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EXTENDED REPORT

Trajectories of Alcohol and Drug Use and Dependence From Adolescence to Adulthood: The Effects of Familial Alcoholism and Personality

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This study describes trajectories of substance use and dependence from adolescence to adulthood. Identified consumption groups include heavy drinking/heavy drug use, moderate drinking/experimental drug use, and light drinking/rare drug use. Dependence groups include alcohol only, drug only, and comorbid groups. The heavy drinking/heavy drug use group was at risk for alcohol and drug dependence and persistent dependence and showed more familial alcoholism, negative emotionality, and low constraint. The moderate drinking/experimental drug use group was at risk for alcohol dependence but not comorbid or persistent dependence and showed less negative emotionality and higher constraint. Familial alcoholism raised risk for alcohol and drug use and dependence in part because children from alcoholic families were more impulsive and lower in agreeableness.

Substance use and substance use disorders show systematic age-related patterns, with adolescent onset, peaks in use and diagnosed disorders in "emerging adulthood" (ages 18–25; Arnett, 2000), and declines in use after the mid-twenties (Bachman, Wadsworth, O'Malley, Johnston, & Schulenberg, 1997; Chen & Kandel, 1995). However, despite these overall trends, there is also considerable heterogeneity in the developmental course of substance use and substance use disorders, and this heterogeneity may be of etiological significance. For example, researchers have suggested that alcohol disorders vary in their antecedents as a function of their age of onset (Cloninger, 1987) and course ("developmentally limited" to emerging adulthood vs. persistent; Zucker, Fitzgerald, & Moses, 1995). Thus, to understand the etiology of substance use disorders, it may be important to distinguish among groups who follow different trajectories over time in addition to comparing those who do and do not manifest the disorder at a single time point.

For these reasons, researchers have attempted to identify groups who follow a particular developmental course and compare them with other groups in terms of the antecedents and consequences of their substance use trajectories (Bennett, McCrady, Johnson, & Pandina, 1999; Kandel & Chen, 2000; Schulenberg, Wadsworth, O'Malley, Bachman, & Johnston, 1996). Recent advances in mixture modeling have provided a methodology to empirically identify heterogeneity in trajectories over time by clustering individuals according to their trajectories rather than their raw scores (B. O. Muthén & Shedden, 1999; Nagin, 1999). Using these methods, researchers have examined trajectories of alcohol-related outcomes in adolescence (Colder, Campbell, Ruel, Richardson, & Flay, 2002), from adolescence to emerging adulthood (Chassin, Pitts, & Prost, 2002; Hill, White, Chung, Hawkins, & Catalano, 2000; Tucker, Orlando, & Ellickson, 2003) and from emerging adulthood to adulthood (Jackson, Sher, & Wood, 2000). Other studies have examined cigarette smoking or tobacco dependence (Chassin, Presson, Pitts, & Sherman, 2000; Colder et al., 2001; Jackson et al., 2000; White, Pandina, & Chen, 2002). It is difficult to characterize this literature because of its wide variation in dependent measures and the varying ages of participants. However, studies have often identified an early onset, heavily using group, more normative non-using or light-using groups, and, sometimes, later onset groups as well as distinguishing more persistent trajectories from developmentally limited groups.

In terms of risk factors, a family history of substance use has been associated with trajectories that are characterized by both early onset and persistence. For example, a family history of smoking has been associated with early and persistent cigarette smoking (Chassin et al., 2000), and parental alcoholism has been associated both with an early onset of drinking (Chassin et al., 2002; Dawson, 2000) and with trajectories of persistent alcohol

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use disorders (Jackson et al., 2000). In terms of intrapersonal characteristics, personality traits that reflect disinhibition or behavioral undercontrol (e.g., sensation seeking, impulsivity, low constraint) are associated with trajectories that show earlier onset, heavier consumption, and greater persistence of alcohol use (Bennett et al., 1999; Chassin et al., 2002; Hill et al., 2000) as well as persistence of alcohol disorders (Jackson et al., 2000). In addition, negative emotionality has predicted escalating trajectories of adolescent alcohol use (Chassin et al., 2002; Colder et al., 2002), although, in one study, depressive symptoms did not predict trajectories of alcohol and tobacco disorders in adulthood after correlated risk factors were controlled (Jackson et al., 2000).

This rapidly expanding literature has provided important insights into the heterogeneity of substance use trajectories (particularly for alcohol and tobacco). However, this literature also has some limitations that the current study attempts to address. Most importantly, with the exception of Jackson et al. (2000), who examined alcohol and tobacco disorders, past studies have considered a single substance in isolation without addressing relations with other forms of substance use. However, trajectories of comorbid alcohol and drug outcomes may have important etiological significance because the factors that produce comorbid disorders may differ from those that produce a single disorder. For example, recent twin data (Kendler, Prescott, Myers, & Neale, 2003) showed a shared genetic liability for alcohol and drug dependence that was reflected in their loading on a common externalizing factor but also showed significant disorder-specific genetic influence. This suggests that etiological mechanisms may reflect shared pathways that elevate risk for both alcohol and drug disorders as well as specific mechanisms that elevate risk for either alcohol or drug disorder alone. For example, personality diatheses for behavioral undercontrol could produce shared vulnerability for both alcohol and drug disorders (Krueger et al., 2002; Vanyukov et al., 2003). Undercontrolled individuals are less likely to be constrained by social norms or laws, they are more likely to be embedded in social contexts that support alcohol and drug use, and they have stronger sensation-seeking motives. These pathways could produce both alcohol and drug use and disorders (see Sher & Trull, 1994, for a discussion of mechanisms relating personality to substance use disorders). In contrast, the disorder-specific genetic influences reported by Kendler et al. (2003) might reflect mechanisms that determine alcohol or drug outcomes in isolation. For example, heritable individual differences in alcohol sensitivity or metabolism might raise risk for alcohol disorders without comorbid drug disorder (Vanyukov et al., 2003). Thus, trajectory groups of alcohol or drug outcomes alone (compared with comorbid outcomes) might reflect differences in underlying mechanisms.

In addition, examining bivariate trajectories of alcohol and drug outcomes can help to determine whether risk factors that predict trajectories for one substance are true predictors or are actually related to other co-occurring substance use. For example, McGue, Slutske, and Iacono (1999) related personality to alcohol and drug disorders at one point in time. Their data suggested that alcohol disorders were related to negative emotionality but that the often-observed relation between alcohol disorders and lack of constraint might actually be due to co-occurring drug disorder.

For these reasons, the current study extended previous research by examining the relation of personality and familial alcoholism to bivariate trajectories of alcohol and drug outcomes. We focused on personality characteristics that reflected negative emotionality and behavioral undercontrol because they have been shown to relate to substance use disorders at one point in time. Negative emotionality has been consistently correlated with alcoholism and drug abuse cross-sectionally (Loukas, Krull, Chassin, & Carle, 2000, Martin & Sher, 1994), although there is weaker evidence that it is a prospective predictor (Sher & Trull, 1994). Behavioral undercontrol or lack of constraint (e.g., sensation seeking, impulsivity, low conscientiousness) has been established as both a cross-sectional correlate and a prospective predictor of substance use and substance use disorders (McCormick, Dowd, Quirk, & Hernando Zegarra, 1998; Sher & Trull, 1994). To better establish the directionality of the relation between personality and trajectories, we tested adolescent characteristics (emotionality and impulsivity measured by the Emotionality, Activity, Sociability, and Impulsivity Questionnaire [EASI]; Buss & Plomin, 1984) as prospective predictors. However, to better compare the current findings to previous studies of adults, we also tested young adult personality measured by the NEO—Five-Factor Inventory (NEO–FFI; Costa & McCrae, 1992) as a correlate of trajectory group membership. NEO personality characteristics have been shown to reflect negative emotionality and constraint. Specifically, Church (1994) showed that NEO agreeableness and neuroticism were indicators of negative emotionality, whereas conscientiousness and aspects of openness were indicators of constraint.

In addition to personality characteristics, we also tested familial alcoholism as a predictor. Jackson et al. (2000) found that a family history of alcoholism predicted membership in a comorbid alcohol and tobacco disorder group as well as in a chronic alcohol disorder group, but they did not assess illegal drug use. Moreover, because Jackson et al. (2000) did not assess parent psychopathology other than alcoholism, they did not determine whether parent alcoholism was a specific risk factor for offspring alcohol and tobacco trajectories, or whether any form of parent psychopathology might elevate risk. Thus, the current study extended previous research by directly assessing parent psychopathology and testing whether parent alcoholism had unique effects on alcohol and drug trajectories (above and beyond other parental disorders).

Moreover, some previous studies have suggested that the effects of familial alcoholism on alcohol disorders may be mediated by personality characteristics (Iacono, Carlson, Taylor, Elkins, & McGue, 1999; Sher, Walitzer, Wood, & Brent, 1991). For example, Martin and Sher (1994) found that agreeableness and conscientiousness were related both to parent alcoholism and to alcohol disorders, and Loukas et al. (2000) found that neuroticism and agreeableness partially mediated parent alcoholism effects on young adult alcoholism. Accordingly, we tested whether the effects of familial alcoholism on alcohol and drug trajectories were mediated by these personality characteristics.

Finally, we sought to address several methodological limitations of previous studies. Most trajectory studies have focused on a single dimension of substance use (usually frequency or quantity of consumption). This is a limitation because different etiological factors might predict trajectories of consumption rather than trajectories of disorders (Glantz & Pickens, 1992). Second, few studies have spanned the age range from substance use initiation in adolescence to substance use in the late twenties when developmentally limited forms might be identified (see Bennett et al., 1999; Chassin et al., 2000; and White et al., 2002 for some

exceptions). Third, few studies have examined community samples, instead relying more typically on school-based samples or college samples which may underrepresent high-risk individuals. Finally, few studies have directly diagnosed parent alcoholism and other psychopathology (so that the unique effects of parent alcoholism above and beyond other parent psychopathology can be tested). The current study uses data from a community sample that spans early adolescence to the late twenties with excellent participant retention over time, direct assessment of parent psychopathology, and multiple dimensions of substance use (consumption and diagnosed disorders) as outcome variables.

