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# Genetic and Environmental Predictors of Latent Trajectories of Alcohol Use from Adolescence to Adulthood: A Male Twin Study

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## Abstract

**Background—**Adolescence is characterized by higher levels of novelty-seeking and risk-taking behavior, including initiation of alcohol use. Also, there is considerable heterogeneity in the change and continuity of alcohol use over time, which emphasizes the need to examine factors predicting alcohol use and the patterns of use over time.

**Methods—**Retrospective data on average monthly alcohol use and risk and protective factors were obtained through interviews and questionnaires in 1,560 adult male twins. Latent class growth analysis in Mplus was performed on data of alcohol use over ages 15 to 36. Second, logistic regression analyses were used to associate risk and protective characteristics with membership in distinct latent trajectories of alcohol use.

**Results—**Six trajectories of alcohol use were identified, varying in the level of alcohol use, the rate of change in use in early adolescence and the persistence of use into adulthood. Genetic risk of externalizing disorder and peer deviance showed the greatest risks for unfavorable alcohol trajectories with higher levels of use and higher rates of early increase in use. Parental monitoring and involvement in social activities showed protective effects. Involvement in religious activities was strongly associated with reduced persistence of high-level drinking in univariate but not multivariate regression analyses.

**Conclusions—**Risk and protective factors impacted differentially on *level* of alcohol use, *rate of increase* in use during adolescence, and *persistence* of heavy alcohol use over time. Insight into the different ways in which predictors impact on alcohol use is relevant for the development of new intervention strategies. For this purpose, causality of the associations should be further examined.

### Keywords

Latent Class Growth Analysis; Adolescence; Alcohol Use; Genetic and Environmental Risk Factors

Alcohol dependence is widespread with a life-time prevalence of 14% in the general population of the United States and 29% in first-degree relatives of affected individuals (Nurnberger et al., 2004) and imposes enormous societal costs (0.45 to 5.44% of gross



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domestic product in developed countries; Mohapatra et al., 2010). Adolescence is a developmental period characterized by higher levels of novelty- seeking and risk-taking behavior, including initiation of alcohol use (Dayan et al., 2010). High alcohol consumption, especially during adolescence, is a strong risk factor for problematic alcohol use later in life and other psychological and behavioral problems (Crews et al., 2007; Nixon and McClain, 2010; Steinhausen et al., 2008). Yet despite the high prevalence of lifetime adolescent alcohol use (90%), only a subgroup (around 10 to 30%) develops problematic drug use (Weber et al., 1989).

Heavy alcohol use is associated with a number of risk factors. For instance, both genetic and environmental factors are found to affect levels of alcohol use from adolescence to adulthood (Meyers and Dick, 2010). Risk factors include family history of substance misuse and parent alcohol use (Chassin et al., 2002; White et al., 2000), poor school performance, peer deviance (Corte and Sommers, 2005), easy access to alcohol (Danielsson et al., 2010), poor behavioral regulation and attention deficit hyperactivity disorder (ADHD; Colder et al., 2002; Knop et al., 2009; Lopez et al., 2008; Tucker et al., 2003; Wennberg, 2002), early life stress (Enoch, 2011), delinquency and personality traits like sensation seeking, impulsivity (Bates and Labouvie, 1997), and neuroticism (Grabe et al., 2011). Protective factors include high levels of religiosity and church attendance (Bates and Labouvie, 1997; Boomsma et al., 1999; D'Onofrio et al., 1999; Kendler et al., 2003), increased parental monitoring, a nonpermissive attitude regarding substance use (Capaldi et al., 2009; Duncan et al., 1998; Moore et al., 2010), and involvement in social activities (Kristjansson et al., 2010). However, it is unclear how these risk factors affect varying patterns and stages of alcohol consumption.

Previous studies in adolescents have shown considerable heterogeneity in various aspects of alcohol use patterns, such as heterogeneity in onset and level of drinking (Chassin et al., 2002; Danielsson et al., 2010; Li et al., 2002; Van der Vorst et al., 2009; White et al., 2000), in the rate with which alcohol use increases at the start of adolescence (Colder et al., 2002; Danielsson et al., 2010; Tucker et al., 2003), and in the persistence of high-level alcohol use (Colder et al., 2002; Van der Vorst et al., 2009; White et al., 2000; Wiesner et al., 2007). It needs to be further elucidated how both risk and protective factors differentially influence these aspects of alcohol use trajectories. For instance, early onset of alcohol use and a rapid increase in use following onset have been associated with a developmental course of increased problem behavior (Weber et al., 1989) and peer deviance (Bates and Labouvie, 1997; Duncan et al., 1995, 1998; Li et al., 2002). One study found that parental and peer alcohol use was associated with initial levels of alcohol consumption, whereas peer alcohol use was more predictive of changes in alcohol use over time (Capaldi et al., 2009). Finally, a twin study showed that genetic factors accounted for a large part of the variation in age of alcohol initiation but not for frequency of drinking (Poelen et al., 2008).

