Developmental Prediction Model for Early Alcohol Initiation in Dutch Adolescents

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ABSTRACT. Objective: Multiple factors predict early alcohol initiation in teenagers. Among these are genetic risk factors, childhood behavioral problems, life events, lifestyle, and family environment. We constructed a developmental prediction model for alcohol initiation below the Dutch legal drinking age (16 years), elaborating on the pathways identified by earlier studies. **Method:** A set of 22 prospectively measured variables, previously associated with alcohol initiation, was examined by path analytic techniques in a sample of 1,804 Dutch adolescents (ages 13–15 years, 56% girls). The predictors included genetic risk for alcohol initiation and behavioral/emotional problems; prenatal and childhood stressors and childhood behavioral/emotional problems; and adolescent behavioral/emotional problems, lifestyle, family functioning, and peer-related factors. **Results:** The model explained 66% of variance in early alcohol initiation. Subjects at higher genetic risk of alcohol initiation who had friends who drank alcohol and who had started smoking at an early age were at increased risk of initiating alcohol use before age 16. Behavioral (externalizing) problems were moderately and indirectly associated with early alcohol initiation, and emotional (internalizing) problems were marginally and indirectly associated with alcohol initiation. **Conclusions:** The Netherlands has relatively lenient alcohol laws. In this permissive environment, early alcohol initiation is explained by alcohol-specific genetic risk, smoking initiation, and peer-related factors, whereas behavioral and emotional problems are only indirectly related to early alcohol initiation. (*J. Stud. Alcohol Drugs, 74,* 59–70, 2013)

EARLY INITIATION OF ALCOHOL USE is associated with numerous adverse outcomes, such as increased risk of adolescent problem drinking, delinquency, risky sexual behavior, academic problems, and adult alcohol dependence (e.g., Donovan and Molina, 2011). The timing of alcohol initiation is associated with multiple factors occurring throughout development that either increase risk of early initiation or protect against it (Kendler et al., 2011b; see also review by Zucker et al., 2008). We aim to determine which factors are the most powerful in predicting whether Dutch adolescents start drinking alcohol before reaching the minimum legal age. The Netherlands has relatively permissive alcohol laws—buying soft alcoholic beverages (beer, wine, and distilled drinks containing under 15% alcohol by volume) is legal from age 16, and to purchase strong alcoholic drinks (distilled drinks containing at least 15% alcohol by volume) the buyer must be 18 years old (Ministry of Health Welfare and Sport, 2009). These laws are not always strictly enforced, and buying alcoholic beverages is often possible for those younger than 16 years (van Hoof et al., 2011). Moreover, parental attitudes toward early drinking are lenient; more than 50% of teenagers younger than 16 years are allowed to drink alcohol at home (van Laar et al., 2010).

Below, we first review the literature on risk and protective factors, ranging from prenatal exposure to adolescence, that have been associated with timing of alcohol initiation. A large set of risk and protective factors was assessed in Dutch adolescents (1,007 girls and 797 boys), and these factors are examined simultaneously in a prediction model for alcohol initiation before age 16.

Sex

Donovan (2004) concluded in a review on predictors of alcohol initiation that there was no convincing evidence that sex influences timing of alcohol initiation. This finding has since been corroborated in several American samples (Donovan and Molina, 2011; Goldschmidt et al., 2012; Malone et al., 2012). However, in an American sample, male sex was associated with earlier alcohol initiation, and in Dutch adolescents, more boys than girls had started drinking before age 16 (Geels et al., 2012; Poelen et al., 2005; Sartor et al., 2007). In contrast, in an Australian sample and in the Finnish Twin studies, girls started drinking earlier than boys (Heath and Martin, 1988; Rose et al., 2001; Viken et al., 1999).

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Genetic risk for alcohol use and comorbid disorders

Alcohol use by family members predicts adolescent early alcohol initiation and use. Early regular drinking of the co-twin is more strongly related to adolescent alcohol use in monozygotic twin pairs than in dizygotic twin pairs (Poelen et al., 2007). These results indicate that the predictive value of familial alcohol initiation/use is partly attributable to shared genes, in addition to shared family environment. The timing of parental alcohol initiation also predicts when children will start drinking alcohol (Donovan, 2004). Hopfer (2003) reviewed twin studies on alcohol initiation and reported genetic influences between 14% and 40%. In Dutch twins, the genetic influence on alcohol initiation was 31% (Geels et al., 2012), and in an Australian sample, 36% (Sartor et al., 2009). Alcohol initiation is associated with behavioral (externalizing) problems, and this comorbidity likely results from a common, highly heritable vulnerability to disinhibitory behavior (Hicks et al., 2011; Kendler et al., 2003; review by Zucker et al., 2008).

Prenatal exposure and childhood stressors

Prenatal alcohol exposure has been associated with childhood externalizing problems, adolescent conduct-disorder symptoms, and alcohol disorders in Australian and American studies (Alati et al., 2006; D'Onofrio et al., 2007; Disney et al., 2008). Maternal prenatal smoking has been related to adolescent and adult behavioral (externalizing) problems and early alcohol initiation (Cornelius and Day, 2009; Goldschmidt et al., 2012; Knopik, 2009; Paradis et al., 2011). These associations are commonly observed, but to what extent they reflect causal, teratogenic effects of prenatal exposure or confounding effects of genetic or shared environmental factors is unclear (Thapar and Rutter, 2009). Childhood stressors such as parental divorce are related to early alcohol initiation (McCarty et al., 2012; Sartor et al., 2007). There is some evidence that low socioeconomic status (SES) is related to early alcohol use (review by Wiles et al., 2007; Zucker et al., 2008), although Donovan (2004) concluded that childhood SES does not affect early alcohol initiation.