Method

Participants

Participants were from an ongoing study of parental alcoholism (Chassin, Curran, Hussong, & Colder, 1996; Chassin, Pillow, Curran, Molina, & Barrera, 1993; Chassin, Rogosch, & Barrera, 1991). At Time 1 (T1), there were 454 adolescents ranging in age from 10.5 to 15.5 years ($M = 13.22$), 246 of whom had at least one alcoholic biological parent who was also a custodial parent (COAs) and 208 demographically matched adolescents with no alcoholic biological or custodial parents (control group). There were three annual assessments (T1–Time 3 [T3]) of the adolescents and their parents and two long-term follow-ups (Times 4 and 5 [T4, T5]).

The follow-ups were conducted when the original adolescents were in emerging adulthood (T4: ages 18–23, *Mdn* = 20) and in young adulthood (T5: ages 22–30, *Mdn* = 25). At both follow-ups, sample retention was excellent (T4: $n = 407$, 90% of the total sample, 83.6% of COAs, 93.3% of the control group; T5: $n = 415$, 91% of the total sample, 88% of COAs, 94% of the control group). At both follow-ups, retention was unbiased by gender and ethnicity, but somewhat more COAs than control participants were lost at T4, $\chi^2(1, N = 454) = 5.45$, $p < .05$, and at T5, $\chi^2(1, N = 454) = 4.12$, $p < .05$.

Details of sample recruitment are reported elsewhere (Chassin, Barrera, Bech, & Kossak-Fuller, 1992). COA families were recruited through the use of court records of arrests for driving under the influence of alcohol ($n = 103$), health maintenance organization questionnaires ($n = 22$), and community telephone screening ($n = 120$). One family was referred by a local hospital. COAs had to meet the following criteria: They had to (a) range in age from 10.5 to 15.5 years, (b) be residents of Arizona, (c) be the offspring of Hispanic or Caucasian parents, (d) be English speaking, and (e) have no cognitive limitations that would preclude interview. Direct interview data had to confirm that a biological and custodial parent met *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; *DSM–III*; American Psychiatric Association, 1980) criteria for lifetime alcohol abuse or dependence by means of the Diagnostic Interview Schedule (DIS; L. Robins, Helzer, Croughan, & Ratcliff, 1981) or the Family History—Research Diagnostic Criteria (FH–RDC; Endicott, Andreason, & Spitzer, 1978), and through reports by the other parent (if the alcoholic parent was not interviewed). At T1, 75.6% of biological fathers and 86.6% of biological mothers were interviewed. If there were multiple age-eligible children, the one closest to age 13 was selected.

We recruited demographically matched control participants via telephone interviews. When a COA family was recruited, we used reverse directories to locate families in the same neighborhood. Control participants were screened to match the COAs in ethnicity, family structure, age (within 1 year), and socioeconomic status (which we obtained via the property value code from the reverse directory). Interview data confirmed that neither biological nor custodial parents met *DSM–III* criteria (or FH–RDC criteria) for lifetime alcohol abuse or dependence. At T1, we interviewed 71.2% of biological fathers and 93.8% of biological mothers.

Sample representativeness is discussed in detail elsewhere (Chassin et al., 1992). The sample was unbiased with respect to alcoholism indicators available in archival records (e.g., blood alcohol levels recorded at the time of the arrest). Moreover, the alcoholic sample had rates of other psychopathology similar to those that were reported for a community-dwelling alcoholic sample (Helzer & Pryzbeck, 1988); those who refused participation were most often Hispanic, suggesting some caution in generalization.

At T4, for the first time, full-biological siblings were included if they were in the age range of 18–26 (and all of these siblings were again invited to participate at T5). At T4, we interviewed 327 siblings (78% of whom met eligibility requirements; *Mdn* age = 22). At T5, we interviewed 347 siblings (83% of whom met eligibility requirements; *Mdn* age = 27). The combined sample of original targets and their siblings was $N = 734$ at T4, $N = 759$ at T5, and $N = 817$ with at least one measurement.

Demographic characteristics of the sample at T5 are in Table 1. COAs and control participants did not differ in age, gender, full-time employment, parent status, or full-time student status. However, COAs were more likely than were non-COAs to be Hispanic and less educated.

Procedure

At recruitment, we presented the study as an attempt to understand why some adolescents but not others develop problems, including alcohol and drug problems. Data were collected with computer-assisted interviews at families' homes or on campus. To minimize contamination, family members were interviewed individually, on the same occasion, and by different interviewers when possible. When a family moved out of state, an interviewer from a nearby university administered a shortened version, and the diagnostic interview was done by telephone; the entire interview was done by telephone if no nearby interviewer was available. Interviewers were unaware of the family's group membership. Interviews lasted from 1 to 3 hr and participants were paid up to $65 over the waves. To encourage honesty, we reinforced confidentiality with a Department of Health and Human Services Certificate of Confidentiality. To maximize privacy, participants could enter their responses on the keyboard rather than verbally.

Measures

Parent alcoholism and psychopathology. At T1, lifetime *DSM–III* diagnoses of parent alcoholism (abuse or dependence), affective disorder (major depression or dysthymia), and antisocial personality disorder were assessed with the Diagnostic Interview Schedule (Version 3; DIS; L. Robins et al., 1981). For parents who were not interviewed, lifetime alcoholism diagnoses were established with FH–RDC criteria on the basis of spouses' reports. For the current analyses, diagnoses were dichotomous:

Table 1
Demographic Characteristics (in Percentages) of the Sample at Time 5 Follow-Up

Measure	Total	COA	Control
Female	48.1	47.2	49.1
Male	51.9	52.8	50.9
Hispanic*	25.4	30.2	20.7
Caucasian	74.6	69.8	79.3
Age (*M*)	26.6	26.5	26.7
Ever married*	50.2	45.5	54.8
Had a child	44.4	45.2	43.6
Employed full-time	74.0	75.9	72.1
Full-time students	9.4	9.5	9.3
Some college education*	66.6	61.2	71.8

Note. COA = group with at least one alcoholic biological custodial parent.
* Comparison of COAs and control particpants was significant at $p < .05$.

either present (at least one biological parent met lifetime criteria) or absent (neither biological parent met lifetime criteria).[1]

We also assessed grandparent alcoholism with FH–RDC criteria on the basis of parent report (considering a grandparent to be alcoholic if he or she met criteria by either parent's report; $N = 713$ available reports). We created family history density scores (FHD) following the methods of Stoltenberg, Mudd, Blow, & Hill (1998). Each nonalcoholic parent and grandparent scored zero, each alcoholic parent scored .50, and each alcoholic grandparent scored .25. Summing these scores created a range from 0 (*no alcoholic parents or grandparents*) to 2 (*all alcoholic parents and grandparents*); for the total sample, *M*s = .48 (.79 for COA families and .14 for non-COA families, respectively).

Alcohol consumption. At each wave, participants reported their frequency of past-year consumption of beer/wine and hard liquor (2 items) with responses that ranged from 0 (*never*) to 7 (*every day*). Quantity of consumption (2 items) ranged from 1 (*one*) to 9 (*nine*) or more drinks per occasion. For beer/wine and for hard liquor, we computed Quantity × Frequency products and averaged them to index consumption. Because the modeling techniques are sensitive to non-normality, we used a log transformation to reduce skewness and kurtosis and multiplied the log-transformed variable by 10 to facilitate interpretation.

Drug consumption. At each wave, participants reported their frequency of past-year use of eight different drugs (e.g., marijuana, amphetamines, cocaine, opiates, inhalants) on a scale ranging from 0 (*never*) to 7 (*every day*). For the current analyses, the sum of these items, log-transformed and multiplied by 10, served as the drug consumption measure.

Alcohol and drug dependence diagnoses. At T4 and T5, *DSM–III–R* (3rd ed., rev.; American Psychiatric Association, 1987) alcohol and drug dependence diagnoses were obtained with a computerized version of the DIS. For each participant who met criteria for lifetime diagnosis, we used the DIS onset and recency information (i.e., the age at which the first symptom occurred and the age of most recent symptom) to create past year diagnoses (i.e., lifetime diagnosis with the presence of a symptom in the past year) for four ages. Information for alcohol and drug diagnosis at a fifth adolescent age point was taken from T3 DICA—Parent interview (DICA–P; Herjanic & Reich, 1982).[2] Rates of lifetime alcohol dependence were 23% at T4 and 25.2% at T5. Rates of lifetime drug dependence were 13.1% and 15.5%. Participants who met criteria for drug dependence were most commonly dependent on marijuana (66%), amphetamines (50%), and cocaine (26%; note that individuals could be dependent on more than one substance). Consistent with our oversampling of COAs, our rates of substance disorders were somewhat higher than in national data. For example, National Comorbidity Survey (Kessler, 2002) participants ages 18–25 showed 17.5% lifetime alcohol dependence and 9% lifetime drug dependence.

Adolescent negative emotionality and impulsivity. Adolescents' negative emotionality and impulsivity were measured by T1 parent report on the EASI (Buss & Plomin, 1984). Internal consistency (coefficient alpha) ranged from .73 to .77 across reporters and scales. Because maternal and paternal reports were correlated for emotionality ($r = .45$) and for impulsivity ($r = .48$), we used an aggregated parent report (unless only one parent report was available).

Young adult personality. At T4 and T5, young adults self-reported their personality with the NEO–FFI. Internal consistencies ranged from .72 to .86 across the scales and measurement waves. Because self-reported personality was relatively consistent over the two times (correlations ranged from .53 to .63), scores from the two waves were averaged; if a participant was missing from one of the waves, the other score was used.