There is therefore a need to examine not only *what* risks and protective factors are related to alcohol use in adolescence, but also *how* these factors relate to patterns of alcohol use over time. Knowledge on whether factors influence the level of drinking, the rate of change in average alcohol use following initiation of alcohol use or the persistence of drinking in the transition toward adulthood has relevance for future intervention strategies.

In this article, we model the association between risk and protective factors and patterns of alcohol use from adolescence to adulthood using a large sample of male twins. Strengths of the current approach are as follows: (i) this is the first study to our knowledge to examine the role of genetic risk in trajectories of alcohol, (ii) the inclusion of a broad range of predictors—both risk and protective factors—for alcohol use, and (iii) the wide age interval (15 to 36) over which the development of alcohol use is examined.

# MATERIALS AND METHODS

## Sample

Data were collected as part of the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders (VATSPSUD). Described in detail elsewhere, the VATSPSUD includes 3 waves of male–male and male–female twin pair data collection (wave 1: 1993 to 1996, wave 2: 1994 to 1998, and wave 3 1998 to 2004; Kendler et al., 2008). The third wave was restricted to male–male twins only. This report uses the male twin pairs (1,796 male twins) who participated in both the second and third waves (Kendler et al., 2008). At wave 3, the mean age of the sample was 40.3 (SD = 9.0, range, 24 to 62). The sample for the present analysis ($n$ = 1,796 individuals) consisted of 469 monozygotic (MZ) and 287 dizygotic (DZ) twin pairs as well as 290 twins whose co-twin did not participate. For 2 sets of triplets, the number of twin pairs was based on all possible within-family pairings; therefore, 6 twin pairs are represented by 6 individuals. Most subjects were interviewed by telephone. The study was approved by the Ethics Committee, and informed consents were obtained before face-to-face or telephone interviews took place. Although most subjects were interviewed by telephone for the male–male third wave, a small number were interviewed in person because of subject preference, residence in an institutional setting, or not having telephone service.

Interviewers possessed a master's degree in social work, psychology, another mental health–related field, or a bachelor's degree in 1 of these areas plus a minimum of 2 years relevant clinical experience. They received 40 hours of classroom training plus regular individual and group review sessions. Two senior staff members reviewed each interview for completeness and consistency. Each member of a twin pair was interviewed by a different interviewer who was blind to clinical information about his co-twin. Zygosity assignment was based on a combination of self-report measures, photographs, and DNA polymorphisms (Kendler et al., 2000).

## Assessments

**Alcohol Use—**Alcohol use from adolescence to adulthood was assessed retrospectively at wave 3. To improve recall accuracy, a life history calendar method (Freedman et al., 1988) was used. Empirically, this approach has been shown to improve the accuracy of retrospective reporting by providing multiple cues to increase the chances of accurate recall (Belli, 1998). The method makes the task more akin to the accurate and well-retained process of recognition than to the less reliable task of free recall. Measures included the average number of days per month on which alcoholic beverages were consumed and the number of drinks consumed per day when drinking. One drink was defined as 1 bottle of beer, 1 glass of wine, or 1 shot of liquor. For alcohol use, the average number of drinks per month was calculated for each year starting from age 15 to age 36. To correct for skewness, the monthly average was recoded onto a 5-point ordinal scale (0 = 0, 1 = 1 to 12, 2 = 13 to 50, 3 = 51 to 190, and 4 = 190 > drinks per month).

**Risk Factors—**Based on family reports on father's, mother's, and co-twin's alcohol use ascertained at wave 2, *genetic risks for problem alcohol use and externalizing disorders* were estimated using "relative to an identified distribution" (RIDIT) scores. A RIDIT score is a nonparametric method for scoring ordinal information without assuming any underlying theoretical distribution. Analogous to an average percentile, RIDIT scores are deviations or expected values away from a mean of 0.5 under a uniform distribution (0 to 1). They is an estimate of the chance that an individual in a given ordinal class is "worse off" compared with individuals in other classes. RIDITs are calculated as the proportion of individuals in the preceding category plus one-half of the proportion of individuals in the category itself (Bross, 1958). A binary variable for example with prevalence rates of 40% or 0.4 and 60%

or 0.6 (i.e., 0 to 40%, 41 to 100%) would therefore be scored as 0.2 (0.0 + 0.5 × 0.4) and 0.7 (0.4 + 0.5 × 0.6). However, if we assume that genetic factors are responsible for familial aggregation in certain complex traits (Meyers and Dick, 2010), then less regression toward the population mean of 0.5 is expected among individuals who are more genetically related. Therefore, RIDIT scores for DZ twins (who share 50% of their genes identical by descent) are weighted half-way back toward the mean of 0.5 because of the greater expected regression to the mean. Individual risk was thus calculated as the average of the adjusted RIDIT scores. This weighting was not applied to MZ twins because they are genetically identical; less regression to the mean is expected because the MZ co-twin provides a better index of risk (Kendler et al., 2011). A genetic risk of externalizing disorder was calculated in a similar way based on the co-twin's self-report symptoms of Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) conduct disorder, antisocial personality disorder (American Psychiatric Association, 1994), as well as the twin's reporting of antisocial personality disorder in their co-twin and father using the Family History–Research Diagnostic Criteria (Lavori et al., 1988).

