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Trends in adolescent alcohol use: effects of age, sex and cohort on prevalence and heritability

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ABSTRACT

Aims To determine the effect of age, sex and cohort on the prevalence and genetic architecture of adolescent alcohol use (AAU). **Design** Survey study in participants registered with the Netherlands Twin Register. **Setting** Twins from the general population. **Participants** Two cohorts (data collected in 1993 and 2005–08) of twins aged 13–15, 16–17 and 18–21 years. In 1993 and 2005–08 a total of 3269 and 8207 twins, respectively, took part. **Measurements** Survey data on initiation and frequency of alcohol use and quantity of alcohol consumed. **Findings** The prevalence of alcohol initiation increased between 1993 and 2005–08 for both males and females. The largest difference was for girls observed at ages 13–15, where the prevalence increased from 59.5% to 72.4%. We also found increases in prevalence across cohorts for quantity of alcohol consumed and non-significant increases for frequency of alcohol use. From age 16 onwards, boys drank more frequently and larger quantities than girls. Genetic model fitting revealed that the genetic architecture of AAU did not differ between birth cohorts, nor were there differences between boys and girls. Genetic factors explained between 21% and 55% of individual differences in alcohol measures throughout adolescence. Shared environment explained between 17% and 64% of variance in alcohol use, across different age groups and alcohol measures. **Conclusions** In the Netherlands, the prevalence of alcohol initiation, frequency and quantity has increased in adolescents over a 15-year period, but there are no changes in the genetic architecture of adolescent alcohol use.

Keywords Adolescents, alcohol use, cohort study, environmental influence, heritability, prevalence, twins.

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INTRODUCTION

Over the past decades, an increase in alcohol consumption has been reported in adolescents in the Netherlands. Both an increase in quantity consumed and an increase in the number of youngsters who start drinking at earlier ages have been described [1–4]. In 1992, 69% of Dutch adolescents between ages 12 and 18 had initiated alcohol use. In 2007 this had increased to 79%. Over the same period, the percentage of adolescents between ages 12 and 18 years who had consumed alcohol in the past month increased from 45% to 51% [2]. Different European countries have reported minor fluctuations in the prevalence of adolescent alcohol use (AAU) between 1995 and 2007, but overall the prevalence across Europe remained relatively stable [2,5].

Dutch law prohibits selling alcohol to adolescents under age 16 years. Selling mildly alcoholic beverages is legal when buyers are 16 or older, and for strong alcoholic spirits the buyer has to be over 18 [1]. These rules are not always strictly enforced. Moreover, although buying alcohol under age 16 is illegal, Dutch alcohol law does not specify a minimum legal age for alcohol consumption [6]. It is not a taboo for researchers to ask questions about alcohol use in youngsters under age 16.

Studies on the heritability of AAU often obtain heritability estimates as a function of sex and age (e.g. reviews by Hopfer *et al.* [7] and Dick *et al.* [8]). Studies examining

age effects have commonly found that from early adolescence to adulthood, the importance of genetic factors in the aetiology of AAU increases, while the influence of environmental factors that are shared by offspring within a family declines [7,9–12]. Twin studies on sex differences in the genetic architecture of AAU have yielded mixed results. Some observed a higher heritability in boys than in girls [13–15], others did not find sex differences in AAU heritability [16–18], and higher heritability in girls has also been reported [17,19]. Secular changes in the heritability of AAU, and whether these interact with age and gender, have been examined in an early study by Kaprio *et al.* in Finnish twins [20]. Between 1975 and 1981 there were no changes in alcohol prevalence. For the youngest of two age groups (ages 18–24 in 1975) heritability increased in both sexes across the 6-year study period.

In this paper we explore changes in prevalence and heritability of alcohol traits and possible interactions with age and gender in Dutch adolescents aged 13–21 years. Data were collected in longitudinal survey studies of the Netherlands Twin Register (NTR) [21,22]. We first describe to what extent the prevalence of AAU has changed over a period of 15 years by contrasting the alcohol use in a cohort of twins who were adolescents in 1993 to the alcohol use of a cohort of twins who were adolescents between 2005 and 2008. Data on alcohol initiation, frequency and quantity are analysed as a function of sex, age of the twins at data collection (13–15, 16–17 and 18–21 years), and cohort. Secular differences in AAU can be assessed because in both cohorts identical questions about alcohol use were asked.

Secondly, we describe whether there are secular changes in the genetic architecture of AAU. A change in the genetic architecture of AAU, occurring simultaneously with an increase in prevalence, would constitute evidence for moderation of heritable influences by environmental conditions (genotype × environment interaction).

METHODS

Subjects

Participants come from the Netherlands Twin Registry (NTR), established in 1987 at the VU University in Amsterdam. Twins and their family members registered with the NTR are invited approximately every 2 years to participate in longitudinal survey studies [21,22].

Data from two cohorts are analysed. The first cohort participated in the 1993 survey study [22,23]; they were born between 1954 and 1980. At the time of measurement they were on average 17.7 years old [standard deviation (SD) = 4.13, range 12–40]. They were recruited via Dutch City Councils. Recruitment and participation rates have been described in Koopmans *et al.* [23]. For this study subjects between ages 13–21 were selected, resulting in a sample of 3269 twins.