Data analytic strategy: Modeling multiple trajectories. Because there was considerable age heterogeneity at each measurement wave, we modeled trajectories as a function of age rather than of measurement occasion. However, the sparseness of the data at some ages necessitated collapsing age into the following five categories to prevent nonconvergence of model estimation: 11–14 (early adolescence), 15–18 (mid- to late adolescence), 19–22 (emerging adulthood), 23–26 (young adulthood), and 27–30 (adulthood).

We conducted separate latent class analyses for use and diagnoses (bivariate analyses of alcohol and drug outcomes). To model substance use, we extracted latent classes with growth mixture modeling (B. O. Muthén & Shedden, 1999; B. O. Muthén & Muthén, 2000) using Mplus (Version 2.13, L. K. Muthén & Muthén, 1998, 2001). This allows estimation of trajectory shapes as random rather than fixed effects, thus modeling individual variation in trajectory shape within each latent class. We adjusted standard errors to account for interdependencies resulting from the clustered (sibling) data by using aggregated analysis under complex sampling as described by B. O. Muthén and Satorra (1995). To ensure that the sibling data did not affect the pattern of findings, we estimated all models with and without sibling data, without any substantive changes. We accounted for missing data by using maximum-likelihood model estimation assuming ignorable missingness at random (Little & Rubin, 1987; L. K. Muthén & Muthén, 1998, pp. 363–364). We included all those with data at two or more different age categories ($N = 660$).[3]

For diagnoses, which are binary variables, we used Mplus to model a latent continuous variable representing a propensity for diagnosis at a given age category and to derive threshold values along the latent continuum to estimate the population proportion of individuals with diagnoses. For these models, we included all participants with the computerized DIS (C–DIS) data from either T4 or T5, supplemented with parent-reported T3 DICA–P

[1] Noninterviewed parents were considered not to meet criteria (except for alcoholism, for which FH–RDC criteria were used for diagnosis on the basis of spousal reports). This allowed us to include single-parent families, but it underestimates the prevalence of parental psychopathologies other than alcoholism, which could produce negatively biased estimates of their effects. Note that such underestimates could not occur when the interviewed parent met diagnostic criteria because in those cases parent psychopathology was coded as present. Thus, these errors could occur only in cases in which the interviewed parent did not meet criteria and the noninterviewed parent would have. Given our high interview rates, this was not frequent. On the basis of data from our two-interviewed-parent families, estimates of potential misclassification errors were only 1% for antisocial personality diagnoses and 3% for depression. Thus, misclassification error should not substantially affect the findings. Although diagnostic interviews were not used for parent drug disorders, parents were asked to report their highest levels of lifetime use of seven types of illegal drugs and their lifetime drug consequences and dependence symptoms using 22 items from the DIS and from Sher's (1987) questionnaire. There were 70 families with a parent who reported more than two lifetime consequences or dependence symptoms (25% of the COA families and 4% of the non-COA families).

[2] Because parent reports might underestimate the extent of adolescents' drug problems, we calculated the percentage of adolescents without a parent-reported diagnosis who self-reported more than two substance-use related problems. These percentages were small (3% for alcohol problems and 1% for drug problems), suggesting minimal error resulting from parental under-reporting.

[3] A small number of participants ($n = 17$) were excluded from the consumption model because their responses were either inconsistent or could not be tied to specific age categories, and 74 lifelong abstainers were classified a priori in an abstainer group. Thus, $N = 586$ in the mixture modeling analyses, with *n*s at each individual age category ranging from 307 to 426. Although these sample sizes reflect substantial missing data at each individual age category, this was largely the result of the fact that siblings were interviewed only twice. To ensure that the missing data did not distort the modeling results, we estimated the models with targets only and obtained virtually identical results.

data ($N = 816$).[4] We created separate dichotomous variables representing the presence or absence of dependence symptoms at each age category for alcohol and drug dependence. Participants with no lifetime dependence diagnosis received scores of "0" across all age categories until their age of last interview, after which their age categories were assigned missing values. Participants with a lifetime dependence diagnosis received "0" scores for each age category before their earliest reported DIS symptom onset. Every age category at which symptoms were reported received a "1," reflecting the presence of active symptoms in the context of a dependence diagnosis. Age categories after the most recent DIS report of symptoms received a score of "0." To avoid making assumptions about the continuity versus remission of symptoms in the absence of information, age categories between the age of onset and recency that did not have symptoms reported were assigned missing values, as were age categories after the age of the most recent interview date.[5]

For each of the models, we first specified a single latent class and then tested a series of models, increasing the number of classes and using the Bayes Information Criterion (BIC; Schwartz, 1978), sample-size adjusted BIC (adjBIC; Sclove, 1987), and the entropy measure (ENT; e.g., Ramaswamy, DeSarbo, Reibstein, & Robinson, 1993) to evaluate model fit. Although there are no well-developed techniques for assessing absolute fit for growth mixture models (Bauer & Curran, 2003; B. O. Muthén, 2003), relatively superior fit is seen in smaller values of BIC and adjBIC. In addition, ENT summarizes the degree to which latent classes are clearly distinguishable by the estimated posterior probabilities of group membership, with higher values (on a scale from 0 to 1) indicating clearer classification. We did not consider groups with fewer than 5% of the sample because they were likely to have poor replicability (cf. Jackson et al., 2000) and because recent work has cautioned against overextraction of latent classes in the presence of nonnormal data (Bauer & Curran, 2003).

After extracting latent classes for each outcome, we used nominal logistic regression to predict group membership from parent alcoholism (and FHD scores in separate models) over and above the effects of gender, parental affective disorder, parental antisocial personality disorder, and personality (over and above the effects of gender). Continuous predictors were standardized to facilitate interpretation by making the odds ratio (OR) metric equivalent to the change in odds for a one standard deviation increase in the predictor. We tested two-way and three-way interactions among parent alcoholism, gender, and personality, and trimmed nonsignificant interactions. Because of the nonindependent (sibling) data, we used multilevel modeling with MIXNO software (Hedeker, 1999). To determine the omnibus effect of each predictor, we used likelihood ratio tests (i.e., χ^2 tests of nested models) to examine the significance of the decrement in model fit when an individual variable is removed from the model. We then tested each pairwise comparison among trajectory groups. We tested each personality characteristic for unique effects first separately and then together.

Finally, we assessed whether personality mediated the relations between parent alcoholism and latent class memberships and tested only those personality characteristics that were both unique predictors of class membership in the absence of parent alcoholism and significantly predicted by parent alcoholism. For these variables, we tested for mediation with the product of coefficients method (e.g., MacKinnon & Dwyer, 1993) to determine the significance of the indirect effect of parental alcoholism on group membership through personality. We also estimated all of these effects by using FHD scores instead of parent alcoholism. Because measures of effect size are not well established for mediational models with categorical outcomes, we report only the proportion of the direct effect that was accounted for by the indirect effect (MacKinnon, Warsi, & Dwyer, 1995). This is an estimate of the percentage reduction in the direct effect of familial alcoholism resulting from the addition of mediators in the model. However, because this statistic is a function of the coefficients in the model and not their variance, it provides only a relatively crude description of the size of the mediated effect.

Results

Alcohol and Drug Consumption

We defined abstainers (i.e., those who did not report any use of alcohol or drugs at any point) a priori and they were not included in the mixture analysis ($n = 74$; 11.2% of the sample). For the remaining participants ($n = 586$), we used the methods described above to estimate a series of latent class growth mixture models. For each group, we specified a quadratic growth function to allow for curvilinear trends across the ages. A quadratic growth function contains an intercept factor (which we defined as the average consumption during the third age period [19–22]), a linear factor, and a quadratic factor. The mean parameters of each of these factors varied across groups, whereas we constrained residual variances and covariances among the growth factors to be equal across groups.[6] We tested one-group through four-group solutions. With the one-group solution, there was significant intraindividual variance in both alcohol and drug trajectories (ps $< .05$ for the variance of the intercept and linear factors and ps $< .10$ for the quadratic factors), thus justifying the extraction of additional groups to account for this heterogeneity. The four-group solution had a group that represented only 4.2% of the sample and did not produce substantial improvement in fit statistics. The three-group solution was an improvement over the two-group solution (BIC = 23,320 vs. 22,969; adjBIC = 23,193 vs. 22,813; ENT = .66 vs. .77 for the two-group and three-group solutions, respectively). Moreover, for the three-group solution, the variance terms for the latent growth factors within each group were not significantly different from zero, implying that the heterogeneity observed in the one-group solution was completely described by the three-group solution.[7] Also in support of the three-group solution, only 10.9% of cases might be considered "difficult to classify" in the sense that they had a probability of being assigned to a different group that was above chance. Removing these cases did not substantially alter

[4] Of those 816 participants, 498 did not develop any lifetime substance use disorder over the course of the study, and were assigned a priori to a nondiagnosed group. An additional 38 participants with current diagnoses (i.e., diagnoses with active symptoms in the past year) at both waves in which the C–DIS was administered were assigned a priori to a persistent group, leaving 279 participants in the mixture modeling (*ns* from 226 to 279 at the first four age categories with $n = 109$ at age 27–30; missing data from 0% to 19% at the first four age categories with 60.9% missing data at age 27–30).

[5] We also estimated the diagnosis models with diagnosis defined as *abuse* or *dependence* rather than dependence only without substantive changes. Because dependence is the clearer diagnostic outcome in *DSM–III–R,* the dependence models are presented here.

[6] Freeing variances across classes resulted in model nonconvergence. By not allowing growth factor variances and covariances to differ across classes, the classes were identified on the basis of the means of the growth factors alone. This approach has been adopted by other substance use trajectory studies (e.g., Colder et al., 2002; Sher & Jackson, 2004; Tucker et al., 2003).

[7] As described in Footnote 6, the mixture model for the full sample was estimated with the variances of the growth factors constrained to be equal across classes. To derive estimates of within-class growth factor variances, we estimated separate growth models for each class, constraining the mean growth parameters to equal those found with the three-class mixture model for the full sample.

the mean observed trajectory for each group. The average probability of group membership was .89, .96, and .85 for the three groups (see Figure 1 for the model-implied and average observed trajectories).

