol) their first experience with alcohol was also their first intoxication experience, but it also suggests that an early age at onset for first intoxication experience is not likely to be an important indicator of biological risk for more persistent alcohol use. For the other three transitions we examined, a simple additive genetic model was sufficient to explain twin resemblance for the transition latencies. These data suggest that how quickly one makes the transition from first use to regular use of tobacco and alcohol may be more genetically mediated than the timing or onset of the transitions themselves. However, special MZ environment effects could not be ruled out as an alternative explanation of the data.

Future research would benefit from prospective longitudinal data where transitions could be assessed without relying on long-term recall of substance use milestones or events. Although a number of studies have examined familial resemblance for substance use transitions, to our knowledge, no other studies to date have examined twin resemblance for transition *latencies* between first use and other patterns of use or problem use. How quickly one moves from one stage to another may be more predictive of later substance use patterns than simply whether or not an individual makes a particular transition. Further, physiological sensitivity to alcohol has been proposed as a potential mediator of the genetic influence on alcoholism risk (Schuckit, 1980, 1985, 1994; Schuckit and Smith, 1997; Pollock, 1992). Clearly, sensitivity to the rewarding properties—and/or insensitivity to the negative aspects—of tobacco and alcohol would be expected to be related to how quickly one makes the transition from initial exposure to regular use.

Limitations

A number of methodological limitations of the current study should be discussed. First, this study relied on the retrospective reporting of the age at onset for various substance use events or milestones. Particularly in a sample of older individuals (median age at assessment, 66 years), such data are likely to be influenced by reporting biases (Brewer, 1988; Henry *et al.,* 1994; Robins *et .al.,* 1985). However, it is unlikely that such biases would be substantially different for MZ and DZ twins; it would simply introduce measurement error in recall for all participants. Obviously, prospective longitudinal data with shorter recall intervals would be preferable. However, an important advantage of this sample was that most individuals were past the age of highest risk for initiating alcohol and tobacco use, and lifetime versus current patterns of use were utilized, so censoring problems would be expected to be minimal compared to younger samples. Further, although exact ages at onset may not be recalled accurately, studies investigating the reliability of retrospectively reported age at onset suggest that the relative rank ordering of subjects is probably fairly stable (Henry *et al.,* 1994; Labouvie *et al.,* 1997).

Second, our samples sizes were relatively small, particularly for examining transition latencies. Although our genetic analyses suggested that a simple additive genetic model underlying twin resemblance for three of the four transition latencies could not be rejected, there was relatively low power to test alternative models. The observed pattern of MZ and DZ twin correlations suggests that the potential for nonadditive genetic effects and/or special MZ twin environmental influences should be investigated in larger samples. Further, our sample sizes restricted our ability to examine gender differences in our genetic analyses. Heath and Martin (1988) have reported gender differences in the genetic and environmental factors influencing the onset of alcohol use in a sample of Australian adult twins. In our analyses, the small number of males precluded tests of gender differences.

It should also be reemphasized that our sample was an older cohort, born between 1904 and 1937. The environmental culture in the United States regarding smoking and drinking practices has changed considerably since the time when the majority of our subjects passed through the period of greatest risk for initiating alcohol or tobacco use. In addition, 75% of our sample were women and our subjects were generally of middle-class SES. These sample characteristics should be considered when interpreting our results, particularly with regard to smoking and drinking behaviors for which gender differences in prevalence and exposure are known to exist.

Finally, in the basic twin model it is assumed that mating is random in the parents of the twins for the phenotypes of interest. Evidence suggests that the assumption of random mating for drinking and smoking behaviors is incorrect, thus some proportion of the variance attributable to shared environment in our analyses is likely due to assortative mating in the parents of our twins.

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