The second twin cohort participated between 2005 and 2008. They were born between 1987 and 1994 and were registered at the NTR at birth by their parents. At the time of assessment they were on average 15.7 years old (SD = 1.51, range 13–21). Recruitment and participation rates have been described in Bartels *et al.* [24]. After selecting twins between ages 13–21, this sample consisted of 8207 twins. IRB approval was obtained for both studies.

Within both cohorts, twins were stratified by age: 13–15, 16–17 and 18–21 years. In all groups, slightly more girls than boys participated (54.2–63.3%). The longitudinal data collection resulted in some overlap (8–17% of individuals) between age groups in the 2005–08 cohort because individuals participated at multiple ages.

Information on zygosity and number of complete/incomplete twin pairs is given in Table 1. In same-sex twin pairs zygosity was determined based on DNA polymorphisms or on parental/self-report items about

Table 1 Sample size and number of complete twin pairs stratified by zygosity in each age group and cohort.

	1993		*2005–08*	
	n	*n complete pairs*	*n*	*n complete pairs*
Age 13–15 years				
MZM	202	98	580	274
DZM	148	70	542	253
MZF	281	138	917	433
DZF	171	83	587	274
DOS	157/155	150	604/674	576
Total	1114		3904	
Age 16–17 years				
MZM	133	65	526	248
DZM	119	56	388	175
MZF	200	99	748	348
DZF	161	80	540	243
DOS	105/105	104	392/464	358
Total	823		3058	
Age 18–21 years				
MZM	230	112	171	77
DZM	191	91	148	61
MZF	325	160	325	147
DZF	207	101	281	127
DOS	189/190	182	138/182	123
Total	1332		1245	

MZM: monozygotic male; DZM: dizygotic male; MZF: monozygotic female; DZF: dizygotic female; DOS: dizygotic opposite sex.

physical resemblance between twins. Zygosity classification based on these items has shown more than 93% agreement with DNA polymorphisms [25,26]. Zygosity was based on DNA polymorphisms for 39.5% of individuals in same-sex twin pairs in the 1993 cohort and 27.6% in the 2005–08 cohort. In this cohort, an additional 11.3% of individuals in same-sex pairs had zygosity typed from blood polymorphisms.

Measures

Three alcohol measures were analysed: initiation and frequency of alcohol use and quantity of alcohol consumed. Subjects were asked if they had ever used alcohol, to which they responded 'no', 'a few times' or 'yes'. The categories 'a few times' and 'yes' were collapsed, creating a binary initiation variable. Additionally, subjects were asked about their frequency of alcohol use over the past year. This question had eight response categories, ranging from 'never' to 'daily'. Because alcohol use obviously increases with age during adolescence, the response distribution of alcohol frequency differed substantially between age groups. Moreover, across all groups the response distributions showed considerable positive skewness (see Table S1; details of supporting information are given at the end of the paper). Therefore, the eight categories of alcohol frequency were combined into three. The most appropriate and meaningful categorization was applied at each age. At ages 13–15, the resulting categories were 'once a year or less', 'several times a year–monthly' and 'several times a month–daily'. At ages 16–17 the categories were 'several times a year or less', 'monthly–several times a month' and 'weekly–daily' and at ages 18–21: 'monthly or less', 'several times a month–weekly' and 'several times a week–daily'. Subjects who had initiated alcohol use were asked about the quantity of alcohol consumed per week, scored in seven categories (ranging from 'less than one glass' to 'more than 20 drinks'). Quantity was also collapsed into three categories because of age differences and the overall positive skewness of the response distributions (see Table S1). Again, the most meaningful categorization was applied to each age group. For ages 13–15, this resulted in categories 'less than 1 glass', '1–2 glasses' and '3 glasses or more'. At ages 16–17, the categories were 'less than 1 glass', '1–5 glasses' and '6 glasses or more' and at ages 18–21: '1–2 glasses or less', '3–10 glasses' and '11 glasses or more'.

Missing data on alcohol initiation ranged from 0% to 2.2% across cohorts, age and sex. For alcohol frequency missing values ranged from 0% to 12.4%. Alcohol quantity was not observed in all cases (61.5–95.1% observed), because this question was not asked in those who had not started drinking alcohol.

Analyses

Prevalence of alcohol use

Prevalences of alcohol initiation and frequencies of alcohol use and alcohol quantity were reported as a function of cohort, age and sex. Data management and preliminary analyses were performed using SPSS version 15.0 [27].

Twin correlations

Structural equation modelling was used to test cohort and sex differences in prevalence and twin correlations of alcohol initiation, frequency and quantity within age groups. Analyses were performed under the assumption that these categorical measures have an underlying continuous, normally distributed liability which can be influenced by genetic and non-genetic factors. Thresholds divide this continuous liability into discrete categories [28]. Sex and cohort effects on the thresholds were tested by estimating separate thresholds and subsequently constraining them to be equal across sex or cohort for all zygosity groups simultaneously.