As noted earlier, we defined abstainers a priori and they were 11.2% of the sample. One group, light drinking with rare drug use, consisted of 159 participants (24.1% of the sample). It was characterized by modest increases in drinking over adolescence to an average of approximately one drink per occasion, three to five times per year (corresponding to a value of 13 on our alcohol scale; see Figure 1), with a slight decline after age 22. This group showed almost no use of illegal drugs, although no member of this group

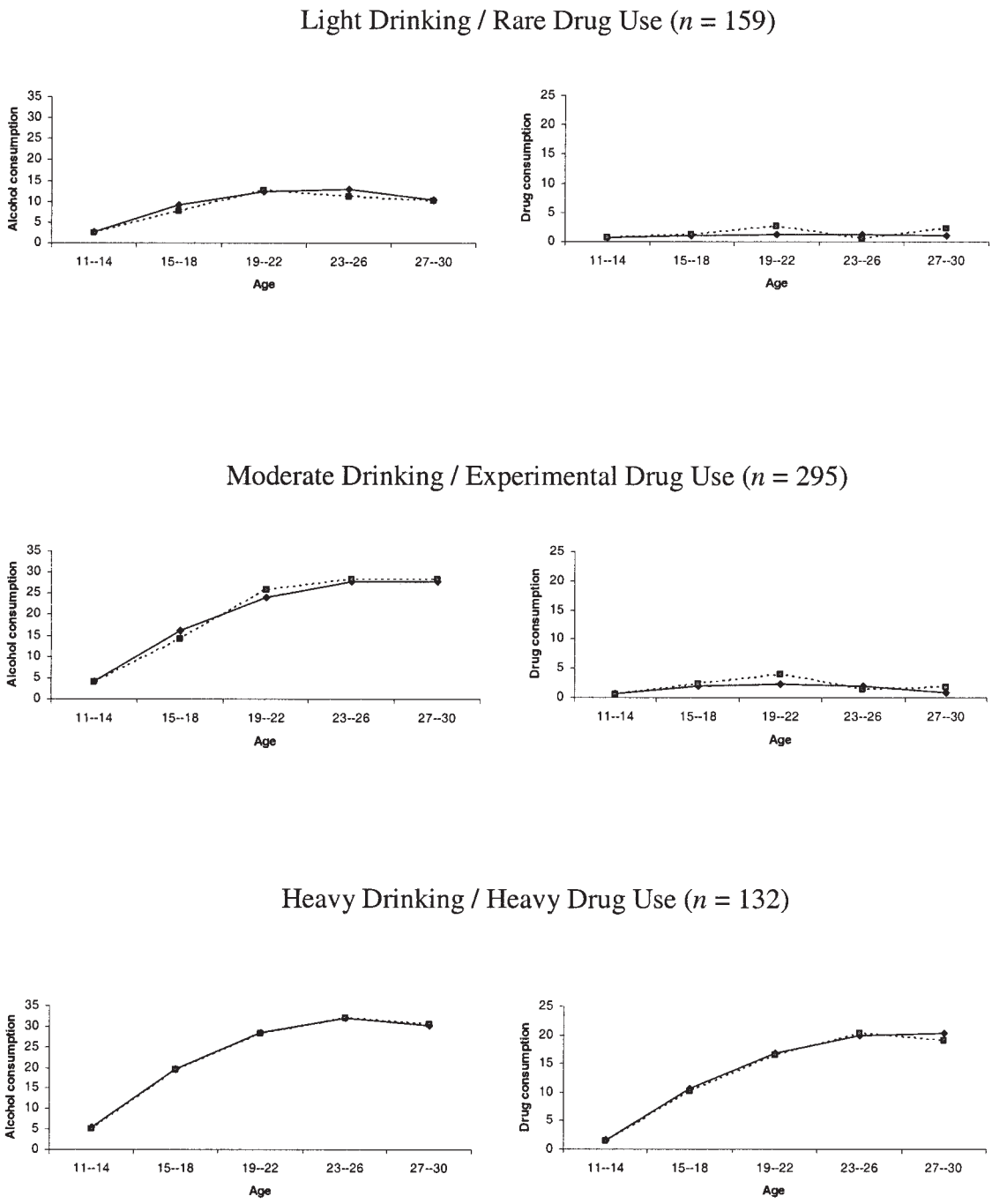

Figure 1. Alcohol and drug consumption trajectories by latent class. Because its trajectories overlap completely with the abscissa, the abstaining class ($n = 74$) is not pictured. Graphs on the left side represent mean alcohol consumption by age; graphs on the right side represent mean drug consumption by age. Solid diamonds connected by solid lines represent model-implied (i.e., predicted) trajectories; solid squares connected by dashed lines represent mean observed trajectories.

completely abstained from drug use at all five age periods. The largest group, moderate drinking with experimental drug use, consisted of 295 participants (44.6% of the sample). Their drinking showed steep initiation in the high school years that stabilized at age 23. On average, their peak alcohol consumption can be described as three drinks per occasion, one to three times per month (corresponding to a score of 28 on our scale). By contrast, they had very low rates of drug use that increased slightly in adolescence; on average they used one illegal drug approximately once or twice a year (corresponding to a score of 4 on our scale) and declining after age 22. A final group, heavy drinking and drug use consisted of 132 participants (20% of the sample) and was characterized by escalating trajectories of both alcohol and illegal drug use. At their peak level of alcohol use (ages 23–26), they averaged approximately four drinks per occasion, one to two times per week (corresponding to a score of 32 on our scale). Their peak drug use (after age 22) was at "almost daily" use (corresponding to a score of 20 on our scale).[8] For all three nonabstaining groups, the most commonly used drug was marijuana, followed by amphetamines, such that the nonabstaining groups were not distinguishable in their choice of a particular illegal drug.

Prediction of Consumption Groups

As described earlier, we first predicted trajectory group membership from gender, parent alcoholism, parent antisocial personality, and parent affective disorder. There was a significant effect of gender, $\chi^2(3, N = 660) = 32.00$, $p < .01$, such that there was a greater proportion of males in the heavy drinking/heavy drug use group (65.9%) than in all the other groups (abstainers = 48.6%; light drinking/rare drug use = 35.2%; all ps $< .05$) except the moderate drinking/experimental drug use group (56.9%). Also, there was a greater proportion of males in the moderate drinking/experimental drug use group than in the light drinking/rare drug use group ($p < .01$).

Above and beyond gender and other parent psychopathology, parental alcoholism was a significant predictor of group membership, $\chi^2(3, N = 660) = 33.54$, $p < .01$, such that there was a greater proportion of COAs in each of the drinking and drug use groups than was found in the abstaining group (20.3%; ORs ranged from 3.40 to 8.97; all ps $< .01$). Moreover, there was a greater proportion of COAs in the heavy drinking/heavy drug use group (68.9%) than in either the moderate drinking/experimental drug use group (53.6%; OR = 2.24) or the light drinking/rare drug use group (45.9%; OR = 3.05; both ps $< .01$). There were no significant effects of other parental disorders and no interaction between parent alcoholism and gender.

Estimating these models with FHD scores instead of parent alcoholism produced identical results. FHD had a significant unique effect, $\chi^2(3, N = 590) = 91.54$, $p < .01$, such that higher FHD scores predicted increased odds of membership in each of the nonabstaining groups relative to the abstaining group (ORs ranged from 5.41 to 9.56; all ps $< .01$) as well as an increased chance of membership in the heavy drinking/heavy drug use group relative to both the moderate drinking/experimental drug use and the light drinking/rare drug use groups (all ORs = 1.46; ps $< .04$, 1.86, and .01, respectively). The mean FHD scores were .20 for abstainers, .40 for the light drinking/rare drug use group, .40 for the moderate drinking/experimental drug use group, and .60 for the heavy drinking/heavy drug use group. FHD did not interact with gender.[9]

Adolescent impulsivity and negative emotionality. Next, we tested whether adolescent impulsivity and negative emotionality prospectively predicted trajectory group membership (above and beyond the effects of gender). Because these predictors were measured at Time 1, this analysis only applied to the original targets with consumption group data ($N = 437$; see Table 2). There were significant effects of impulsivity, $\chi^2(3, N = 437) = 15.90$, $p < .01$, such that higher impulsivity increased the odds of membership in the heavy drinking/heavy drug use group compared with all of the other groups (ORs ranged from 1.30 to 1.82; all ps $< .05$). These effects were maintained after we added emotionality to the model, and there were no interactions with gender. There were no significant effects of emotionality, with or without impulsivity, in the model.

We next tested whether impulsivity mediated the relation between parent alcoholism and consumption group membership. Controlling for gender, parent alcoholism significantly predicted impulsivity, $t(435) = 3.87$, $p < .01$, such that COAs were more impulsive than were non-COAs ($M = 3.01$, $SD = .59$ vs. $M = 2.81$, $SD = .53$). Moreover, impulsivity significantly mediated the relation between parent alcoholism and the odds of membership in the heavy drinking/heavy drug use group compared with both the light drinking/rare drug group ($z = 2.29$, $p < .05$, accounting for 9.09% of the parent alcoholism effect) and the abstainers ($z = 1.96$, $p < .05$, accounting for 16.44% of the parent alcoholism

[8] Because past research has often examined trajectories of "five drinks per occasion," we also modeled trajectories of binge drinking using that single item. Results yielded virtually identical trajectory groups. The only exception was that the light drinking /rare drug use group showed a steeper decline in their drinking after ages 19–22. Because our quantity-frequency measure has better psychometric properties and growth mixture modeling assumes a continuous variable, we present here the results for the quantity-frequency composite.