Childhood behavioral and emotional problems

Childhood behavioral problems (e.g., impulsivity, hyperactivity, and aggressiveness) are strongly related to alcohol initiation (reviews by Donovan, 2004; Zucker et al., 2008). In samples from the United States, Canada, Finland, and New Zealand, conduct disorder, attention-deficit/hyperactivity disorder (ADHD), and delinquent behavior as early as at ages 3–5 years have been related to early alcohol initiation (Mayzer et al., 2009; Sartor et al., 2007). Nonsignificant associations between childhood ADHD and later alcohol initiation/use have also been reported (review by Zucker et al., 2008). The relationship between childhood emotional (internalizing) problems and alcohol initiation is less well established and more ambiguous. Internalizing psychopathology is associated with early alcohol initiation, but some internalizing symptoms, such as withdrawn behavior, have also been found to be protective against alcohol initiation (review by Donovan, 2004; Hussong et al., 2011; review by Zucker et al., 2008).

Adolescent predictors

Behavioral problems during adolescence (e.g., impulsivity, disinhibition, and attention problems) are highly comorbid with alcohol initiation (Anderson and Brown, 2010; Donovan, 2004; Goldschmidt et al., 2012; Iacono et al., 2008). Alcohol initiation is also related to aspects of sensation seeking (e.g., boredom susceptibility; Koopmans et al., 1997a). Kendler et al. (2011b) used a path modeling approach to predict adolescent alcohol use and symptoms of alcohol use disorder in young adult male American twins and observed a strong externalizing pathway. Emotional problems in adolescents, such as depression and anxiety, co-occur with alcohol initiation, although associations are often weaker than with externalizing problems. Moreover, some aspects (e.g., withdrawn behavior) may protect against alcohol initiation (Hussong et al., 2011). Kendler et al. (2011b) similarly observed weak and mixed associations of internalizing symptoms on adolescent alcohol use and symptoms of alcohol use disorder. Early alcohol initiation is related to behavioral and emotional problems, and heavy alcohol use has been associated with lower well-being and decreased life satisfaction in Australian and Finnish adults (Dear et al., 2002; Koivumaa-Honkanen et al., 2012). Therefore, general well-being may protect against early alcohol initiation.

Early alcohol initiation is strongly associated with characteristics of friends and peers. Peer group deviancy/delinquency and peer alcohol use are important predictors of early alcohol initiation (Anderson and Brown, 2010; Donovan and Molina, 2011; Trucco et al., 2011). Another chief predictor of alcohol initiation is the family environment. Positive parental attitudes toward alcohol use and alcohol availability at home predict whether adolescents start drinking early (Donovan and Molina, 2011; Hung et al., 2009). General parenting skills (e.g., less strict, less involved parenting) as well as lower familial support and more family conflict increase risk of early initiation (Donovan and Molina, 2011; Goldschmidt et al., 2012; Hung et al., 2009; Ryan et al., 2010). Living with a single parent or a stepparent also adds to risk of early initiation (review by Donovan, 2004; Donovan and Molina, 2011). In contrast, American and Lithuanian studies show that eating daily dinners with family members and spending time on family activities protect against early alcohol initiation (Fisher et al., 2007; Garmienė et al., 2006). Again, the

extent to which these associations reflect causal mechanisms is unclear.

Lifestyle factors, such as smoking cigarettes, are related to alcohol initiation and early alcohol use (review by Donovan, 2004; Fisher et al., 2007; Koopmans et al., 1997b; MacArthur et al., 2012). Exercise behavior has not been linked specifically to initiation but is protective against adolescent alcohol use (Terry-McElrath et al., 2011). Less religious behavior increases risk of early alcohol initiation in some studies (Donovan and Molina, 2011) but not in others (Koopmans et al., 1999). School-related factors are associated with timing of alcohol initiation as well. Lower expectations for school achievement, negative attitudes toward school, and lower grades are associated with early alcohol initiation (review by Donovan, 2004; Donovan and Molina, 2011). Last, degree of urbanization may be associated with alcohol initiation in that living in a more rural environment has been linked to increased alcohol use in American adolescents (Swaim and Stanley, 2011).

Aim of the present study

A predictive model of risk and protective factors—identified from the literature—for alcohol initiation was developed and tested on data that were prospectively collected in Dutch adolescents. We based our approach on the path model proposed by Kendler et al. (2011b), which predicted adolescent alcohol use and symptoms of alcohol use disorder in a sample of American twins. Data on alcohol initiation that were collected in a population-based sample of Dutch adolescents (1,804 twin pairs) ages 13–15 years from the Netherlands Twin Register were analyzed. A set of 22 risk and protective factors, prospectively collected in this group, were evaluated. These included genetic risk factors, and variables measured in childhood and adolescence. By examining all factors simultaneously, we assessed which factors are associated with early alcohol initiation and whether associations reflected direct or indirect effects.

Method

Sample

Participants were registered with the Netherlands Twin Register at birth. Recruitment for the Netherlands Twin Register started in 1987 at the VU University Amsterdam and is ongoing at present (Boomsma et al., 2006). Survey data are collected longitudinally in young twins, starting with maternal reports on the pregnancy, health, and temperament of the twins during their first 2 years of life. Parental reports on behavioral and emotional problems, health, school performance and SES are collected at ages 3, 5, 7, 10, and 12 years. Data collection and participation rates have been described in Bartels et al. (2007). When twins are 14, 16, and 18 years old, they are invited to complete self-report questionnaires on topics such as health, lifestyle, behavior problems, well-being, and school performance. Descriptions of data collection and response rates can be found in Bartels et al. (2011).