In each age group, twin resemblances were summarized into tetrachoric (initiation) and polychoric (frequency/quantity) correlations. Cohort effects were tested by constraining all five twin correlations across cohorts simultaneously. Quantitative sex differences in twin correlations were tested by equating the correlations across sex (within mono- and dizygotic same-sex groups). Qualitative sex differences were examined by equating the dizygotic opposite-sex correlation to the dizygotic same-sex correlation (see page 3 of the supporting information for a more detailed description of the model testing procedure). Models were fitted on raw data.

For alcohol quantity, defined as number of glasses per week, the prevalence of any weekly alcohol use was very low at ages 13–15. For alcohol initiation from age 16 onwards, the prevalence approached 100%. For these two phenotypes there was no meaningful population variance to analyse.

Relationship between alcohol initiation and frequency/quantity of alcohol use

In the young adolescents, within each cohort three genetic models were considered to describe the relationship between alcohol initiation and frequency. These analyses addressed the question of how to handle subjects who have not initiated drinking in the analysis of frequency/quantity, because in those subjects alcohol frequency/quantity are unobserved [29,30]. This is necessary in young adolescents (ages 13–15), as not all of them have started using alcohol yet. As the prevalence of alcohol initiation increases with age and approaches

100%, the distinction becomes unnecessary. If alcohol frequency/quantity are determined by a single liability, subjects who have not initiated drinking are excluded from the analysis (because we cannot observe their scores on frequency/quantity) and the liability distribution for frequency/quantity is truncated [29]. If, on the other hand, frequency/quantity are determined by separate liabilities, including those who have not initiated alcohol use in the analysis of frequency/quantity may lead to biased heritability estimates [29]. We compared three liability models: (i) the single liability dimension (SLD) model; in which initiation and frequency/quantity are modelled on a single underlying liability; (ii) the independent liability dimension (ILD), model which assumes that initiation and frequency/quantity have separate, unrelated liabilities and (iii) the combined model (CM), which postulates separate but related liabilities for initiation and frequency/quantity. The combined model allows for people to be non-drinkers either because they have never started or because they have started but are low on frequency/quantity liability. Detailed descriptions of these models can be found in Vink *et al*. [31] and in Koopmans *et al*. [32]. For these models, model fit was determined based on the χ^2 goodness-of-fit statistic and on Akaike's information criterion (AIC) [33].

The combined models were fitted to 4×4 contingency tables of the drinking behaviour of the firstborn twin cross-classified with the co-twin. Thus only data from complete twin pairs were included in these analyses ($n = 1078$ in the 1993 cohort and $n = 3620$ in the 2005–08 cohort). No bias was found when results were compared from univariate genetic models on all raw data and on the data excluding incomplete twin pairs (data available on request from corresponding author). Frequency and quantity of alcohol use consisted previously of three categories. For these analyses, frequency and quantity data were divided into four categories, with subjects who had not initiated alcohol use in the lowest category.

Genetic architecture of alcohol use

The genetic architecture of alcohol initiation was analysed in univariate models. For frequency of alcohol use, the best-fitting liability model was explored in the youngest group, while univariate models were used in the two older groups. The total variance in alcohol initiation, frequency and quantity was partitioned into an additive genetic component (A), shared environmental component (C) and non-shared environmental component (E) [34]. Shared environment represents environmental factors that cause twins to become more similar, whereas non-shared environment refers to environmental influences that make twins less similar [7].

If the pattern of twin correlations indicated qualitative sex differences, genetic models were specified accordingly. At ages 13–15, qualitative sex differences were evaluated for the shared environment based on previous literature [17,18,35]. Specifically, the correlation between shared environments of dizygotic opposite twin pairs was estimated as a free parameter. At ages 18–21 qualitative sex differences were modelled in the genetic component, because that is where the qualitative sex difference was significant. It was not significant for the shared environment (results available on request from corresponding author). The significance of genetic, shared environmental and unique environmental components was examined by constraining them at zero one at a time.

Statistical testing

All statistical testing was performed by comparing nested submodels and evaluating the difference in –2 log-likelihood of the restricted model and the more general model (likelihood-ratio test). If models are nested, this difference is χ^2-distributed. The degrees of freedom equal the difference in estimated parameters [16]. Analyses were performed in Mx [36]. Because of the large number of tests (multiple variables, age groups, and cohorts), all tests throughout the study were evaluated at a 0.01 significance level.

RESULTS

Prevalence of alcohol use

The prevalence of initiation, frequency and quantity of alcohol use was examined as a function of age and sex within the two cohorts (Table 2). At ages 13–15, more boys had started drinking alcohol than girls (for model fitting results see Table S3, model 3). In all age groups, boys outnumbered girls in the highest category of alcohol frequency (for model fitting results see Table S4, model 3). In the older age groups, boys consumed larger quantities than girls (see Table S5). Table 2 shows an increase in alcohol initiation, frequency and quantity between 1993 and 2005–08, across sex, although for alcohol frequency the increases were not significant at alpha = 0.01 in the older age groups.