To more formally compare the extent of consumption among the three nonabstaining classes, we used a series of multilevel analyses of variance to compare their alcohol and drug use at each age category. The three groups differed in both alcohol and drug use at all ages (Fs ranged from 7.75 to 1168.30; all ps $< .01$). In particular, although the drinking of the heavy and moderate groups appears visually similar in Figure 1, the heavy drinking/heavy drug use group drank significantly more than did the moderate drinking/experimental drug use group at all but the first age period (i.e., from age 15 on; all ps $< .05$). Similarly, although the drug use of the light drinking/rare drug use and moderate drinking/experimental drug use groups appears visually similar in Figure 1, the moderate drinking/experimental drug use group reported significantly more drug use at ages 23–26 ($p < .05$).

[9] To ensure that these effects were not the result of co-occurring parental drug problems, we also estimated these models with the addition of a proxy variable for parental lifetime drug disorder (a dichotomous variable comparing parents who did and did not report more than two lifetime consequences or dependence symptoms). Parental drug problems did not significantly predict class membership above and beyond gender and either parental alcoholism or FHD, $\chi^2(3, N = 660) = 0.47$, $p = .93$ and $\chi^2(3, N = 660) = 1.04$, $p = .79$, respectively. The proportions of each class with at least one parent reporting more than two lifetime drug problems were as follows: abstainers = 5.4%, light drinking/rare drug use = 11.9%, moderate drinking/experimental drug = 13.6%, and heavy drinking/heavy drug = 15.9%.

Table 2
Descriptive Statistics for Personality Variables By Consumption Class

	Parent report EASI				Self-Report NEO–FFI							
	Impulsivity		Emotionality		NE		OP		AG		CO	
Class and informant sample size	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Abstainers	2.77_a	0.48	2.43_a	0.74	2.57_a	0.60	3.08_a	0.50	3.88_a	0.49	3.80_a	0.47
Parents ($n = 40$)												
Participants ($n = 70$)												
Light drinking/rare drug use	2.77_a	0.59	2.46_a	0.61	2.73_a	0.61	3.18_{ab}	0.50	3.69_b	0.49	3.78_a	0.55
Parents ($n = 114$)												
Participants ($n = 152$)												
Moderate drinking/experimental drug use	2.94_a	0.55	2.43_a	0.64	2.70_a	0.61	3.30_b	0.46	3.54_b	0.46	3.71_a	0.56
Parents ($n = 191$)												
Participants ($n = 283$)												
Heavy drinking/heavy drug use	3.10_b	0.58	2.62_a	0.60	2.90_b	0.68	3.50_c	0.55	3.43_c	0.46	3.48_b	0.55
Parents ($n = 92$)												
Participants ($n = 125$)												

Note. Within each column, mean comparisons sharing the same subscript do not predict significant differences in likelihood of class membership. EASI = Emotionality, Activity, Sociability, and Impulsivity Questionnaire; NEO–FFI = NEO–Five-Factor Inventory; NE = Neuroticism; OP = Openness; AG = Agreeableness; CO = Conscientiousness.

effect). Impulsivity was a significant but partial mediator, and parent alcoholism still had a significant ($p < .01$) effect after we added impulsivity.

We found a similar pattern when we tested whether impulsivity mediated the relations between FHD scores and consumption group membership. Controlling for gender, FHD significantly predicted impulsivity, $t(379) = 4.43$, $p < .01$, such that higher FHD was associated with greater impulsivity ($r = .22$). Impulsivity significantly mediated the relation between FHD and the odds of membership in the heavy drinking/heavy drug use group compared with the light drinking/rare drug use group ($z = 2.28$, $p < .05$, accounting for 18.70% of the FHD effect), and marginally mediated the comparison with the abstaining group ($z = 1.87$, $p < .06$, accounting for 8.84% of the FHD effect). Again, impulsivity was only a partial mediator, and its inclusion in the model did not eliminate the significant effect of FHD on group membership ($p < .01$).

Adult NEO–FFI predictors. Next, we tested whether young adult personality distinguished among the latent classes above and beyond gender. There were no significant interactions with gender (see Table 2 for means across the trajectory groups).

There was a significant effect of neuroticism, $\chi^2(3, N = 630) = 15.00$, $p < .01$, such that higher neuroticism predicted significantly greater odds of membership in the heavy drinking/heavy drug use group compared with all others (ORs ranged from 1.53 to 1.86; all ps $< .05$). However, these effects were not maintained when the other NEO–FFI variables were included in the model.

There was also a significant effect of openness, $\chi^2(3, N = 630) = 35.36$, $p < .01$. Higher openness led to increased odds of belonging to the heavy drinking/heavy drug use group compared with all others (ORs ranged from 1.62 to 2.82; all ps $< .01$; see Table 2) and to the moderate drinking/experimental drug use group rather than to the abstainer group (OR = 2.38, $p < .01$). These effects were maintained when the other NEO–FFI variables were included in the model.

There was also a significant effect of agreeableness, $\chi^2(3, N = 630) = 27.78$, $p < .01$. Those who were higher in agreeableness were more likely to be in the abstainer group than in any other (ORs ranged from 2.14 to 3.38; all ps $< .01$) and less likely to be in the heavy drinking/heavy drug use group than in any other (ORs ranged from 1.31 to 3.38; all ps $< .01$). When the other personality variables were included in the model, these effects were maintained except for the difference between the heavy drinking/heavy drug use and moderate drinking/experimental drug use groups.

Finally, there was a significant effect of conscientiousness, $\chi^2(3, N = 630) = 18.60$, $p < .01$. Those who were lowest in conscientiousness were most likely to be in the heavy drinking/heavy drug use group than in any other (ORs ranged from 1.58 to 1.74; all ps $< .01$). However, these differences were not maintained when we added the other personality variables to the model.

Next, we tested whether openness and agreeableness mediated the relations between parent alcoholism and group membership. (Neuroticism and conscientiousness were not considered because they were not unique predictors of group membership.) First, we tested the relations between parent alcoholism and personality using a multilevel regression model in SAS PROC MIXED (to account for nested sibling data). Agreeableness (but not openness) was related to parent alcoholism, $t(410) = 2.86$, $p < .01$, such that COAs had lower agreeableness ($M = 3.53$, $SD = .49$) than did non-COAs ($M = 3.65$, $SD = .48$).

Agreeableness significantly mediated the relation between parent alcoholism and consumption group. Parent alcoholism predicted lower agreeableness, which then increased the odds of belonging to all of the nonabstaining groups relative to the abstainers (z scores ranged from 2.10 to 2.56; all ps $< .05$) and to the heavy drinking/heavy drug use group relative to the light drinking/rare drug use group ($z = 2.04$, $p < .05$). Agreeableness was a partial mediator, accounting for between 7.41% and 11.02% of the COA effect, which remained significant at $p < .01$.

Identical results were produced for FHD scores. FHD was related to agreeableness, $t(367) = 4.19$, $p < .01$ (but not openness), such that higher FHD predicted lower agreeableness ($r = -.18$). There was significant mediation such that FHD predicted lower agreeableness, which then increased the odds of belonging

to each of the nonabstaining groups relative to the abstaining group (z scores ranged from 2.00 to 2.64; all ps $< .05$) and to the heavy drinking/heavy drug use group relative to the light drinking/rare drug use group ($z = 2.09$, $p < .05$). Again, agreeableness was a partial mediator, accounting for between 36.80% and 44.50% of the FHD effect, which remained significant at $p < .01$.

Trajectories of Dependence Diagnoses

We defined an a priori "no diagnosis" group that had no alcohol or drug dependence diagnosis during the study ($n = 499$; 61.1% of the sample), and a "persistent dependence" group ($n = 38$; 4.7% of the sample) who had active (past year) diagnoses on both C–DIS measurements ($n = 35$ on alcohol and $n = 3$ on drugs).[10] For the remaining 279 participants, we estimated a series of latent group mixture models, modeling the same age categories described earlier. We tested one-group through four-group solutions, with the three-group solution producing the best model fit where no groups represented less than 5% of the sample. Model fits for the two-group versus the three-group solution were BIC = 2,393 vs. 2,361; adjBIC = 2,327 vs. 2,259; ENT = .71 vs. .86, respectively. In support of the adequacy of model fit, only 2.5% of cases might be considered difficult to classify in the sense that they have an above chance probability of being assigned to another group. The average predicted probabilities of group membership were high (.93, .95, and .94, respectively; see Figure 2 for the model-implied probabilities).

As noted earlier, the majority of the sample ($n = 499$; 61.1%) was nondiagnosed and a small persistent subgroup had persistent dependence (most on alcohol, $n = 38$; 4.7%). The mixture modeling produced a large group ($n = 151$; 18.6% of the sample) who could be considered "alcohol dependent only." This group showed probabilities of alcohol dependence that peaked at .74 at ages 19–22 and then declined in the context of virtually no drug dependence diagnoses. Another group could be thought of as "drug dependent only" ($n = 78$ participants; 9.7% of the sample). It was characterized by low predicted probabilities of alcohol dependence at all ages, but with drug dependence probabilities that peaked at .57 at ages 15–18 and 19–22. A final group could be characterized as "comorbid" ($n = 50$; 6.1% of the sample). They showed high predicted probabilities of both alcohol and drug dependence that peaked at ages 19–22, when 100% of the group showed both alcohol and drug dependence symptoms.

Next, we examined the relation between membership in our consumption and diagnoses groups (excluding abstainers, who by definition were not diagnosed; see Table 3). As expected, the consumption and diagnosis groups were related, $\chi^2(8, N = 582) = 139.07$, $p < .01$. Those in the heavy drinking/heavy drug use group were most likely to be diagnosed and were equally likely to be in the alcohol, drug, comorbid, or persistent groups. Those in the moderate drinking/experimental drug use group were less likely to be diagnosed, but those who were diagnosed were most likely to be in the alcohol dependence only group. The light drinking/rare drug use was the least likely to be diagnosed (significantly less likely than the moderate drinking/experimental drug use group, $\chi^2(4, N = 450) = 25.16$, $p < .01$).