*Sensation seeking* was based on a continuous measure based on 11 items from the Sensation Seeking Scale at wave 3 (Zuckerman and Neeb, 1979). We calculated kappas and intraclass correlation (ICC) for ordinal and continuous response distributions, respectively. Reliability for this variable (ICC) equaled 0.81. *Childhood physical or sexual abuse* was assessed using 2 items assessing (i) physical abuse as a child or (ii) sexual abuse or molestation before the age of 16.

*Peer group deviance* was assessed using 12 items based on 2 validated instruments (Bachman et al., 1981; Tarter and Hegedus, 1991) that evaluated the proportion of the respondent's friends who smoked; drunk alcohol; had problems with alcohol; had been in trouble with the law; stole or damaged property; smoked marijuana; used inhalants; used other drugs like cocaine, downers, or LSD; and sold or gave drugs to other kids. Reliability (ICC) equaled 0.81. Early peer group deviance was defined as peer deviance at ages 12 to 14 while late peer group deviance was measured between ages 22 and 25.

*Attention deficit hyperactivity disorder* was measured using 14 items based on the DSM-IV symptoms for ADHD (11 items) and oppositional defiant disorder (3 items) which asked subject what they were like when they were "growing up" 37. Reliability (ICC) equaled + 0.81. Response options for items such as "How often did you have difficulty staying seated" were "never," "rarely," "sometimes," and "often." *Neuroticism* was assessed by the Short-Scale version from the EPQ-R at wave 1 (Eysenck and Barrett, 1993) scored as a 5 level ordinal measure.

*School performance* was assessed using 4 questions on failing subjects, repeating grades, suspension, and expulsion from school. These questions were asked for the time period up to the end of high school or the highest grade completed.

Protective factors *Involvement in social activities* was assessed at ages 8 to 11, 12 to 14, and 15 to 17 using 3 items, each on a 4-point scale ("never," "rarely," "sometimes," and "often"), from the Monitoring the Future (MTF) questionnaire. These items asked how often subjects participated in organized sports activities (including little league or school teams), school clubs or bands, or community activities (YMCA, scouts or other clubs).

Two additional items of the MTF questionnaire assessed *involvement in church-related activities* for the same age periods. These items asked how often subjects participated in church activities or youth groups ("never/rarely/sometimes/often") and how often they attended religious services ("more than once a week/once a week/a few times a month/once a month/less than once a month/never"). Reliability for this variable (ICC) equaled 0.88.

*Parental attitudes* toward drug use were based on 7 items asking subjects about their parents' toward the tobacco, alcohol, and other drug use up until age 18. Subjects were asked whether their parents would "strongly agree/agree/disagree/strongly disagree" or "don't know" with statements such as "Is it OK for a teenage to smoke cigarettes?" "Is it OK for a teenage to get drunk?" and "Is it OK for a teenage to use marijuana?" The reliability (weighted kappa) was 0.43. Higher scores reflect stronger attitude against drug use.

*Parental monitoring* was assessed using 3 (age period, 8 to 11) or 4 items (age, 12 to 14, 15 to 17) based on previous work examining parental effects on risk of drug use and delinquency (Kerr and Stattin, 2000). Items asked subjects how much their parents or guardians knew who their friends were, how they spent money, and what they did with free time, and knew where they were at night. Response options were (they) "didn't know/knew a little" or "knew a lot." Reliability (weighted kappa) equaled 0.69.

Short-term test–retest reliability was available on variables assessed at wave 2 (195 subjects interviewed an average of 31 days apart) and wave 3 interviews (141 subjects interviewed an average of 29 days apart), but not for variables entirely or partially assessed at the first wave interview.

**Marginal Maximum Likelihood Risk Factor Scores—**With the exception of the genetic risks, and physical or sexual abuse, we estimated for each individual a maximum likelihood factor score using the Mx software program (Neale, 1999). Based on a single common factor model, this method isolated factor- and item-specific variance including measurement error for each risk factor to quantify more precisely each individual's latent risk.