Twin correlations

Twin correlations for alcohol initiation, frequency and quantity were examined for cohort and sex differences in univariate saturated models, as a function of age (see Tables S2–S5). Significantly lower correlations were observed in the dizygotic opposite-sex twins than in the dizygotic same-sex twins for frequency of alcohol use at

Table 2 Frequencies (percentages) of alcohol initiation, alcohol use and quantity as a function of cohort, age and gender.

	Male		*Female*	
Initiation	1993	2005–2008	1993	2005–08
Age 13–15 years				
Initiated alcohol use	65.5	75.0	59.5	72.4[a,b]
	n = 507	*n* = 1698	*n* = 607	*n* = 2130
Age 16–17 years				
Initiated alcohol use	91.0	94.9	91.6	94.9
	n = 357	*n* = 1306	*n* = 466	*n* = 1752
Age 18–21 years				
Initiated alcohol use	96.7	97.6	94.9	96.2
	n = 610	*n* = 457	*n* = 722	*n* = 788
Frequency				
Age 13–15 years				
Once a year or less	52.9	41.4	63.6	42.7[a,b]
Several times a year—monthly	37.8	37.2	30.2	38.4[a,b]
Several times a month—daily	9.2	21.5	6.2	18.9
	n = 444	*n* = 1666	*n* = 563	*n* = 2093
Age 16–17 years				
Several times a year or less	33.9	19.3	43.1	26.6[b]
Monthly—Several times a month	23.0	24.9	31.3	34.9[b]
Weekly—daily	43.1	55.8	25.5	38.5
	n = 357	*n* = 1287	*n* = 466	*n* = 1722
Age 18–21 years				
Monthly or less	18.8	15.0	40.3	33.8[b]
Several times a month—weekly	34.8	41.2	41.8	46.5[b]
Several times a week—daily	46.5	43.8	17.9	19.7
	n = 607	*n* = 447	*n* = 720	*n* = 775
Quantity				
Age 13–15 years				
Less than 1 glass per week	85.1	77.9	88.3	78.2[a,b]
1–2 glasses per week	9.5	14.3	7.1	13.2[a,b]
3 glasses or more per week	5.4	7.8	4.6	8.6
	n = 349	*n* = 1185	*n* = 367	*n* = 1467
Age 16–17 years				
Less than 1 glass per week	39.2	25.4	60.0	36.8[a,b]
1–5 glasses per week	33.7	40.5	31.6	46.0[a,b]
6 glasses or more per week	27.2	34.0	8.5	17.2
	n = 309	*n* = 1187	*n* = 412	*n* = 1601
Age 18–21 years				
1–2 glasses a week or less	30.7	25.2	62.4	53.5[a,b]
3–10 glasses per week	38.8	45.5	31.1	38.3[a,b]
11 glasses or more per week	30.5	29.3	6.5	8.3
	n = 580	*n* = 437	*n* = 657	*n* = 737

[a]Significant cohort difference within age group; [b]significant sex difference within age group. For frequency and quantity each test was performed on both thresholds simultaneously. All tests were evaluated at $\alpha = 0.01$.

ages 13–15 and for quantity at ages 18–21 (for model fitting results see Tables S4–S5, model 6). No quantitative sex differences in the correlation structure of alcohol use were found, nor were differences between cohorts observed. Twin correlations were therefore estimated on combined cohorts for mono- and dizygotic twins, stratified by age (Table 3).

Correlations suggest that, at ages 13–15, individual differences in alcohol use were explained mainly by shared environmental factors, whereas in later adolescence genetic factors became more important because, with age, the difference between the monozygotic and dizygotic twin correlations increased.

Genetic architecture of alcohol use

In each age group, the genetic architecture of AAU was explored. At ages 13–15 a combined model was specified

Table 3 Tetra- and polychoric twin correlations with 95% confidence intervals for alcohol frequency and quantity in each age group, estimated in best-fitting saturated models.

	Age 13–15 years	*Age 16–17 years*	*Age 18–21 years*
Initiation			
MZ	0.86 (0.81–0.90)	–	–
DZ	0.71 (0.65–0.76)	–	–
Frequency			
MZ	0.83 (0.80–0.86)	0.79 (0.74–0.82)	0.65 (0.58–0.71)
DZ	0.74 (0.68–0.78)	0.59 (0.53–0.64)	0.41 (0.33–0.49)
DOS	0.59 (0.52–0.66)		
Quantity			
MZ	–	0.76 (0.71–0.81)	0.68 (0.60–0.74)
DZ	–	0.51 (0.44–0.57)	0.56 (0.44–0.66)
DOS			0.31 (0.17–0.44)

MZ: monozygotic twins; DZ: dizygotic twins; DOS: dizygotic opposite sex twins.

Table 4 Estimates of genetic and environmental variance components with 95% confidence intervals for initiation, frequency and quantity of alcohol use in each age group.