Prediction of Diagnosis Group Membership

We predicted group membership from gender, parent alcoholism, parent affective disorder, and parent antisocial personality. There were significant effects of gender, $\chi^2(4, N = 816) = 45.72$, $p < .01$, such that there was a significantly smaller proportion of males in the nondiagnosed group (44.5%) than in any other (alcohol only = 72.2%, drug only = 52.6%, comorbid = 66.0%, and persistent = 68.4%; all ps $< .05$) and a larger proportion of males in the alcohol only group than in the drug only group ($p < .01$). Above and beyond gender and other parent psychopathology, parent alcoholism showed a significant unique effect, $\chi^2(4, N = 816) = 40.21$, $p < .01$, such that there was a significantly greater proportion of COAs in each of the diagnosed groups than in the nondiagnosed group (nondiagnosed = 39.7%, alcohol only = 66.2%, drug only = 60.3%, comorbid = 76.0%, persistent = 65.8%; ORs ranged from 3.00 to 5.93; all ps $< .05$). However, parent alcoholism did not distinguish among the diagnosed groups. There were no significant effects of other parent disorders and no significant interactions between parent alcoholism and gender. Estimating these models with FHD scores instead of parent alcoholism showed a significant unique effect of FHD, $\chi^2(4, N = 733) = 48.98$, $p < .01$. Higher FHD predicted increased odds of membership in each of the diagnosed groups compared with the nondiagnosed (ORs ranged from 1.53 to 2.62; all ps $< .05$) and with increased odds of membership in the alcohol only or comorbid groups than in the drug only group (ORs 1.51 and 1.87, respectively; both ps $< .05$). The mean FHD scores were nondiagnosed = .37, drug only = .49, persistent = .50, alcohol only = .63, and comorbid = .70. There were no unique effects of other parent psychopathology, nor did FHD interact with gender.[11]

Adolescent impulsivity and negative emotionality. We next tested whether adolescent impulsivity and negative emotionality prospectively predicted diagnosis group membership over and above the effects of gender. Because these variables were measured at T1, these analyses were restricted to the original target adolescents ($n = 440$ with complete data; see Table 4). There was a significant effect of impulsivity, $\chi^2(4, N = 440) = 18.37$, $p < .01$, such that impulsivity predicted increased odds of membership in the drug only and comorbid groups relative to the nondiagnosed (ORs = 1.62 and 2.01, respectively; both ps $< .01$) and increased odds of membership in the comorbid compared with the alcohol only group (OR = 1.55, p $< .05$). These effects were maintained

[10] Because the persistent group represented such a small proportion of the total sample, it would be unlikely to be identifiable in the mixture analysis. However, on the basis of past theory and data that distinguish between more normative "developmentally limited" and persistent groups (Jackson et al., 2000; Zucker, Ellis, & Fitzgerald, 1994), it represents a theoretically important group. Thus, we defined it a priori. Larger samples that follow participants to later ages would be expected to produce clearer distinctions between developmentally limited and persistent diagnosed groups.

[11] We also estimated these models after adding parental drug problems as a predictor. Parental drug problems did not significantly predict class membership above and beyond gender and either parental alcoholism or FHD, $\chi^2(4, N = 816) = 4.03$, $p = .40$ and $\chi^2(4, N = 816) = 1.02$, $p = .91$. The proportions of each class with at least one parent reporting more than two lifetime drug problems were nondiagnosed = 8.62%, alcohol only = 16.6%, drug only = 14.1%, comorbid = 22.0%, and persistent = 10.53%.

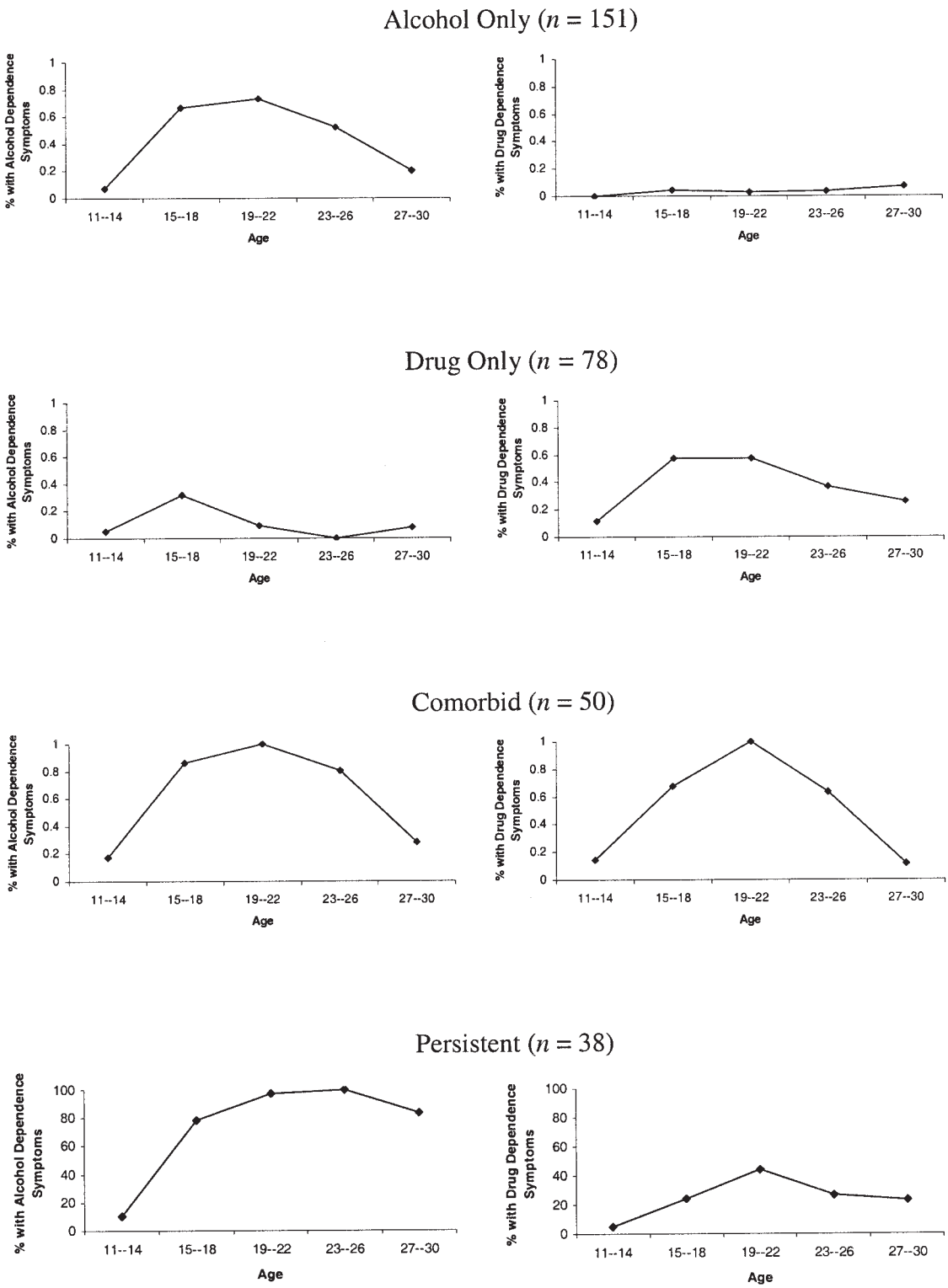

Figure 2. Alcohol and drug dependence diagnosis trajectories by latent class. Because its trajectories overlap completely with the abscissa, the nondiagnosed class ($n = 499$) is not pictured. Graphs on the left side represent proportion with alcohol dependence symptoms by age; graphs on right side represent proportion with drug dependence symptoms by age.

Table 3
Cross-Classification of Consumption Classes Versus Diagnosis Classes

Consumption class	Diagnosis class				
	Nondiagnosed	Alcohol only	Drug only	Comorbid	Persistent
Abstainers	71 (100)	0	0	0	0
Light drinking/Rare drug use	122 (77.2)	15 (9.5)	12 (7.6)	8 (5.1)	1 (0.6)
Moderate drinking/Experimental drug use	177 (60.6)	76 (26.0)	15 (5.1)	10 (3.4)	14 (4.8)
Heavy drinking/Heavy drug use	30 (22.7)	28 (21.2)	28 (21.2)	24 (18.2)	22 (16.7)

Note. $N = 653$ with non-missing values on both consumption class membership and diagnosis class membership. Values in parentheses represent percentage of members from a consumption class falling in a given diagnosis class.

after we included emotionality in the model. There were no interactions with gender.

There was a significant effect of emotionality, $\chi^2(4, N = 440) = 16.13$, $p < .01$, such that higher emotionality increased the odds of membership in the drug only and comorbid groups compared with the nondiagnosed group (ORs =1.46 and 1.96, respectively; both ps $< .01$). However, these effects were not maintained when impulsivity was added to the model.

We next tested whether impulsivity mediated the relation between parent alcoholism and diagnosis group. (Emotionality was not considered because it was not a unique predictor.) As noted above for the consumption models, parent alcoholism significantly predicted higher impulsivity. Impulsivity was a significant mediator. Parent alcoholism predicted greater impulsivity, which in turn increased the odds of membership in the drug only and comorbid groups relative to the nondiagnosed group ($z = 2.03$ and 2.30, respectively; both ps $< .01$). Impulsivity was a partial mediator, accounting for 13.48% and 13.76% of the parent alcoholism effect, which remained significant at $p < .01$ after we added impulsivity to the model.

A similar pattern was found for the FHD effect. As noted in the earlier consumption models, FHD significantly predicted higher impulsivity. Impulsivity was a significant mediator. FHD predicted greater impulsivity, which in turn increased the odds of membership in the drug only group relative to the nondiagnosed ($z = 1.97$, $p < .05$). This reflected partial mediation, accounting for 18.76% of the FHD effect, which stayed significant at $p < .01$.

Adult NEO–FFI predictors. Next, we assessed whether adult personality predicted group membership above and beyond the effects of gender (see Table 4). There were no interactions between gender and NEO–FFI variables.