The data included in this study comprise maternal reports on alcohol use and cigarette smoking during pregnancy; maternal reports on childhood behavioral problems, emotional problems, attention problems, and SES; and adolescent self-reports on behavioral and emotional problems, lifestyle (smoking, exercise behavior), family functioning, well-being, amount of time spent with friends, peer alcohol use, urbanization, religiousness, and school performance. Data from the adolescent survey were available for 6,217 twins (individuals) between ages 13 and 15 years, of whom 5,898 had stated whether they had initiated alcohol use (2,637 complete twin pairs). Data on alcohol initiation and all predictor variables were available for 1,804 complete twin pairs. From each twin pair, one member was randomly selected as the index case, and data from his or her co-twin were used to specify the genetic risk variables. Subjects ranged in age from 13 to 15 (1.6% were 13 years old, 65.3% were 14, and 33.1% were 15 years old). Slightly more girls than boys participated (56%).

Measures

Early alcohol initiation was defined as ever having used alcohol (at age 13–15). Response categories were *no, a few times,* and *yes.* The categories *a few times* and *yes* were collapsed, creating a binary variable.

Table 1 shows all predictor variables and their measurement scales.

Genetic risk for alcohol use and co-morbid disorders. Genetic risk for alcohol initiation, internalizing, and externalizing problems were indexed from co-twin data. Internalizing and externalizing problems were assessed with the Youth Self-Report (Achenbach and Rescorla, 2001). The internalizing scale consists of 32 items and the externalizing scale of 30 items. To obtain genetic risk measures for internalizing and externalizing problems, continuous scores were first transformed into *z* scores. Zygosity was used as a weight factor to correct for the difference in genetic similarity between mono- and dizygotic twins (cf. Kendler et al., 2011b). In regression terms, the outcome variable was predicted differentially for mono- and dizygotic twins:

$Y = \beta X$ for monozygotic twins, and $Y = 0.5 \times \beta X$ for dizygotic twins, where X could be externalizing, internalizing, or alcohol initiation.

Prenatal and childhood predictors. Prenatal alcohol and tobacco exposure were obtained shortly after birth of the twins by asking mothers if they had used cigarettes (ranging from *no* to *more than 10 cigarettes per day*) or alcohol (ranging from *no* to *more than one glass per week*) in the

Table 1. Overview of model variables, grouped by developmental timing

Genetic risk for alcohol use and co-morbid disorders	
Genetic risk for alcohol initiation	0 = having a nondrinking MZ co-twin; 1 = having a non-drinking DZ co-twin; 2 = having a drinking DZ co-twin; 3 = having a drinking MZ co-twin
Genetic risk for externalizing	continuous; range: -1.53–6.85, high scores indicating high risk
Genetic risk for internalizing	continuous; range: -1.24–4.72, high scores indicating high risk
Sex	0 = male; 1 = female
Prenatal and childhood predictors	
Smoking during pregnancy	0 = not exposed; 1 = exposed
Alcohol during pregnancy	0 = not exposed; 1 = exposed
Childhood externalizing behavior problems	0 = low; 1 = middle; 2 = high
Childhood internalizing behavior problems	0 = low; 1 = middle; 2 = high
Childhood attention problems	0 = low; 1 = middle; 2 = high
Childhood socioeconomic status	0 = low; 1 = middle; 2 = high
Parental divorce	0 = not divorced; 1 = divorced
Adolescent predictors	
Family functioning	continuous; range: 1.20–4.80, high scores indicating good family functioning[a]
Adolescent externalizing	continuous; range: 0.00–5.80, high scores indicating more externalizing problems[a]
Adolescent internalizing	continuous; range: 0.00–5.40, high scores indicating more internalizing problems[a]
Urbanization	continuous; range: 1–5, high score indicating low urbanization level
Well-being	continuous; range: 1.00–6.30, high scores indicating higher well-being[a]
Socializing with friends	continuous; range: 3–21, high scores indicating more frequent socializing with friends
Regular exercise	0 = don't exercise regularly; 1 = exercise regularly
Peer alcohol use	0 = none of friends drink alcohol; 1 = 1–5 friends drink alcohol; 2 = more than 5 friends drink alcohol
Smoking initiation	0 = not initiated smoking; 1 = initiated smoking
Religiousness	0 = not religious; 1 = religious
Secondary school level	0 = low; 1 = middle; 2 = high

[a]To avoid computational difficulties with model fitting due to large variance differences, all scores on these scales were divided by 10.

first pregnancy trimester, the last trimester, or during the entire pregnancy. Most mothers had not used any alcohol while pregnant (80%; n = 1,440), 4% had used alcohol in the first trimester (n = 72), 6% in the last trimester (n = 105), and 10% throughout the entire pregnancy (n = 187). A total of 81% of mothers had not smoked while pregnant (n = 1,457), 3% had smoked in the first trimester (n = 60), 2% in the last trimester (n = 41), and 14% had smoked during the entire pregnancy (n = 246). The categories of both variables were collapsed to *no* versus *any alcohol use/smoking* because cross-classification with other variables in the model resulted in empty cells.

Childhood externalizing, internalizing, and attention problems were measured with the Child Behavior Checklist (Achenbach, 1992; Achenbach and Rescorla, 2001), completed by mothers when twins were 3, 7, 10, and 12 years old (Bartels et al., 2007). For each of these scales, longitudinal measurements were summarized in a single score, which was based on t scores and represented low, middle, or high probability of externalizing, internalizing, or attention problems. Subjects were classified as scoring high if they had $t \geq 65$ at least once and $t \geq 60$ at every available assessment. Subjects scoring $t \leq 55$ at each available time point were classified as low scorers, and if they scored in between they were in the middle category (cf. Lehn et al., 2007).