## Statistical Analysis

Analyses were performed in 3 steps similar to a recent study examining latent trajectories (Barker and Maughan, 2009). First, models for developmental latent trajectories of alcohol use were estimated using Mplus version 5.1 (http://www.statmodel.com/). To reduce dimensionality (and thereby computational complexity), we averaged the number of drinks per month for each 2-year period, which resulted in 11 time points from ages 15 to 36. Participants who had never drunk any alcohol during their lifetime were excluded from the analyses, because they would form a separate latent class by definition. The Mplus cluster option was used to control for clustering of individuals within twin pairs.

To identify subgroups of alcohol users, latent class growth analysis (LCGA) models were fit to the longitudinal data. LCGA assumes that the sample is composed of a mixture of distinct subgroups with different growth patterns and because LCGA allows for between-group differences in the shape of alcohol use trajectories over time, it can identify and model heterogeneity in terms of developmental trajectories.

To determine the number of latent classes (trajectories), several model fit indices were used. A lower log-likelihood indicates better fit of the model. Similarly, lower Akaike's information criteria (AIC) and Bayesian information criteria (BIC) values indicate a better compromise between explanatory power and fewer estimated parameters (parsimony). Additionally, a small $p$-value for the adjusted Lo–Mendell– Rubin likelihood ratio test (LMR-LRT) suggests that the model with $k$ classes is preferred over $k - 1$ classes. Entropy refers to the average classification accuracy of participants to classes and ranges from 0 to 1. The higher the entropy, the better the model can classify individuals into 1 of the latent classes. The best-fitting model was used for further analyses as described under step 2 and 3.

In step 2, we fitted *univariate* logistic regression models in STATA 11.1 (StataCorp, College Station, TX). Predictors were thus entered separately into the regression models to examine the capacity of risk and protective characteristics to distinguish membership in the identified alcohol use trajectories. Analyses were weighted by the individual class membership probabilities estimated in Mplus. Age at interview was included as a covariate because of prior evidence that individuals across this age span differ in the prevalence of drug use disorders (Prescott and Kendler, 1999). In step 3, we fitted *multivariate* logistic regression models in a similar way as described under step 2, but now by entering all predictors simultaneously in the regression models. Bonferroni correction was applied to the analyses performed under step 2.

## RESULTS

### Step 1: Latent Class Growth Analysis

Among the 1,796 male twin individuals sampled, 1,560 (86.9%) reported alcohol use between 15 and 36 years. Participants under age 36 reported only on their alcohol consumption up to their current age. Therefore, the number of reports on alcohol use in age ranges above age 24 declined (≤24: 1,560 obs; 25 to 26: 1,535 obs; 27 to 28: 1,461 obs; 29 to 30: 1,359 obs; 31 to 32: 1,242 obs; 33 to 34: 1,104 obs; and 35 to 36: 984 observations). As shown in Table 1, entropy across all of the LGCA models was satisfactory (>0.800 [based on personal communication at the MPlus Home website]). The *p*-value of the LMR-LRT test was no longer significant with a 7-class model. However, as most fit indices continued to decline, post hoc we also looked at additional factors including the prior literature (Colder et al., 2002; Van der Vorst et al., 2009; Wiesner et al., 2007) and interpretability to arrive at a 6-class solution. The 6-class solution was easier to interpret than other solutions as it presented contrasting classes on dimensions of alcohol use, persistence, and peak use, which are tested below.

Figure 1 shows these 6 latent trajectories of the development of alcohol use with the total number (and percentage) of individuals in each class. These trajectories differ from each other in some relevant characteristics such as the maximum level of alcohol use, persistence, and rate of change in average alcohol use early in adolescence. Regarding the maximum level of alcohol use, class 5 and 6 both show low maxima, class 3 and 4 show average maxima, and class 1 and 2 show the high maximum amounts of alcohol use. Furthermore, class 1 and 2 both show a similar large increase in alcohol use during adolescence. However, after age 23 to 24, alcohol use declines in class 1 but persists in individuals in class 2. Finally, class 3 and 5 are characterized by relatively rapid increases toward the maximum level of alcohol use, while class 4 and 6 are characterized by a relatively slow increase in alcohol use toward the maximum level.