There was a significant effect of neuroticism, $\chi^2(4, N = 778) = 37.23$, $p < .01$. Higher neuroticism increased the odds of membership in each of the diagnosed groups compared with the nondiagnosed (ORs ranged from 1.36 to 2.63; all ps $< .01$) and in the comorbid and drug only groups compared with the alcohol only

Table 4
Descriptive Statistics for Personality Variables By Diagnosis Class

	Parent report EASI				Self-Report NEO–FFI							
	Impulsivity		Emotionality		NE		OP		AG		CO	
Class and informant sample size	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Nondiagnosed Parent ($n = 267$) Participant ($n = 479$)	2.82_{a}	0.52	2.40_{a}	0.60	2.63_{a}	0.67	3.24_{a}	0.55	3.71_{a}	0.54	3.79_{a}	0.58
Alcohol only Parent ($n = 77$) Participant ($n = 141$)	3.00_{ab}	0.59	2.54_{ab}	0.71	2.72_{b}	0.56	3.32_{ab}	0.53	3.48_{b}	0.44	3.66_{b}	0.55
Drug only Parent ($n = 45$) Participant ($n = 72$)	3.09_{bc}	0.64	2.63_{b}	0.60	3.01_{c}	0.62	3.35_{ab}	0.50	3.47_{bc}	0.47	3.49_{c}	0.50
Comorbid Parent ($n = 28$) Participant ($n = 48$)	3.24_{c}	0.68	2.82_{b}	0.64	3.11_{c}	0.76	3.42_{b}	0.57	3.25_{c}	0.42	3.52_{bc}	0.64
Persistent Parent ($n = 23$) Participant ($n = 38$)	2.99_{abc}	0.63	2.48_{ab}	0.65	2.85_{bc}	0.63	3.53_{b}	0.49	3.43_{bc}	0.40	3.48_{bc}	0.53

Note. Within each column, mean comparisons sharing the same subscript do not predict significant differences in likelihood of class membership. EASI = Emotionality, Activity, Sociability, and Impulsivity Questionnaire; NEO–FFI = NEO–Five-Factor Inventory; NE = Neuroticism; OP = Openness; AG = Agreeableness; CO = Conscientiousness.

group (ORs = 1.77 and 1.45, respectively; both ps $< .05$). When the other NEO–FFI variables were included, the nondiagnosed differed only from the drug only and comorbid groups, and the alcohol only and drug only groups did not differ.

There was a significant effect of openness, $\chi^2(4, N = 778) = 11.60$, $p < .05$. Relative to the nondiagnosed group, higher openness increased the odds of membership in the comorbid and persistent groups (ORs = 1.44 and 1.61, respectively; both ps $< .05$) but did not distinguish among the diagnosed groups. When the other NEO–FFI variables were included, openness also significantly distinguished the alcohol only and drug only groups from the nondiagnosed (ORs = 1.41 and 1.49, respectively; both ps $< .05$).

There was also a significant effect of agreeableness, $\chi^2(4, N = 778) = 47.55$, $p < .01$. Those with lower agreeableness were more likely to be in any of the diagnosed groups rather than in the nondiagnosed group (ORs ranged from 1.75 to 3.00; all ps $< .05$). Among the diagnosed groups, those lowest in agreeableness were more likely to be in the comorbid than in the alcohol only group (OR = 1.87, $p < .01$). These effects were maintained when we added the other NEO–FFI variables, and agreeableness also distinguished the comorbid from the drug only group (OR = 1.84, $p < .01$).

Finally, there was a significant effect of conscientiousness, $\chi^2(4, N = 778) = 29.98$, $p < .01$. Lower conscientiousness increased the odds of membership in each diagnosed group relative to the nondiagnosed group (ORs ranged from 1.38 to 1.98: all ps $< .05$) and in the drug only compared with the alcohol only group (OR = 1.42, $p < .05$). However, these differences were not maintained when we added the other NEO–FFI variables to the model.

Next, we examined whether personality mediated the relation between parent alcoholism and diagnosis group membership. As described above, for the consumption models, parental alcoholism predicted lower agreeableness. Parent alcoholism was also related to higher neuroticism, $t(423) = 2.38$, $p < .05$, but not to openness and conscientiousness. Thus, openness and conscientiousness were not considered further.

Agreeableness significantly mediated the relations between parent alcoholism and group membership. COAs had lower agreeableness, which then predicted increased odds of membership in each diagnosed group (except the persistent group) relative to the nondiagnosed group (z scores ranged from 1.97 to 2.47; all ps $< .05$). Agreeableness was a partial mediator, accounting for between 7.41% and 10.19% of the parent alcoholism effect, which remained significant at $p < .01$.

Neuroticism was also a significant but partial mediator. Parental alcoholism predicted increased neuroticism, which then increased the odds of belonging to the comorbid group relative to the nondiagnosed group ($z = 2.00$, $p < .05$, accounting for 6.26% of the parent alcoholism effect, which remained significant at $p < .01$).

Similar patterns were found for FHD scores. Higher FHD predicted higher neuroticism $t(379) = 3.10$, $p < .01$, $r = .13$. As described earlier, for the consumption models, FHD was significantly related to agreeableness but not to openness or conscientiousness. Thus, we did not consider openness and conscientiousness further.

Agreeableness was a significant but partial mediator. FHD predicted lower agreeableness, which, in turn, increased the odds of membership in any of the diagnosed groups relative to the nondiagnosed group (z scores ranged from 2.07 to 3.15; all ps $< .05$, accounting for between 34.48% and 54.75% of the FHD effect, which remained significant at $p < .01$).

Neuroticism was a significant but partial mediator. FHD predicted increased neuroticism, which, in turn, increased the odds of belonging to the drug only and comorbid groups relative to the nondiagnosed group (zs = 2.01 and 2.33, respectively; both ps $< .05$, accounting for between 25.88% and 38.62% of the FHD effect, which remained significant at $p < .01$).

Discussion

The current study sought to describe and predict trajectories of alcohol and illegal drug use and dependence from early adolescence to adulthood. Consistent with national data, lifelong abstinence was rare, and only 11.3% of our participants were lifelong abstainers from alcohol and drugs. This is similar to findings from the Monitoring the Future Study (Johnston, O'Malley, & Bachman, 2002) for individuals of similar ages and geographic residence (i.e., ages 19–32 in the western region of the United States), which showed that 11.5% of individuals were lifelong abstainers from alcohol. Rather than abstinence, the most common trajectory involved moderate alcohol use coupled with low levels of drug use, and it was also relatively common for individuals to drink lightly and infrequently with only very rare drug use. As expected developmentally, the use of alcohol and drugs increased during adolescence and peaked in emerging adulthood (Arnett, 2000; Chen & Kandel, 1995). Although our consumption trajectories did not show the dramatic downturns at later ages that might be expected from individuals "maturing out" of substance use in their mid-twenties (Bachman et al., 1997), the extent of use did decline in adulthood (particularly among the light drinking/rare drug use group), and trajectory groups of developmentally limited dependence on alcohol and drugs were obtained. In terms of dependence, the mixture modeling identified groups involving alcohol only (the most common), illegal drugs only, or comorbid disorder, with most participants (61%) not developing dependence over the course of the study.

In terms of consumption, the group of most clinical significance was the heavy drinking/heavy drug use group, which showed escalating trajectories of heavy use of alcohol and drugs from adolescence to emerging adulthood. The levels of use in this group went beyond those that are developmentally normative, either as defined by their frequency in the sample (i.e., this group contained 20% of the participants) or by national epidemiological data (e.g., at their peak, this group averaged daily use of an illegal drug). Not surprisingly, these participants were also highly likely to develop a substance use disorder, with almost 80% of them diagnosed as dependent on alcohol or illegal drugs or both. Moreover, this group had the highest risk for disorders that went beyond alcohol dependence only (i.e., they had higher rates of drug, comorbid, or persistent dependence). Members of this group were most likely to be children of alcoholics and had the densest family histories of alcoholism, supporting previous research that links family history risk to trajectories of heavy use and clinical disorders (Chassin et al., 2002; Jackson et al., 2000). They also had the highest levels of impulsivity, lowest agreeableness, and most openness.

However, although the heavy drinking/heavy drug use group had the highest rates of consumption and the highest levels of family history and personality risk, they were not the only group at risk for substance use disorder. The moderate drinking/experimental drug use group also showed elevated risk for dependence, although they were most likely to develop alcohol dependence alone (rather than comorbid or persistent dependence). From their frequency in the sample (45%) and their levels of drug use (at their peak average use of only one drug once or twice a year), this group might be judged to be relatively developmentally normative in their adolescent consumption. This interpretation is also supported by the fact that they significantly differed from the heavy drinking/heavy drug use group in all temperament and personality indicators (except adolescent emotionality) and also had less dense family histories of alcoholism. Moreover, the moderate drinking/experimental drug use group did not differ from the light drinking/rare drug use group in any personality indicators. Thus, from the point of view of their prevalence in the sample, their amount of drug use, and their personality characteristics, the moderate drinking/experimental drug use group was relatively normative. Yet, they still ran the risk of developing alcohol dependence. This might reflect a group who began their drinking in relatively developmentally normative social contexts (that promote alcohol use) but whose exposure to alcohol progressed into a clinical disorder. Moreover, compared with the heavy drinking/heavy drug use group, their alcohol dependence was less likely to be either comorbid with drug dependence or persistent over time. This consumption trajectory might be more likely to produce the "developmentally limited" alcohol disorder that has been described by other researchers (Zucker et al., 1995).