Childhood SES was measured longitudinally between ages 3 and 10 years. The most recent SES data available were used. The coding followed that of Statistics Netherlands (Standard Classification of Occupations [SBC], 2001), based on the mental complexity of parental occupation (Lehn et al., 2007). SES had six categories, ranging from unemployed to academic, which were collapsed into three categories (low, middle, and high). Subjects were retrospectively asked about parental divorce in the adolescent self-report survey.

Adolescent predictors. Degree of urbanization of the residential area was a continuous variable, ranging between 1 (*highly urban*) and 5 (*not urban*). Data were based on participants' postal code and obtained from Statistics Netherlands (cf. Willemsen et al., 2005). Secondary school level was measured by asking adolescents which level of secondary school they were in or had last been in (low, middle, high) when completing the questionnaire. In the Dutch education system, there are different levels of secondary school, ranging from lower professional education to pre-university

education, suited to the students' capabilities (National Reference Point, 2009).

Family functioning was measured with the general family functioning subscale of the McMaster Family Assessment Device (De Coole and Jansma, 1983; Epstein et al., 1983). Subjective well-being was indexed with a sumscore of the Satisfaction with Life Scale and the Subjective Well-being Scale (Diener et al., 1985; Lyubomirsky and Lepper, 1999).

Smoking initiation was indexed by asking subjects whether they had ever smoked. Answer categories were *no, a few times,* and *yes.* The latter two categories were collapsed. Religiousness was defined as being religious (yes/no) when completing the survey. Regular exercise was measured by asking subjects if they exercised regularly (yes/no). Subjects were asked about the frequency with which they spent leisure time with friends in their own home, in the homes of friends, and on the street. Answer categories were 1 (*never*), 2 (*once until now*), 3 (*less than once a week*), 4 (*once a week*), 5 (*a few days per week*), 6 (*almost daily*), and 7 (*daily*). Scores on these three items were summed into an overall score for frequency of socializing with friends, ranging from 3 to 21 (cf. van der Aa et al., 2012). Peer alcohol use was measured by asking participants how many of their friends used alcohol. The answer categories were none, one friend, two to five friends, and more than five friends. The two middle categories were infrequently endorsed and were therefore collapsed into *one to five friends.*

Model

A path model was specified in Mplus 5.21 (Muthén and Muthén, 2010) in which variables were grouped in the model according to developmental timing (Table 1). A fully saturated model was specified in which each variable was related to all other variables. Within developmental groups (genetic risk, prenatal, childhood, adolescence), the covariance between each pair of variables was estimated. Between developmental groups, regressions were specified between each pair of variables. The variables in the genetic risk group functioned solely as independent variables, predicting all downstream variables. Alcohol initiation, the final outcome variable, only functioned as a dependent variable. The variables in the intermediate groups (prenatal, childhood, adolescence) had multiple functions in the model. Each functioned as an independent variable, predicting all downstream variables. These intermediate variables also functioned as dependent variables, being predicted by all upstream variables.

The continuous variables (family functioning, internalizing, externalizing, urbanization, well-being, socializing with friends) were predicted with linear regressions. The binary and categorical variables (all prenatal and childhood factors, regular exercise, peer alcohol use, smoking initiation, religiousness, secondary school level) were assumed to reflect an underlying normal distribution. These variables were analyzed with probit regressions and predict probability of the categories of the dependent variable with a linear combination of predictors, multiplied by the cumulative distribution function (Garwood, 1941).

All nonsignificant regression coefficients or covariances were removed (constrained at 0) from the saturated model. Parameter significance was determined by evaluating whether the parameter z value (parameter estimate divided by its standard error) was significant according to the z distribution. Parameters were removed sequentially, starting with those with the smallest z values (cf. Kendler et al., 2011b). While dropping parameters, model fit was evaluated using three statistics: the Tucker–Lewis Index (TLI), comparative fit index (CFI), and root mean square error of approximation (RMSEA). For the CFI and TLI, values greater than .95 indicate good model fit. RMSEA values below .05 reflect good model fit (Schermelleh-Engel et al., 2003; Tucker and Lewis, 1973). A parsimonious model was created by removing nonsignificant parameters until the fit statistics reached these boundaries.

Because the model contained ordinal variables, weighted mean squares estimation with the theta parameterization was used. This parameterization allows estimation of the residual variance of the normally distributed variable assumed to underlie each categorical variable (Muthén and Muthén, 2010).

Results

Sample characteristics

A total of 1,189 (65.9%) adolescents between ages 13 and 15 years stated that they had initiated alcohol use. Table 2 shows the mean and prevalence of all model variables.

The distributions of genetic risk for internalizing and externalizing problems were skewed, with more observations in the lower range of genetic risk. A similar distribution was observed for genetic risk for alcohol initiation. A total of 20% of the subjects had been prenatally exposed to alcohol and 19% to tobacco. Parental divorce was reported by 12% of the subjects. More than half of the subjects had low probability of childhood externalizing problems (55.1%), 41.6% of subjects were classified in the middle category, and 3.3% of the subjects had high probability of childhood externalizing problems. Very similar distributions were observed for childhood internalizing and attention problems (Table 2). Nearly 17% of the subjects had low childhood SES, 44.5% were classified as having intermediate childhood SES, and 38.8% had high childhood SES.