	A	*C*	*E*
Initiation			
Age 13–15 years	0.31 (0.17–0.45)	0.55 (0.43–0.67)	0.14 (0.10–0.19)
Frequency			
Age 13–15 years	0.21 (0.03–0.42)	0.64 (0.44–0.79)	0.15 (0.11–0.22)
Age 16–17 years	0.42 (0.29–0.54)	0.36 (0.25–0.47)	0.22 (0.19–0.26)
Age 18–21 years	0.47 (0.27–0.67)	0.17 (0.00–0.34)	0.36 (0.30–0.42)
Quantity			
Age 16–17 years	0.55 (0.42–0.69)	0.22 (0.10–0.34)	0.23 (0.19–0.27)
Age 18–21 years	0.36 (0.20–0.56)	0.35 (0.17–0.48)	0.29 (0.24–0.36)

A: genetic factors; C: shared environment; E: non-shared environment. For initiation, variance components were estimated in univariate models. Variance components of frequency were estimated under a combined model at ages 13–15 and variance components of frequency and quantity were estimated under single liability models at ages 16–17 and 18–21.

for alcohol initiation and frequency because a combined model fitted the data best (see Table S6 for model fitting results). Based on the results described above (and in Tables S3 and S4), separate prevalences (thresholds) were estimated for each cohort and gender. Variance components were constrained to be equal across gender and cohorts (because no sex/cohort differences in correlation structure were observed). For frequency, the correlation of the shared environmental component between twins in the DOS group was estimated as a free parameter (based on different DZ/DOS correlation in Table 3). This correlation was 0.75 (0.51–0.94).

Table 4 shows that at ages 13–15 alcohol initiation and frequency were influenced mainly by shared environment (55%, 64%), while genetic influences were less important (31%, 21%).

At ages 16–17 a single liability (SLD) model was fitted for alcohol frequency and quantity, because at this age most subjects had initiated alcohol use, making a bivariate model (CM/ILD) unnecessary. Individual differences in alcohol frequency and quantity were explained by genetic factors (42%, 55%) and shared environment (36%, 22%).

At ages 18–21, alcohol frequency and quantity were also analysed under the single liability model. Table 4 shows that genetic factors explained 47% and 36% of individual differences in alcohol frequency and quantity, respectively. Shared environment explained 17–35% of the variance in frequency and quantity. For quantity, the genetic correlation in DOS twin pairs was freely estimated, at 0.00 (0.00–0.48).

The picture that emerges from Table 4 is that, generally, models including a genetic, shared environmental and non-shared environmental factor best explained individual differences in AAU (see also Tables S7–S10). Alcohol initiation at ages 13–15 was explained mainly by shared environmental factors. For alcohol frequency, genetic influences increased between ages 13–15 and the

older two age groups, while shared environmental influences decreased. For alcohol quantity, only analysed at ages 16–17 and 18–21, this pattern was not observed.

DISCUSSION

We report a comparison of adolescent alcohol use (AAU), assessed identically in two cohorts of young twins, in 1993 and 2005–08. The prevalence and genetic architecture of alcohol initiation, frequency and quantity were compared across cohort and sex, as a function of age. Over a 15-year period an increase in AAU was observed. A larger number of young adolescents initiated drinking in 2005–08 than in 1993. In the more recent cohort they also drank larger quantities. Frequency of alcohol use also increased across cohorts, but this increase was non-significant.

The increase in prevalence led to the question whether the genetic architecture of AAU differed as a function of environmental exposure. Changes in social environment with respect to AAU can moderate the genetic influence on drinking behaviour [genotype × environment (G×E) interaction [37]]. This has been observed for several environmental factors, such as peer substance use [38,39], religiosity [40,41], socio-regional factors [12,42] and parental monitoring [43], suggesting that in an environment where alcohol is more readily available to adolescents their alcohol use is more heritable.

In the current study no specific environmental variable was tested, but instead changes in adolescent drinking patterns (prevalence) were used as proxy for environmental changes. An increased prevalence of AAU was seen for the period under study; however, no change in the genetic architecture of AAU was observed. This finding is analogous to what has been observed for human height, for example, which has increased substantially over the past 150 years, due to improved environmental circumstances [44,45]. The heritability of height, however, has not changed over this time-period [45].

Several circumstances possibly led to the increase in AAU over the 15 years under study. Teenagers have more money to spend and more adolescents in high school worked (35–58% increase) [46,47]. Also, the variety of pre-mixed alcoholic drinks offered by stores has increased. These drinks are especially popular among teenagers [48]. These factors have led to a widespread availability of alcohol, which can be compared to the universal improvement in environmental circumstances leading to increases in height. Analogous to the human height example, these environmental changes seem to affect different genotypes in a similar manner: heritability did not change (no genotype × cohort interaction) with an increase in the prevalence of alcohol use. This observation may imply that as 'interventions' that modify drinking behaviour towards larger consumption do not depend on genotype, the reverse is also true. If a reduction in alcohol use would be a desirable target, intervention could be equally effective for different genotypes.