### Step 2: Prediction of Type of Trajectory by Relevant Risk and Protective Factors

The effect of risk and protective variables was tested using 4 contrasts chosen to illustrate differences in the level of use, persistence of use, and peak use of alcohol. These included the effect of risk and protective variables on (i) high alcohol use (class 1,2) versus low alcohol use (class 5,6); (ii) high alcohol use (class 1,2) versus moderate alcohol use (class 3,4; however, note that class 4 shows more alcohol use than class 2 after age 28); (iii) high and persistent alcohol use (class 1) versus high but decreasing alcohol use (class 2); and (iv) shape of curve: early rise in alcohol use followed by decline (class 3,5) versus a gradual increase and leveling off (class 4,6). These contrasts were chosen to illustrate differences in level of use, persistence of use and peak use of alcohol. As class 1 and 2 were similar in the timing and steepness of increase in use and also resembled one another in the maximum

amount of alcohol use, these 2 classes were contrasted with each other to examine risk factors influencing persistence.[1]

**Risk Factors: Univariate Regression Results—**Following Bonferroni correction, nearly all risk factors were significantly and positively associated with "high" versus "low" drinking trajectories (Table 2). Some additionally discriminated between "high" versus "moderate" drinking trajectories. The largest effects were those of early and late peer deviance and genetic risk of externalizing disorder. One SD above average on late peer deviance increased the risk of belonging to a high alcohol use trajectory by more than 4 times. Furthermore, genetic risk of externalizing disorder and alcohol use disorder (the latter only with borderline significance), early and late peer deviance and ADHD symptoms were associated with a "shape of curve" over time of alcohol use. After Bonferroni correction, no risk factors were associated with "persistent high" drinking when compared with "high but later reduced" drinking.

**Protective Factors: Univariate Regression Results—**Three predictors showed significant protective effects. These effects concerned associations with membership of "high" versus "low" alcohol use trajectories and "peak and decline" versus "gradual increase" increase in alcohol use trajectories. Only involvement in church-related activities was borderline significantly (nominal $p = 0.001$) associated with lower persistence of high-level drinking (see Table 2).

### Step 3: Multivariate Regression Results

Only peer deviance and genetic risk of alcohol use disorder, but not genetic risk of externalizing disorder, were significantly associated with patterns of alcohol use in the multivariate analyses. Both were associated with the level of alcohol use and with an early peak versus gradual increase in alcohol use (see Table 3).

## DISCUSSION

### Findings

Consistent with reports that have sampled subjects at similar age groups (34 to 37), alcohol users differed in average alcohol use and change in rates of alcohol consumption over time. Differences in developmental patterns were associated with environmental risk profiles. A number of risks that predicted *average alcohol consumption* did not always predict *the pattern (shape) of alcohol use over time*. For instance, sensation seeking and low school performance were associated with a higher average level, but not with an early peak pattern of alcohol use. On the other hand, peer deviance, ADHD symptoms, genetic risk of externalizing disorders, and alcohol use additionally impacted on the timing (peak and decline vs. gradual increase) of alcohol use in early adolescence.

Among the most striking findings was that genetic risk for externalizing problem behavior and peer deviance were the strongest predictors of class memberships with initial high average consumption and with a peak pattern in consumption in use during adolescence (see Table 2 for odds ratios). Noticeably, genetic factors associated with externalizing problem behavior seemed to have stronger effects on the level of alcohol use than genetic factors

[1]Although class 1 and 2 could also be differentiated in an early rise with decline and a gradual increase in alcohol category, we did not include them in the fourth contrast. The other classes are comparable in that the maximum of class 3 is similar to that of class 4 and the maximum of class 5 is similar to that of class 6. Class 1 and 2 are not comparable in that sense. Class 1 consists of individuals who always consume similar or higher amounts of alcohol than those in class 2. Adding class 1 to the other "gradual increase" classes and class 2 to the other would confound "shape of curve" with "level of alcohol use".

associated with alcohol use disorders directly. This is consistent with findings of a previous study (Kendler et al., 2011).

It was striking that no risk factor influenced the *persistence* of extremely high levels of alcohol use in the transition toward adulthood. Although genetic risk for externalizing problem behavior, sensation-seeking, late peer deviance, and neuroticism all showed large effects on membership of the high and persistent use versus the high but decreasing use trajectory (OR = 1.37, OR = 1.39, OR = 1.63, and OR = 1.35, respectively), the results were no longer significant after Bonferroni correction. Given their potential clinical significance, future studies should examine these associations in larger samples and what evidence there may be for a causal relationship.

The main protective factors were parental monitoring and involvement in regular or church-related social activities. All reduced the likelihood of belonging to *high-level alcohol use* trajectories, but showed different patterns regarding associations with the *timing in average alcohol use* in adolescence and *persistence of high-level drinking* in the transition toward adulthood.

Another striking finding was that the effects of genetic risk of alcohol use disorder were independent from the effects of late peer deviance, while the effects of genetic risk of externalizing disorders were not (see Table 3). Likely, the latter mediated the effects of a cluster of behaviors under which peer deviance, low school performance, and ADHD symptoms, resulting in non significant associations when entered in the model simultaneously. In contrast, genetic risk for alcohol use seems to be relatively independent from this cluster of behaviors in its influence on alcohol use.