About 42% of adolescents stated that they were religious when completing the survey. Low level of secondary school was reported by 41.9%, intermediate school level by 25.9%, and high school level by 32.2%. The average frequency of socializing with friends was 10.91 (SD = 3.65), and 42.7%

TABLE 2. Mean, standard deviation, and range for continuous model variables and frequency distributions/prevalences of categorical/binary model variables

Variable	*M*	*SD*	Range
Genetic risk for alcohol use and co-morbid disorders			
Genetic risk externalizing problems	-0.03	0.74	-1.53–6.85
Genetic risk internalizing problems	0.00	0.73	-1.24–4.72
		Distrib.	%
Genetic risk alcohol initiation			
	0:	258	14.3
	1:	366	20.3
	2:	740	41.0
	3:	440	24.4
Sex	Girls:	1,007	55.8
		Distrib.	%
Prenatal and childhood predictors			
Childhood externalizing problems	Low	994	55.1
	Middle	750	41.6
	High	60	3.3
Childhood internalizing problems	Low	993	55.0
	Middle	769	42.6
	High	42	2.3
Childhood attention problems	Low	932	51.7
	Middle	832	46.1
	High	40	2.2
Childhood socioeconomic status	Low	301	16.7
	Middle	803	44.5
	High	700	38.8
	Prevalence	%	
Prenatal alcohol exposure	364	20.2	
Prenatal tobacco exposure	347	19.2	
Parental divorce	217	12.0	
Adolescent predictors	*M*	*SD*	Range
Family functioning	3.88	0.51	1.20–4.80
Adolescent externalizing	0.83	0.55	0.00–5.80
Adolescent internalizing	0.85	0.70	0.00–5.40
Urbanization	3.46	1.17	1.00–5.00
Socializing with friends	10.91	3.65	3.00–21.00
Well-being	5.06	0.89	1.00–6.30
		Distrib.	%
Peer alcohol use	None	425	23.6
	1–5 friends	608	33.7
	>5 friends	771	42.7
Secondary school level	Low	756	41.9
	Middle	468	25.9
	High	580	32.2
	Prevalence	%	
Religiousness	762	42.2	
Smoking initiation	376	20.8	
Regular exercise	1,562	86.6	

Note: Distrib. = distribution.

of the subjects had more than five friends who used alcohol. A total of 21% of the subjects had initiated smoking, and 87% exercised regularly (Table 2).

Correlations

Table 3 shows correlations between all predictor variables and alcohol initiation. These correlations show that alcohol initiation was most strongly associated with genetic risk for alcohol initiation, smoking initiation, and peer alcohol use. Moderate positive correlations were observed with prenatal alcohol and tobacco exposure, childhood externalizing behaviors, parental divorce, regular exercise, genetic risk for externalizing, adolescent externalizing, urbanization, and socializing with friends. Alcohol initiation was negatively associated with family functioning, SES, secondary school level, and well-being.

The correlations further show clustering between externalizing and substance use measures. These variables were weakly related to the variables indexing internalizing