Within cohorts, sex effects on prevalence and genetic architecture were also explored. At ages 13–15, more boys than girls had started to drink alcohol and drank more frequently. From age 16 onwards, boys drank larger quantities. These findings agree with the 2009 report of the Netherlands Institute of Mental Health and Addiction [2]. No quantitative sex differences in the genetic architecture of AAU were observed. Previous results on sex differences in the genetic architecture of AAU are conflicting: some studies found a higher heritability of alcohol use in boys [13–15] while others did not [16–18,49], and higher heritability in girls has also been reported [17,19]. We found evidence of qualitative sex differences at ages 13–15 and 18–21, i.e. genetic or shared environmental factors that influence alcohol frequency and quantity differed across sex. This has been observed before in Dutch twins (aged 15–24) and in large studies in adolescent Finnish twins [17,18,35].

The liability structure of alcohol use at ages 13–15 was best described by separate but related liabilities for alcohol initiation and frequency. AAU liability structure was examined previously by Fowler *et al.* [50], Koopmans [30] and Heath *et al.* [29], who observed the same structure for alcohol initiation and frequency/quantity [29,30,50]. From age 16 onwards the prevalence of initiation approached 100%, so there were no differences between subjects in alcohol exposure.

Genetic modelling suggested that, within cohorts, the heritability of alcohol frequency increased throughout adolescence while the influence of shared environment declined, in line with what is commonly found in adolescents [7,9–12]. The increase was most apparent when comparing ages 13–15 and 16–17.

Most importantly, in recent years adolescents in the Netherlands consumed more alcohol, drank more frequently and started drinking at a younger age than in the early 1990s, but the relative contributions of genes and environment to individual differences in AAU have not changed across this period.

Declarations of interest

None.

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References

1. Ministry of Health Welfare & Sport. *Dutch Alcohol Policy*. 2009. Available at: http://english.minvws.nl/en/themes/alcohol/default.asp (accessed 16 August 2010; archived by WebCite ® at http://www.webcitation.org/5y5EYGKvv).
2. Trimbos Institute (the Netherlands Institute of Mental Health and Addiction) *National Drug Monitor 2009*. 2010. Available at: http://www.rijksoverheid.nl/documenten-en-publicaties/rapporten/2010/05/27/nationale-drugmonitor-2009.html (accessed 20 January 2011; archived by WebCite® at http://www.webcitation.org/5vsO0yYfu).
3. Poelen E. A. P., Scholte R. H. J., Engels R. C. M. E., Boomsma D. I., Willemsen G. Prevalence and trends of alcohol use and misuse among adolescents and young adults in the Netherlands from 1993 to 2000. *Drug Alcohol Depend* 2005; **79**: 413–21.
4. Statistics Netherlands *Jeugd 2003, feiten en cijfers* [Youth 2003, facts and figures]. 2003. Available at: http://www.cbs.nl/NR/ rdonlyres/9CF19548-A0B9-42BF-9D67 15C6C903AFE6/0/2003g87p051art. pdf (accessed 5 April 2011; archived by Webcite® at http://www.webcitation.org/5xiZU2wJw).
5. European School Survey Project on Alcohol and other Drugs *The 2007 ESPAD Report*. 2009. Available at: http://www.espad.org/documents/Espad/ESPAD_reports/2007/SUMMARY - The_2007_ESPAD_Report.pdf (accessed 5 April 2011; archived by Webcite® at http://www.webcitation.org/5xiix5ST7).
6. Dutch Centre for Crime Prevention & Safety. Alcohol law. 2010. Available at: http://www.hetccv.nl/ dossiers/Drank-+en+Horecawet/index (accessed 12 September 2010; archived by Webcite® at http://www.webcitation.org/5uqZDRhic).
7. Hopfer C. J., Crowley T. J., Hewitt J. K. Review of twin and adoption studies of adolescent substance use. *J Am Acad Child Adolesc Psychiatry* 2003; **42**: 710–9.
8. Dick D. M., Prescott C. A., Mcgue M. The genetics of substance use and substance use disorders. In: Kim Y.-K., editor. *Handbook of Behavior Genetics*, 1st edn. New York, NY: Springer Science + Business Media, LLC; 2009, p. 433–53.
9. Bergen S. E., Gardner C. O., Kendler K. S. Age-related changes in heritability of behavioral phenotypes over adolescence and young adulthood: a meta-analysis. *Twin Res Hum Genet* 2007; **10**: 423–33.
10. Dick D. M., Pagan J. L., Viken R., Purcell S., Kaprio J., Pulkkinen L. *et al*. Changing environmental influences on substance use across development. *Twin Res Hum Genet* 2007; **10**: 315–26.
11. Kendler K. S., Schmitt E., Aggen S. H., Prescott C. A. Genetic and environmental influences on alcohol, caffeine, cannabis, and nicotine use from early adolescence to middle adulthood. *Arch Gen Psychiatry* 2008; **65**: 674–82.
12. Rose R. J., Dick D. M., Viken R. J., Kaprio J. Gene–environment interaction in patterns of adolescent drinking: regional residency moderates longitudinal influences on alcohol use. *Alcohol Clin Exp Res* 2001; **25**: 637–43.
13. Han C., Mcgue M. K., Iacono W. G. Lifetime tobacco, alcohol and other substance use in adolescent Minnesota twins: univariate and multivariate behavioral genetic analyses. *Addiction* 1999; **94**: 981–93.
14. Hopper J. L., White V. M., Macaskill G. T., Hill D. J., Clifford C. A. Alcohol-use, smoking-habits and the Junior Eysenck Personality Questionnaire in adolescent Australian twins. *Acta Genet Med Gemellol (Roma)* 1992; **41**: 311–24.
15. Mcgue M., Iacono W. G., Legrand L. N., Elkins I. Origins and consequences of age at first drink. II. Familial risk and heritability. *Alcohol Clin Exp Res* 2001; **25**: 1166–73.
16. Rhee S. H., Hewitt J. K., Young S. E., Corley R. P., Crowley T. J., Stallings M. C. Genetic and environmental influences on substance initiation, use, and problem use in adolescents. *Arch Gen Psychiatry* 2003; **60**: 1256–64.
17. Rose R. J., Dick D. M., Viken R. J., Pulkkinen L., Kaprio J. Drinking or abstaining at age 14? A genetic epidemiological study. *Alcohol Clin Exp Res* 2001; **25**: 1594–604.
18. Viken R. J., Kaprio J., Koskenvuo M., Rose R. J. Longitudinal analyses of the determinants of drinking and of drinking to intoxication in adolescent twins. *Behav Genet* 1999; **29**: 455–61.
19. Heath A. C., Martin N. G. Teenage alcohol-use in the Australian twin register—genetic and social determinants of starting to drink. *Alcohol Clin Exp Res* 1988; **12**: 735–41.
20. Kaprio J., Rose R. J., Romanov K., Koskenvuo M. Genetic and environmental determinants of use and abuse of alcohol: the Finnish Twin Cohort studies. *Alcohol Alcohol Suppl* 1991; **1**: 131–6.
21. Bartels M., van Beijsterveldtl C. E. M., Derks E. M., Stroet T. M., Polderman T. J. C., Hudziak J. J. *et al*. Young Netherlands Twin Register (Y-NTR): a longitudinal multiple informant study of problem behavior. *Twin Res Hum Genet* 2007; **10**: 3–11.
22. Boomsma D. I., de Geus E. J. C., Vink J. M., Stubbe J. H., Distel M. A., Hottenga J. J. *et al*. Netherlands Twin Register: from twins to twin families. *Twin Res Hum Genet* 2006; **9**: 849–57.
23. Koopmans J. R., van Doornen L. J. P., Boomsma D. I. Smoking and sports participation. In: Goldbourt U., DeFaire U., Berg K., editors. *Genetic Factors in Coronary Heart Disease*. Dordrecht, the Netherlands: Kluwer; 1994, p. 217–35.
24. Bartels M., van der Aa N., van Beijsterveldt C. E. M., Middeldorp C. M., Boomsma D. I. Adolescent self-report of emotional and behavioral problems; interactions of genetic factors with sex and age. *J Can Acad Child Adolesc Psychiatry* 2011; **20**: 35–52.
25. Rietveld M. J., van Der Valk J. C., Bongers I. L., Stroet T. M., Slagboom P. E., Boomsma D. I. Zygosity diagnosis in young twins by parental report. *Twin Res* 2000; **3**: 134–41.
26. Willemsen G., Posthuma D., Boomsma D. I. Environmental factors determine where the Dutch live: results from the Netherlands Twin Register. *Twin Res Hum Genet* 2005; **8**: 312–7.
27. SPSS, Inc. *SPSS for Windows [Computer Program]. Version Release 15.0*. Chicago: SPSS, Inc.; 2006.