### Comparison to Previous Literature: Risk Factors

The strong peer effect on alcohol use has been reported elsewhere (Bates and Labouvie, 1997; Duncan et al., 1995, 1998; Li et al., 2002). Also, this study confirms that an early peak in alcohol use is associated with increased levels of ADHD and genetic risk of externalizing disorder, which together with peer deviance fits Weber and colleagues' (1989) hypothesis that a sudden increase in use is associated with higher levels of problem behavior. Chassin and colleagues (2002) also reported that early and heavy alcohol use was associated with higher levels of adolescent problem behavior.

Genetic influence on alcohol use (Lindell et al., 2010; Meyers and Dick, 2010; Zhang et al., 2006) and timing of initiation of use (Poelen et al., 2008) has been established previously. One recent study found that the 5-HTTLPR genotype predicted the adolescent's growth in alcohol use over time (van der Zwaluw et al., 2010). The current study is the first study associating genetic risk with distinct latent trajectories.

### Comparison to Previous Literature: Protective Factors

In general, previous studies have found a protective effect of parental monitoring on alcohol use; however, precise results slightly varied between an effect on rate of change in average alcohol use (Capaldi et al., 2009), *initial levels* at age 14 (Duncan et al., 1998), or *average* adolescent alcohol consumption (Moore et al., 2010). The current study replicated previous findings regarding the associations between higher parental involvement and (i) *lower self-reported levels* of alcohol consumption (Moore et al., 2010) and (ii) a *more gradual increase* in alcohol use during adolescence. Caution, however, should be taken interpreting the association as a protective effect. A score on parental monitoring may well be driven by adolescent disclosure instead of active parenting. The current study is also in accordance with previous studies reporting a protective effect of religiousness or church attendance on alcohol use (Bates and Labouvie, 1997; Boomsma et al., 1999; D'Onofrio et al., 1999;

Kendler et al., 2003; Windle et al., 2005). However, in the current study, the effect of church attendance was no longer significant in the multivariate analyses. It can be speculated that other predictors, such as late peer deviance, now account for variance previously explained by church attendance. Although for "persistence" peer deviance showed a trend only in the multivariate analysis, in combination with other predictors, it likely still reduced the explained variance for church attendance. The current findings suggest that participation in organized social activities (sports, school clubs or bands, community activities), especially in religiously affiliated activities, may be protective against the development and persistence of problematic alcohol use. The potential implications of this finding suggest the need to further examine the causality of these associations.

Finally, in contrast to other studies (Moore et al., 2010; Tucker et al., 2003; Van der Vorst et al., 2009), the current results showed that a higher negative parental attitude toward drug use was associated with higher levels of use and a more sudden increase in use in adolescence. It is possible both that parental attitudes might have been driven by the adolescent's behavior or that adolescents were rebelling against restrictive parental attitudes. A recent systematic review suggests that parental disapproval is associated with lower levels of alcohol use in adolescents (Ryan et al., 2010).

## Methodological Issues

Although our modeling has revealed population heterogeneity in the patterns of alcohol use, our findings must be interpreted in the context of the following limitations. First, our data were drawn from White males born in Virginia and may not extrapolate to females or males from other populations. Males do have a higher prevalence of drug and alcohol use (Nolen-Hoeksema, 2004); however, previous analyses using the same data suggest that this sample is broadly representative of U.S. males and does not differ from the general population in rates of psychopathology, drug use, and abuse (Kendler et al., 2000). Second, our model fitting was not exhaustive. However, we believe that our LGCA model is appropriate because it integrates over developmental time a large number of diverse risk factors related to patterns of alcohol use.

A disadvantage of the current design is that alcohol use was assessed retrospectively. However, it has been established in previous studies that reliability of retrospective recall of alcohol intake using the Life History Calendar method is good (Czarnecki et al., 1990; Koenig et al., 2009). Previous studies even suggested that retrospective assessments may suffer less from underreporting than prospective assessments of alcohol use (Czarnecki et al., 1990; Koenig et al., 2009).

Significant effects of age were found on alcohol use depending on the period on which they reported. Participants in the highest tertile of the age distribution (over 44 years of age) reported less alcohol use between age 15 and 24, but more alcohol use between age 27 and 32. This could mean: (i) either there are cohort effects in that over the years the maximum level of alcohol use has shifted to a younger age (ii) or there is a tendency to "forget" and underestimate alcohol use when more time has passed since the period for which they have to estimate their drinking. However, the negative effect of age on alcohol use was not found for reports on higher age periods (age period, 33 to 36). Therefore, it may be more likely that these represent real cohort differences. This is also supported by a report on alcohol use in the United States over the past decades (Secretary of Health and Human Services, 1997). All analyses were corrected for current age.

Finally, many of our predictors are correlated. Therefore, we can be much more confident about the association than the causes of the associations we are observing.