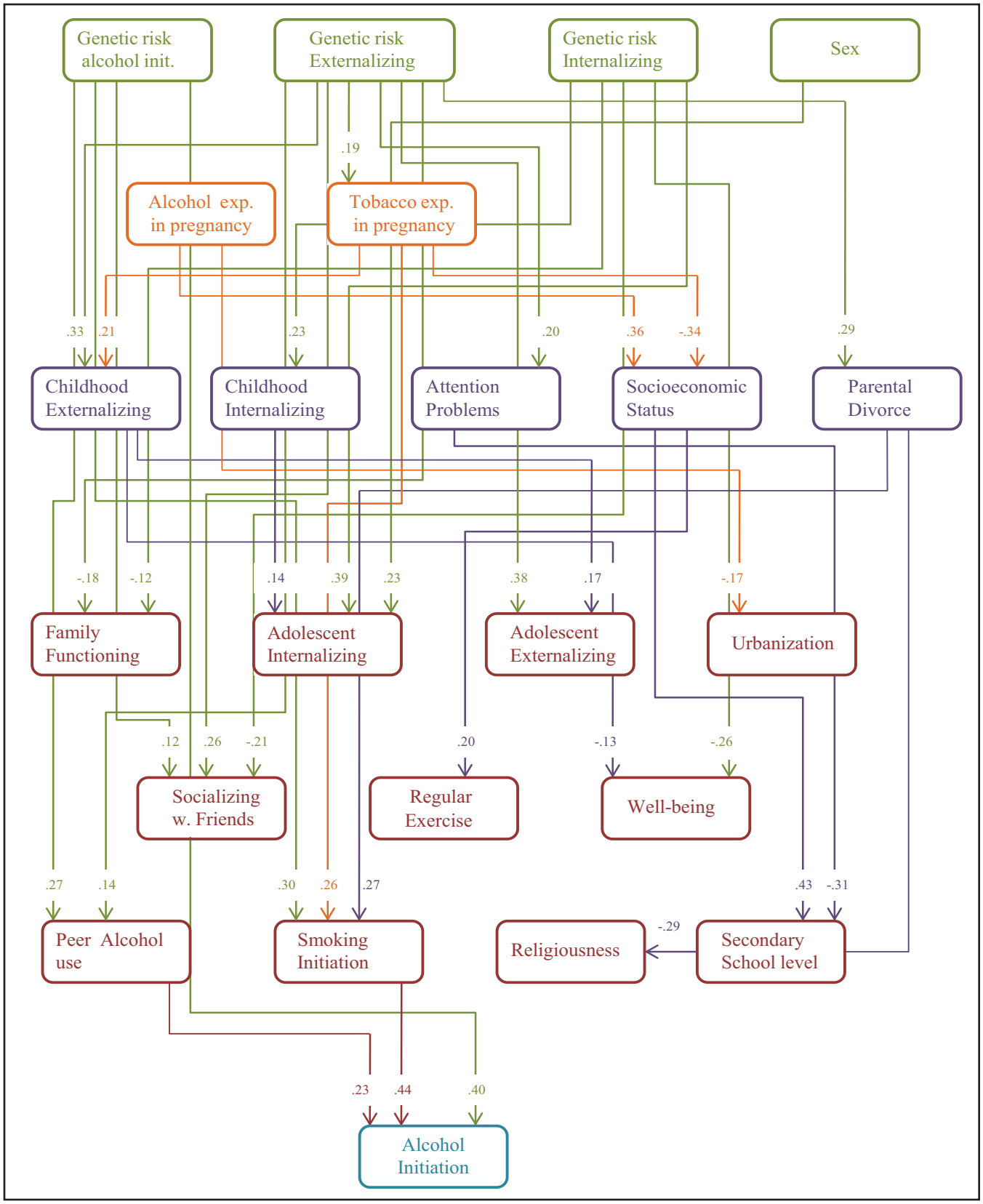

FIGURE 1. Standardized partial regression coefficients estimated under the best fitting model. Each color represents a developmental group of variables (genetic risk, prenatal, childhood, and adolescence). All downstream paths from a particular developmental group are in the corresponding color. Init. = initiation; exp. = exposure; w. = with.

TABLE 3. Observed correlations between all model variables and alcohol initiation

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.
1. Gen. risk alc. init.																						
2. Gen. risk external.	.26†																					
3. Childh. external.	.09†	.20†																				
4. Adol. external.	.16†	.41†	.27†																			
5. Childh. att. prob.	.05	.14†	.68†	.18†																		
6. Social. w/ friends	.20†	.16†	.08†	.23†	.04																	
7. Smoking init.	.31†	.28†	.22†	.39†	.16†	.36†																
8. Peer alc. use	.32†	.19†	.13†	.27†	.06	.29†	.45†															
9. Gen. risk internal.	.03	.44†	.14†	.23†	.10†	-.07†	.05	.06*														
10. Childh. internal.	-.01	.14†	.55†	.14†	.49†	-.01	.09*	-.03	.20†													
11. Adol. internal.	.03	.22†	.11†	.44†	.14†	-.07†	.17†	.07†	.39†	.22†												
12. Parental divorce	.12†	.12†	.07	.13†	.18†	.08*	.22†	.13†	.07	.08	.12†											
13. Family functioning	-.08†	-.18†	-.13†	-.24†	-.06*	-.03	-.23†	-.12†	-.20†	-.09†	-.32†	-.17†										
14. SES	-.03	-.07*	-.14†	-.06*	-.10†	.01	-.08*	-.05	-.04	-.08*	-.07†	-.06	.08†									
15. Regular exercise	.06	-.06	-.03	.00	-.10†	.17†	-.12*	.05	-.11†	-.18†	-.23†	-.10	.05	.16†								
16. Second. school lvl.	-.07†	-.10†	-.21†	-.10†	-.33†	-.15†	-.25†	-.17†	.01	-.09†	-.05	-.15†	.07†	.39†	.16†							
17. Religiousness	.00	-.12†	-.07	-.04	-.05	-.09†	-.12†	-.08*	-.05	-.06	.02	-.20†	.06	-.07†	-.03	.00						
18. Well-being	-.06*	-.16	-.12†	-.24†	-.10†	.05*	-.22†	-.07†	-.26†	-.14†	-.52†	-.16†	.46†	.08†	.16†	.13†	.01					
19. Urbanization	.03	-.09†	.01	-.05*	.00	-.04	-.02	.09†	-.07†	-.03	-.05	-.13†	-.04	-.10†	.06	-.09†	.21†	.00				
20. Sex	.01	-.05	-.02	-.04	-.03	-.03	-.04	.07*	.09†	-.05	.31†	-.03	-.01	-.03	.01	-.02	.09*	-.07*	.01			
21. Alcohol pregnancy	.06	.10†	-.04	.04	-.06	.05	.04	.03	.02	-.08	-.04	-.01	-.02	.31†	.05	.25†	-.15†	.06	-.13†	-.06		
22. Smoking pregnancy	.06	.10†	.18†	.09†	.13†	.11†	.20†	.07	.06	.05	.04	.09	-.02	-.25†	-.09	-.23†	-.19†	-.05	-.04	.00	-.01	
Alcohol initiation	.62†	.26†	.15†	.32†	.01	.28†	.64†	.54†	.03	-.03	.05	.17†	-.13†	-.07*	.10*	-.10†	-.07	-.11†	.07*	-.01	.15†	.18†

Notes: For each pair of variables where both were binary/ordinal, a tetra- or polychoric correlation was estimated. For each pair where both variables were continuous, a Pearson correlation was estimated and for pairs of variables where one was continuous and the other binary/ordinal, a polyserial correlation was estimated. Gen. = genetic; alc. = alcohol; init. = initiation; external. = externalizing; childh. = childhood; adol. = adolescent; att. = attention; prob. = problems; social. = socializing; w/ = with; internal. = internalizing; SES = socioeconomic status; second. = secondary; lvl. = level.
*Correlation is significant at $\alpha = .05$; †correlation is significant at $\alpha = .01$.

psychopathology. Externalizing and internalizing variables were associated with adverse family environment (higher probability of parental divorce, poor family functioning). Higher SES was associated with good family functioning, more regular exercise, and higher secondary school level but lower probability of being religious and of having internalizing and externalizing problems.

Model fitting results

The final, best fitting model had TLI and CFI = .95 and RMSEA = .04 and explained 66% of variance in alcohol initiation.