28. Falconer D. S., Mackay T. F. C. *Quantitative Genetics*. Essex: Longman Group Ltd; 1996.
29. Heath A. C., Meyer J., Eaves L. J., Martin N. G. The inheritance of alcohol-consumption patterns in a general-population twin sample. 1. Multidimensional-scaling of quantity frequency data. *J Stud Alcohol* 1991; **52**: 345–52.
30. Koopmans J. R. The genetics of health-related behaviors. PhD thesis. Amsterdam: VU University; 1997.
31. Vink J., Willemsen G., Boomsma D. Heritability of smoking initiation and nicotine dependence. *Behav Genet* 2005; **35**: 397–406.
32. Koopmans J. R., Slutske W. S., Heath A. C., Neale M. C., Boomsma D. I. The genetics of smoking initiation and quantity smoked in Dutch adolescent and young adult twins. *Behav Genet* 1999; **29**: 383–93.
33. Akaike H. Factor-analysis and AIC. *Psychometrika* 1987; **52**: 317–32.
34. Neale M. C., Cardon L. *Methodology for Genetic Studies of Twins and Families*. Dordrecht, the Netherlands: Kluwer Academic Publishers B.V.; 1992.
35. Koopmans J. R., Boomsma D. I. Familial resemblances in alcohol use: genetic or cultural transmission? *J Stud Alcohol* 1996; **57**: 19–28.
36. Neale M. C., Boker S. M., Xie G., Maes H. H. *Mx: Statistical Modeling*, 7th edn. VCU Box 900126. Richmond, VA 23298: Department of Psychiatry; 2006.
37. Boomsma D. I., Martin N. G. Gene–environment interactions. In: D'haenen H., den Boer J. A., Willner P., editors. *Biological Psychiatry*, Chichester: John Wiley & Sons; 2002, p. 181–7.
38. Agrawal A., Balasubramanian S., Smith E. K., Madden P. A. F., Bucholz K. K., Heath A. C. *et al*. Peer substance involvement modifies genetic influences on regular substance involvement in young women. *Addiction* 2010; **105**: 1844–53.
39. Guo G., Elder G. H., Cai T. J., Hamilton N. Gene–environment interactions: peers' alcohol use moderates genetic contribution to adolescent drinking behavior. *Soc Sci Res* 2009; **38**: 213–24.
40. Button T. M. M., Hewitt J. K., Rhee S. H., Corley R. P., Stallings M. C. The moderating effect of religiosity on the genetic variance of problem alcohol use. *Alcohol Clin Exp Res* 2010; **34**: 1619–24.
41. Koopmans J. R., Slutske W. S., van Baal G. C. M., Boomsma D. I. The influence of religion on alcohol use initiation: evidence for genotype × environment interaction. *Behav Genet* 1999; **29**: 445–53.
42. Legrand L. N., Keyes M., McGue M., Iacono W. G., Krueger R. F. Rural environments reduce the genetic influence on adolescent substance use and rule-breaking behavior. *Psychol Med* 2008; **38**: 1341–50.
43. Dick D. M., Viken R., Purcell S., Kaprio J., Pulkkinen L., Rose R. J. Parental monitoring moderates the importance of genetic and environmental influences on adolescent smoking. *J Abnorm Psychol* 2007; **116**: 213–8.
44. Mcevoy B. P., Visscher P. M. Genetics of human height. *Econ Hum Biol* 2009; **7**: 294–306.
45. Silventoinen K. Determinants of variation in adult body height. *J Biosoc Sci* 2003; **35**: 263–85.
46. National Institute for Family Finance Information *Nibud Scholierenonderzoek 2008–2009* [Nibud Student Survey 2008–2009]. 2009. Available at: http://www.nibud.nl/fileadmin/user_upload/Documenten/PDF/ onderzoeken/NSO_2008-2009.pdf (accessed 14 July 2011; archived by WebCite ® at http://www.webcitation.org/60AeHQ6c4).
47. Boelens A., Sinkeldam I. (Statistics Netherlands). *Werk om te leren—bijbaantjes vooral op lager beroepsniveau* [Work to learn—additional jobs mainly at lower professional education level]. 2000. Available at: http://www.cbs.nl/NR/rdonlyres/D93DBE53-71E1-45BA-B2AA-2E9D66D93A96/0/index1085.pdf (accessed 14 July 2011; archived by WebCite ® at http://www.webcitation.org/60Abf2C0G).
48. Dutch Institute for Alcohol Policy (STAP) *Press Release*. 2008 Available at: http://www.stap.nl/nl/nieuws/persberichten.html/3490/744/aanbod-supermarkt-stimuleert-alcoholmisbruik#p3490 (accessed 14 July 2011; archived by WebCite ® at http://www.webcitation.org/60AYuM6Fc).
49. Maes H. H., Woodard C. E., Murrelle L., Meyer J. M., Silberg J. L., Hewitt J. K. *et al*. Tobacco, alcohol and drug use in eight- to sixteen-year-old twins: the Virginia Twin Study of Adolescent Behavioral Development. *J Stud Alcohol* 1999; **60**: 293–305.
50. Fowler T., Lifford K., Shelton K., Rice F., Thapar A., Neale M. C. *et al*. Exploring the relationship between genetic and environmental influences on initiation and progression of substance use. *Addiction* 2007; **102**: 413–22.