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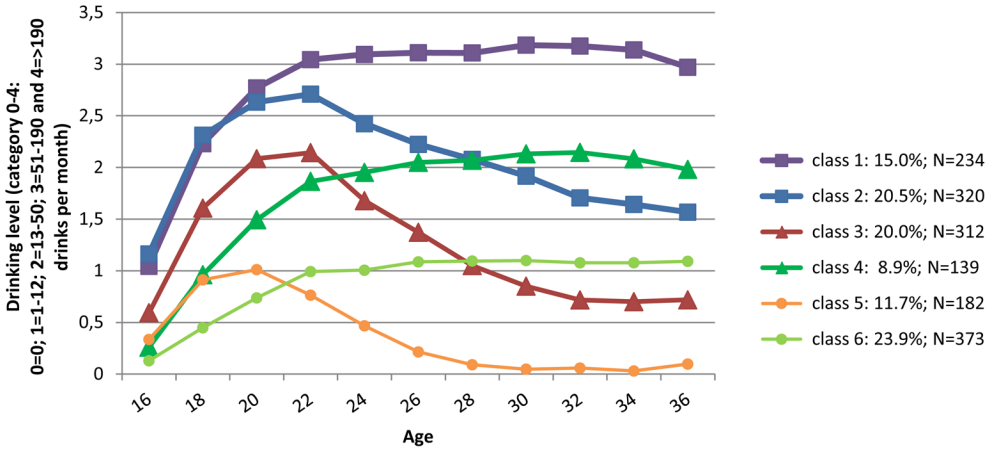


**Fig. 1.**

Best fitting model with 6 latent trajectories of alcohol use. Each measurement point represents the amount of consumed alcohol units (in categories) per month averaged over a period of 2 years (resulting in 11 time points for age 15 to 16; 17 to 18; 19 to 20; 21 to 22; 23 to 24; 25 to 26; 27 to 28; 29 to 30; 31 to 32; 33 to 34; and 35 to 36, here indicated by the age at the end of each period).

**Table 1**

Comparison of Fit of Latent Growth Curve Analysis

| No. classes | LL | AIC | Adj BIC | LMR-LRT | LRT *p*-value | Entropy |
|---|---|---|---|---|---|---|
| 2 | −18,375.86 | 36,791.73 | 36,835.24 | 4,782.16 | <0.0001 | 0.89 |
| 3 | −17,636.34 | 35,320.34 | 35,372.56 | 1,430.74 | 0.0001 | 0.89 |
| 4 | −17,009.69 | 34,075.39 | 34,225.26 | 1,211.75 | 0.0001 | 0.88 |
| 5 | −16,557.68 | 33,179.36 | 33,248.99 | 874.30 | 0.0011 | 0.87 |
| **6** | **−16,314.60** | **32,701.20** | **32,779.52** | **470.18** | **0.0443** | **0.85** |
| 7 | −16,095.16 | 32,270.32 | 32,357.35 | 424.45 | 0.1060 | 0.87 |

LL, log-likelihood; AIC, Aikaike Information Criterion; Adj BIC, sample size adjusted Bayesian Information Criterion; LMR-LRT, adjusted Lo–Mendell– Rubin likelihood ratio test; LRT *p*-value, significance (in *p*-value) of the adjusted LMR-LRT; Entropy (0 to 1), measures the degree to which latent classes are clearly distinguishable from 1 another. Details of the best-fitting model are indicated in bold.

**Table 2**

Results Univariate Regression Analyses: Effect Sizes (in Odds Ratio) of Risk and Protective Variables Regarding Alcohol Use on Latent Trajectory Membership

| | High (class 1, 2) vs. low drinking (class 5, 6) | | High (class 1, 2) vs. moderate drinking (class 3, 4) | | Persistent high drinking (class 1) vs. high but later reduced drinking (class 2) | | Peak and decline (class 3, 5) vs. gradual increase in drinking (class 4, 6) | |
|---|---|---|---|---|---|---|---|---|
| **Risk and protective variables** | **OR** | ***p*** | **OR** | ***p*** | **OR** | ***p*** | **OR** | ***p*** |
| Genetic risk alcohol use disorder | 1.44 | 0.002 | 0.94 | 0.439 | 1.16 | 0.173 | 1.45 | 0.001* |
| Genetic risk externalizing disorder | 2.11 | <0.001* | 1.39 | <0.001* | 1.37 | 0.003 | 1.41 | <0.001* |
| Sensation seeking | 1.39 | <0.001* | 1.42 | <0.001* | 1.39 | 0.009 | 1.00 | 0.993 |
| Early trauma | 1.15 | 0.595 | 1.48 | 0.180 | 0.94 | 0.871 | 0.67 | 0.198 |
| Early peer deviance | 2.17 | <0.001* | 1.53 | <0.001* | 1.02 | 0.895 | 2.00 | <0.001* |
| Late peer deviance | 4.30 | <0.001* | 2.19 | <0.001* | 1.63 | 0.002 | 2.10 | <0.001* |
| ADHD | 1.67 | <0.001* | 1.27 | 0.002 | 1.12 | 0.276 | 1.35 | <0.001* |
| Low school performance | 1.34 | <0.001* | 1.41 | <0.001* | 1.28 | 0.015 | 0.94 | 0.443 |
| Neuroticism | 1.20 | 0.014 | 1.17 | 0.041 | 1.35 | 0.004 | 1.01 | 0.940 |
| Social church activities | 0.64 | <0.001* | 0.87 | 0.107 | 0.66 | 0.001* | 0.72 | <0.001* |
| Social regular activities | 0.72 | <0.001* | 0.87 | 0.060 | 0.72 | 0.003 | 0.83 | 0.023 |
| Negative parental attitude | 1.41 | <0.001* | 1.12 | 0.134 | 0.99 | 0.904 | 1.30 | <0.001* |
| Parental monitoring | 0.53 | <0.001* | 0.74 | <0.001* | 0.95 | 0.668 | 0.69 | <0.001* |