Direct and indirect associations with alcohol initiation

The standardized partial regression coefficients show that genetic risk for alcohol initiation, smoking initiation, and peer alcohol use directly predicted alcohol initiation (Figure 1). The influence of genetic risk for alcohol initiation was partly direct and partly mediated through smoking initiation, peer alcohol use, and socializing with friends.

The correlations, predicted under the best fitting model, reflect the total association between variables (Table 4). Based on these correlations and the standardized partial regression coefficients (Figure 1), the contribution of a direct path (regression coefficient) between two variables in the model can be separated from the total association between those variables (cf. Kendler et al., 2011b). The predicted correlation between alcohol initiation and genetic risk for alcohol initiation was .61 (Table 4). The direct path between these variables was .40 (Figure 1), indicating that 66% (.40 / .61) of the association between alcohol initiation and genetic risk for alcohol initiation was direct, whereas the remaining 34% was mediated through peer alcohol use, socializing with friends, and smoking initiation (Figure 1). The predicted correlation between alcohol initiation and peer alcohol use was .54 and the regression coefficient was .23. This means that 43% (.23 / .54) of the association between peer alcohol use and alcohol initiation was direct, and that 57% of the association was mediated by other factors. The correlation between smoking initiation and alcohol initiation was .67 and the direct path was .44; therefore, 66% (.44 / .67) of the association between smoking and alcohol initiation was explained by the direct path.

Genetic risk for internalizing and externalizing problems, and sex, were indirectly associated with alcohol initiation. Genetic risk for externalizing problems predicted smoking initiation and peer alcohol use, which were positively related to alcohol initiation. Genetic risk for internalizing problems was negatively related to socializing with friends, which was indirectly related to alcohol initiation. Genetic risk for alcohol initiation, in addition to predicting alcohol initiation, was associated with peer alcohol use, smoking initiation, and

TABLE 4. Predicted correlations between all model variables and alcohol initiaton

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.
1. Gen. risk alc. init.																						
2. Gen. risk external.	.31																					
3. Childh. external.	.08	.26																				
4. Adol. external.	.13	.43	.27																			
5. Childh. att. prob.	.06	.20	.67	.19																		
6. Social. w/ friends	.19	.19	.05	.23	.04																	
7. Smoking init.	.34	.22	.11	.40	.04	.36																
8. Peer alcohol use	.31	.23	.06	.22	.05	.29	.44															
9. Gen. risk internal.	.00	.48	.13	.21	.10	-.09	.06	.07														
10. Childh. internal.	.00	.11	.55	.13	.48	-.02	.01	.02	.23													
11. Adol. internal.	.00	.20	.12	.44	.10	-.04	.03	.03	.42	.23												
12. Parental divorce	.09	.29	.08	.10	.06	.05	.31	.06	.14	.03	.06											
13. Family functioning	-.05	-.23	-.06	-.24	-.05	-.02	-.23	-.05	-.21	-.05	-.32	-.07										
14. SES	-.02	-.07	-.08	-.04	-.01	-.16	-.10	-.02	-.03	-.01	-.01	-.02	.02									
15. Regular exercise	.00	-.01	-.02	-.01	.00	.17	-.02	.00	-.01	.00	-.22	.00	.00	.20								
16. Second. school lvl.	-.03	-.09	-.24	-.07	-.31	-.15	-.23	-.16	-.04	-.15	-.04	-.03	.02	.43	.09							
17. Religiousness	-.02	-.08	-.02	-.02	-.02	-.05	-.09	-.02	-.04	-.01	-.01	-.29	.02	.01	.00	.01						
18. Well-being	-.01	-.16	-.16	-.24	-.11	.02	-.22	-.03	-.27	-.13	-.52	-.04	.46	.02	.16	.04	.01					
19. Urbanization	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	-.06	-.01	-.03	.21	.00				
20. Sex	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.23	.00	.00	.00	.00	.00	.00	.00	.00			
21. Alcohol pregnancy	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.36	.07	.15	.00	.00	-.16	.00		
22. Smoking pregnancy	.06	.19	.25	.11	.04	.04	.29	.04	.09	.02	.04	.06	-.05	-.34	-.07	-.16	-.02	-.06	.00	.00	.00	
Alcohol initiation	.61	.27	.09	.29	.05	.30	.67	.54	.04	.01	.02	.19	-.13	-.05	-.01	-.15	-.05	-.10	.00	.00	.00	.16

Notes: For correlations where one or both variables were continuous, covariance was standardized with estimated variance(s). Gen. = genetic; alc. = alcohol; init. = initiation; external. = externalizing; childh. = childhood; adol. = adolescent; att. = attention; prob. = problems; social. = socializing; w/ = with; internal. = internalizing; SES = socioeconomic status; second. = secondary; lvl. = level.

socializing with friends (Figure 1). Within the genetic risk group, genetic risk for alcohol initiation was associated with genetic risk for externalizing problems and genetic risk for internalizing psychopathology.

None of the childhood factors directly predicted alcohol initiation, but some were associated with adolescent factors, which in turn were associated with alcohol initiation (Figure 1). Maternal prenatal smoking and parental divorce were associated with higher probability of smoking initiation, which in turn was strongly related to increased risk of alcohol initiation.

Peer alcohol use and smoking initiation were directly associated with alcohol initiation. During adolescence, they were associated with internalizing and externalizing problems and socializing with friends. These variables were related to poor family functioning, well-being, and secondary school level, which in turn were indirectly related to increased risk of alcohol initiation (Figure 1).