In terms of diagnosed dependence, our mixture modeling revealed groups that tended toward either alcohol or drug dependence alone as well as a comorbid group, with most participants (61%) not developing a dependence diagnosis. Thus, alcohol and drug disorders showed both independence and comorbidity, suggesting that their associated risk pathways should also likely show both some specificity and some commonality. Kendler et al. (2003) found a similar pattern of specificity and commonality in terms of genetic influences on alcohol and drug disorders. They suggested that heritable personality diatheses towards behavioral undercontrol might elevate risk for both alcohol and drug disorders, whereas heritable individual differences in sensitivity to or metabolism of a particular drug might elevate risk for a single substance use disorder in isolation. Similar commonality and specificity might be hypothesized for environmental risk mechanisms. For example, poor parenting is likely to produce conduct problems, which could elevate risk for both alcohol and drug disorders. In contrast, peer social norms favoring the use of a specific substance could raise risk for the use or abuse of a specific substance. Although the current findings support the presence of both common and drug-specific risk mechanisms, they cannot separate genetic and environmental influences (which are likely to interact within multivariate biopsychosocial pathways; Sher, 1991).

Given previous theories about subtypes of substance use disorders (e.g., Cloninger, 1987) as well as previous empirical studies of alcohol or tobacco use (e.g., Chassin et al., 2000), it was somewhat surprising that our mixture modeling did not reveal unique subgroups as a function of age of onset. Instead, all groups showed initiation between adolescence and emerging adulthood. There are several likely reasons why our data may not have revealed different onset subtypes. First, the participants were monitored only to ages 27–30. Later onset forms of alcohol and drug problems may not emerge until later in adulthood, and subgroups with differing adolescent onset (e.g., early vs. late adolescence) may have been too small to be detectable with the current sample size. Second, because we were most interested in comorbidity, we modeled bivariate trajectories of alcohol and drug outcomes. In these analyses, the most distinctive patterns (which are most likely to emerge as distinct groups) are likely to involve differential relations between alcohol and drug outcomes rather than differing ages of onset (Sher & Jackson, 2004).

We also tested whether familial alcoholism was particularly related to trajectories of alcohol outcomes compared with drug outcomes, and whether familial alcoholism was uniquely predictive above and beyond other parent psychopathology. Our results showed that familial alcoholism was a unique predictor above and beyond other parent psychopathology, but that its effects were not restricted to alcohol outcomes. Rather, COAs were overrepresented in the heavy drinking/heavy drug use group and in any of the diagnosed groups compared with the nondiagnosed group. Moreover, family history density of alcoholism was a more sensitive predictor than was parent alcoholism in that it also distinguished among diagnosed groups. Those with dense family histories (averaging more than one first degree alcoholic relative) were overrepresented in both the comorbid and alcohol dependence only groups compared with the drug only and persistent groups. These findings further support the notion of both common and drug-specific risk pathways. They suggest that familial alcoholism effects may best be viewed as conveying both specific risk for alcohol disorder and also broader risk for heavy drug use and comorbid alcohol and drug dependence (particularly in cases in which there is alcoholism among multiple family members). Heritable individual differences in alcohol metabolism and sensitivity to its pharmacological effects may help to explain the association between family history of alcoholism and the alcohol only dependence group. However, broader personality risk for behavioral undercontrol may underlie the association between familial alcoholism and heavy drug use or comorbid dependence. Our finding that adolescent impulsivity mediated the relation between family history risk and drug or comorbid disorders supports this interpretation. It is also consistent with previous theory and data, which suggests that a heritable diathesis toward behavioral undercontrol is associated with both comorbidity of alcohol and drug dependence (Kendler et al., 2003; Krueger et al., 2002, Vanyukov et al., 2003), and with familial alcoholism (Iacono et al., 1999; Sher et al., 1991).

Of course, an effect of familial alcoholism on both alcohol and drug outcomes might also be produced in our data if these alcoholic parents themselves showed high rates of comorbid drug disorders. However, this was not the case in the current sample, in which 25% of the alcoholic families (and fewer than 4% of the nonalcoholic families) had a parent who reported more than two lifetime drug consequences or dependence symptoms at baseline. In our analyses, these parent drug problems could not account for the parent alcoholism effects and did not significantly differentiate the trajectory groups. The relatively low levels of parental drug problems might be due to our requirement that the biological

alcoholic parent also be a custodial parent. Perhaps families with parental comorbid alcohol and drug disorders are less likely to stay together until their children reach adolescence and, thus, are underrepresented in our sample.

The current mediational findings suggest that those with a family history of alcoholism were at risk for heavy alcohol and drug use and diagnosed substance dependence in part because they were impulsive and low in agreeableness. Impulsive COAs are more likely to experience school failure, ejection from conventional peer groups, and affiliation with peers who model substance use, and they are also less likely to be deterred by negative consequences of use. Individuals who are low in agreeableness are not only more likely to be characterized by negative emotionality (Church, 1994) but they also report stronger coping motives for using alcohol (Loukas et al., 2000). Thus, they may use substances to regulate negative affect. Moreover, those who are low in agreeableness are also characterized by hostility, aggression, self-centeredness, and indifference to others (Costa & McCrae, 1992) and may use alcohol and drugs because they place lower value on external norms and expectations. Nevertheless, these significant mediational paths could not entirely account for parent alcoholism effects, suggesting that other etiological pathways also operate.

Finally, following McGue et al. (1999), we tested whether negative emotionality (represented by EASI emotionality and NEO neuroticism and agreeableness) would be more strongly related to alcohol dependence, whereas low constraint (i.e., EASI impulsivity, NEO conscientiousness and openness) would be more strongly related to drug dependence. This hypothesis received only partial support. Neuroticism, agreeableness, and emotionality all differentiated the alcohol dependence only group from the nondiagnosed group. However, although these findings suggest a relation between negative emotionality and alcohol dependence, this relation was not unique to alcohol outcomes. Rather, the drug dependent only group showed similar or higher levels of these characteristics than did the alcohol only group, and the comorbid drug and alcohol dependence group was the most extreme among the diagnosis groups in these indicators. Furthermore, as in previous research (Jackson et al., 2000), the effects of negative emotionality (in this case the effects of neuroticism and emotionality) were decreased or eliminated when indicators of constraint were also included in the models.

Similarly, partial support was found for a stronger link between constraint and drug dependence than between constraint and alcohol dependence. Adolescents who were most impulsive were more likely to show comorbid alcohol and drug dependence than alcohol dependence only, and young adults who were low in conscientiousness were more likely to be in the drug dependence only than the alcohol dependence only diagnostic group. However, again, the support was only partial. The effects of conscientiousness were not maintained over and above the other personality characteristics, and the drug only and alcohol only groups did not significantly differ in other indicators of constraint. Moreover, low conscientiousness, high openness, and high impulsivity also characterized the persistent group (most of whom were diagnosed with alcohol rather than drug dependence).

Weaker findings in our data compared with the McGue et al. (1999) data may be due to differences in samples, measures, and group definitions. McGue et al. examined an older sample, which would be more likely to include late-onset cases with potentially different personality characteristics. Moreover, McGue et al. defined "pure" groups of alcohol and drug disorders (as well as a comorbid group), whereas we compared empirically derived, naturally occurring trajectory groups (making it more difficult for our data to completely separate alcohol and drug outcomes). Finally, indicators of negative emotionality and constraint may be less clear in our five-factor approach to personality than they were in the Multidimensional Personality Questionnaire used by McGue et al. (1999). For example, although low agreeableness has been reported to be an indicator of emotionality (Church, 1994), its correlation with externalizing symptoms suggests that it may also indicate low constraint (R. Robins, John, Caspi, Moffitt, & Stouthamer-Loeber, 1996).

Although our data did not entirely replicate those of McGue et al. (1999), they did show a systematic pattern in which adolescent temperament prospectively predicted trajectories of use and diagnosis, and young adult personality significantly correlated with these trajectories. In our findings, the extreme groups in terms of personality factors generally were the abstainers and the groups that showed comorbidity (i.e., the heavy drinking/heavy drug use group and the comorbid dependence group), suggesting that the combination of alcohol and drug consumption and dependence is characterized by the highest levels of negative emotionality and lowest constraint. These findings support the McGue et al. recommendation that the co-occurrence of alcohol and drug use should be considered in linking personality to substance use disorders.

Finally, it is important to note some of the limitations of the current study. First, participants were followed only to age 30, so that late onset forms of substance use could not be considered. Second, our focus on modeling bivariate trajectories of alcohol and drug outcomes was likely to produce groups that were characterized more by distinct patterns of alcohol and drug co-occurrence than by differences in age of onset or cessation, so these effects may be underemphasized in our data. Although the use of empirically derived groups is advantageous for capturing patterns of naturally occurring substance use phenomena, the patterns that are obtained vary with the characteristics of the study samples and measures that are chosen. For example, studies of alcoholic families in which there is more substantial comorbid drug dependence might produce different findings. Thus, although the current trajectory groups were consistent with theory and epidemiological data, as well as similar to other multiple trajectory study findings, they are not the only way to describe patterns of alcohol and drug use and dependence over time. Finally, diagnostic data were obtained at only three of the five assessment waves so that retrospections about symptom onset and offset contributed to those trajectories. Given these limitations, it would be useful to replicate these findings with studies of larger samples followed more frequently over longer time spans.

In short, the current study sought to describe and predict trajectories of alcohol and illegal drug use and dependence from adolescence to adulthood. Results suggested that a trajectory characterized by heavy alcohol and drug use was most likely to result in diagnosed substance dependence, including the highest risk of comorbid and persistent disorders, and was associated with familial alcoholism and lack of constraint. Moreover, even a less extreme trajectory of moderate alcohol use and experimental drug use was associated with some risk for alcohol dependence, although it was less likely to result in comorbid or persistent disor-

ders, and it had less associated familial alcoholism and personality risk. Familial alcoholism elevated risk for both alcohol and drug dependence in part because of heightened impulsivity and neuroticism and lowered agreeableness. Familial alcoholism may convey both specific risk for alcohol disorders (perhaps through individual differences in alcohol sensitivity and metabolism) and broad risk for comorbid drug and alcohol disorders (in part because of dispositions toward behavioral undercontrol). Naturally occurring trajectories of alcohol and illegal drug use and dependence may be useful outcomes in modeling the etiology of substance use disorders, and future studies might further distinguish these trajectories.

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