OR, odds ratio; ADHD, attention deficit hyperactivity disorder.

Analyses were weighted for the individual probability of class membership. Several contrasts were tested: the effects of the variables on being a member of (i) trajectories characterized by high compared with low level alcohol use, (ii) trajectories characterized by high compared with moderate level alcohol use, (iii) a trajectory characterized by persistence of high alcohol use compared with declining use over time, and (iv) trajectories characterized by a peak and subsequent decline versus a gradual increase in alcohol use.

*Significant following Bonferroni correction (52 tests = 13 variables × 4 contrasts).

**Table 3**

Results of Multivariate Regression Analyses: Effect Sizes (in Odds Ratio) of Risk and Protective Variables Regarding Alcohol Use on Latent Trajectory Membership

| | High (class 1,2) vs. low drinking (class 5,6) | | High (class 1,2) vs. moderate drinking (class 3,4) | | Persistent high drinking (class 1) vs. high but later reduced drinking (class 2) | | Peak and decline (class 3,5) vs. gradual increase in drinking (class 4, 6) | |
|---|---|---|---|---|---|---|---|---|
| **Risk and protective variables** | **OR** | ***p*** | **OR** | ***p*** | **OR** | ***p*** | **OR** | ***p*** |
| Genetic risk alcohol use disorder | 1.41 | 0.007* | 0.98 | 0.807 | 1.11 | 0.372 | 1.45 | 0.003* |
| Genetic risk externalizing disorder | 1.33 | 0.118 | 1.06 | 0.680 | 1.27 | 0.191 | 1.15 | 0.444 |
| Sensation seeking | 0.83 | 0.220 | 1.35 | 0.043 | 1.26 | 0.218 | 0.72 | 0.052 |
| Early trauma | 0.53 | 0.115 | 0.71 | 0.418 | 0.75 | 0.568 | 0.38 | 0.039 |
| Early peer deviance | 1.30 | 0.074 | 1.58 | 0.002* | 0.82 | 0.263 | 0.92 | 0.619 |
| Late peer deviance | 2.95 | <0.001* | 1.92 | <0.001* | 1.56 | 0.074 | 1.81 | 0.003* |
| ADHD | 1.14 | 0.446 | 0.82 | 1.88 | 0.93 | 0.669 | 1.37 | 0.093 |
| Low school performance | 0.87 | 0.410 | 1.25 | 0.153 | 1.05 | 0.785 | 0.78 | 0.159 |
| Neuroticism | 0.94 | 0.685 | 0.90 | 0.450 | 1.13 | 0.472 | 0.84 | 0.261 |
| Social church activities | 0.73 | 0.044 | 1.05 | 0.778 | 0.85 | 0.390 | 0.78 | 0.164 |
| Social regular activities | 0.99 | 0.962 | 1.00 | 0.965 | 1.04 | 0.855 | 0.93 | 0.707 |
| Negative parental attitude | 1.09 | 0.560 | 1.00 | 0.974 | 0.90 | 0.521 | 1.14 | 0.423 |
| Parental monitoring | 0.71 | 0.049 | 0.89 | 0.439 | 1.10 | 0.619 | 0.80 | 0.228 |

OR, odds ratio; ADHD, attention deficit hyperactivity disorder.

Analyses were weighted for the individual probability of class membership. Several contrasts were tested: the effects of the variables on being a member of (i) trajectories characterized by high compared with low level alcohol use, (ii) trajectories characterized by high compared with moderate level alcohol use, (iii) a trajectory characterized by persistence of high alcohol use compared with declining use over time, and (iv) trajectories characterized by a rapid increase compared with a slow increase toward the maximum level of alcohol use.

*Significant following Bonferroni correction (4 tests = 1 × 4 contrasts).