Discussion

A developmental model was constructed in a Dutch adolescent sample (ages 13–15 years) to predict early initiation of alcohol use. A comprehensive set of risk and protective factors, prospectively measured throughout childhood, was evaluated. Direct and indirect associations with alcohol initiation were examined by simultaneously including all factors in the model.

The best model explained 66% of variance in alcohol initiation. Three predictors were directly related to early alcohol initiation: Adolescents who were at higher alcohol-specific genetic risk, who had friends who used alcohol, and who had started smoking were at increased risk of initiating alcohol use early. Adolescents with increased alcohol-specific genetic risk were likely to spend more time with friends, which in turn was directly related to higher levels of peer alcohol use and smoking initiation. The commonly observed association between early alcohol initiation and externalizing behavior was confirmed (r = .32), but in the prediction model this relationship was mediated through other variables. Considered separately, the influence of alcohol-specific genetic risk, peer characteristics, and adolescent smoking on alcohol initiation has previously been demonstrated (e.g., Anderson et al., 2011; Fisher et al., 2007; Geels et al., 2012). We contribute to the knowledge on determinants of early alcohol initiation showing, in contrast to previous findings, that in a permissive environment such as The Netherlands, alcohol initiation is moderately and indirectly related to behavioral problems and only marginally and indirectly related to emotional (internalizing) problems. These differences are obvious when we relate our findings to those of Kendler et al. (2011b), who constructed a similar model predicting alcohol use (ages 15–17 years) and symptoms of alcohol use disorders in young adult American men. A genetic risk/externalizing pathway, social/familial pathway, and minor internalizing pathway were observed. One may hypothesize that the differ-

ences between these findings reflect an interaction between alcohol predictors and cultural attitudes toward early alcohol use. The Netherlands has permissive views on early alcohol use, whereas in the United States early alcohol use is considered a much greater social and behavioral problem. This is reflected in the minimum legal ages for buying alcohol: age 21 in the United States versus age 16 in The Netherlands (World Health Organization, 2004).

Kendler et al. (2011b) examined alcohol use and symptoms of alcohol use disorder, whereas the outcome in the present study was alcohol initiation. It is possible that the association with behavioral and emotional problems was weaker in this study because these factors may be more strongly related to more severe forms of alcohol use.

An alternative explanation is that the variables that were related to alcohol initiation in fact reflect an underlying risk factor for externalizing behavior. Genetic risk for alcohol initiation may capture not only alcohol-specific genetic risk but also risk for other aspects of externalizing behavior because it was strongly related to socializing with friends, peer alcohol use, and smoking initiation. Moreover, genetic risk was based on co-twin alcohol use, and adolescent alcohol use is influenced by a general externalizing factor (Kendler et al., 2011a). Socializing with friends and peer alcohol use may be expressions of the same underlying trait, because adolescents who are more genetically predisposed to drink alcohol tend to select friends who also drink alcohol (Agrawal et al., 2010; Hill et al., 2008). Similarly, the association between cigarette and alcohol use is likely attributable to underlying risk for externalizing behavior (Little, 2000). Alcohol initiation may be related to less severe forms of externalizing behavior than those measured by the Youth Self-Report (Achenbach and Rescorla, 2001). More serious behavioral problems may be related to more advanced forms of adolescent alcohol use.

The simultaneous modeling of many predictors showed that previously observed associations with alcohol initiation may be mediated through other factors. For example, low school grades have been related to early alcohol initiation (Donovan, 2004), but this study shows that the relationship between secondary school level and alcohol initiation was mediated through peer alcohol use and smoking initiation. Similarly, family functioning was not directly associated with alcohol initiation, as previously observed by Hung et al. (2009) and others, but mediated through smoking initiation. These mediation effects might be explained by interpreting peer alcohol use and smoking initiation as expressions of a general underlying externalizing trait that influences secondary school level, family functioning, and alcohol initiation.

Genetic risk factors were significant predictors of early alcohol initiation. Estimating genetic risk requires data from biological relatives such as twins or parents, which raises questions regarding what the predictive value of the model is if genetic risk data are unavailable. In an additional analysis, the best fitting model was rerun excluding the genetic risk variables. The remaining factors explained 52.6% of variance in alcohol initiation, suggesting that alcohol initiation can still be predicted quite well when genetic risk data are unavailable (results available on request).

Because of the large number of factors included, only main effects were examined. Predictive factors likely do not influence alcohol initiation independently but also interact with each other. For example, Kendler et al. (2011a) observed that genetic risk for adolescent alcohol consumption was stronger in a less restricting environment. The predictors identified in this study can provide a starting point for investigating relevant interaction effects on alcohol initiation.

The family environment was indexed by family functioning, which was not significantly associated with early alcohol initiation in the developmental model, possibly because it did not include parenting strategies, which have been consistently related to early alcohol initiation (e.g., Donovan and Molina, 2011; Goldschmidt et al., 2012; review by Ryan et al., 2010). Similarly, parental alcohol use can provide additional information on alcohol views and availability in the family environment, which are also important predictors of early alcohol initiation (Donovan and Molina, 2011; Hung et al., 2009). Parental alcohol use also provides information on genetic risk for alcohol initiation, which was based solely on co-twin data in this study. This may have led to an underestimation of genetic risk because the co-twins were still in the period of alcohol initiation, and genetic risk may not have been entirely expressed yet. In addition, it cannot be ruled out that the co-twin data contained shared environmental effects as well as genetic risk and that this could explain part of the similarity in alcohol initiation between twins (e.g., Geels et al., 2012).

In summary, in a permissive environment genetic risk for alcohol initiation, peer alcohol use, and smoking initiation were directly associated with early alcohol initiation. Other factors, including behavioral and emotional problems, were only indirectly related to early alcohol initiation.

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