Supporting information

Additional Supporting Information may be found in the online version of this article:

Table S1 Frequencies (percentages) of alcohol frequency and quantity in the original categories for each cohort, age group and gender.

Table S2 Tetra- and polychoric twin correlations with 95% confidence intervals for alcohol initiation, frequency and quantity in each age group and cohort, estimated in full saturated models.

Table S3 Initiation of alcohol use: test for cohort and sex differences in thresholds and correlation structure using univariate saturated models.

Table S4 Frequency of alcohol use: test for cohort and sex differences in thresholds and correlation structure using univariate saturated models.

Table S5 Quantity of alcohol use: test for cohort and sex differences in thresholds and correlation structure using univariate saturated models.

Table S6 Goodness-of-fit of the single liability dimension (SLD), independent liability dimension (ILD) and combined model to alcohol frequency and initiation at ages 13–15, within cohorts.

Table S7 Univariate variance decomposition of alcohol initiation: model fitting results in age groups 13–15 and 16–17, on pooled cohorts.

Table S8 Variance decomposition under the combined model: model fitting results for alcohol frequency combined with initiation, age group 13–15, on pooled cohorts.

Table S9 Variance decomposition under the single liability dimension model: model fitting results for alcohol

frequency and quantity, age group 16–17, on pooled cohorts.

Table S10 Variance decomposition under the single liability dimension model: model fitting results for alcohol frequency and quantity in age group 18–21, on pooled cohorts.