RESEARCH REPORT

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Birth cohort effects on the quantity and heritability of alcohol consumption in adulthood: a Finnish longitudinal twin study

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ABSTRACT

Aims To estimate birth cohort effects on alcohol consumption and abstinence in Finland and to test differences between birth cohorts in genetic and environmental sources of variation in Finnish adult alcohol use. **Design** The Older Finnish Twin Cohort longitudinal survey study 1975–2011. **Setting** Finland. **Participants** A total of 26 121 same-sex twins aged 18–95 years (full twin pairs at baseline n = 11 608). **Measurements** Outcome variables were the quantity of alcohol consumption (g/month) and abstinence (drinking zero g/month). Predictor variables were 10-year birth cohort categories and socio-demographic covariates. In quantitative genetic models, two larger cohorts (born 1901–20 and 1945–57) were compared. **Findings** Multi-level models in both sexes indicated higher levels of alcohol consumption in more recent birth cohorts and lower levels in earlier cohorts, compared with twins born 1921–30 (all $P < 0.003$). Similarly, compared with twins born 1921–30, abstaining was more common in earlier and less common in more recent cohorts (all $P < 0.05$), with the exception of men born 1911–20. Birth cohort differences in the genetic and environmental variance components in alcohol consumption were found: heritability was 21% [95% confidence interval (CI) = 0–56%] in the earlier-born cohort of women [mean age 62.8, standard deviation (SD) = 5.3] and 51% (95% CI = 36–56%) in a more recent cohort (mean age 60.2, SD = 3.7) at the age of 54–74. For men, heritability was 39% (95% CI = 27–45%) in both cohorts. In alcohol abstinence, environmental influences shared between co-twins explained a large proportion of variation in the earlier-born cohort (43%, 95% CI = 23–63%), whereas non-shared environmental (54%, 95% CI = 39–72%) and additive genetic influences (40%, 95% CI = 13–61%) were more important among more recent cohorts of men and women. **Conclusion** The contribution of genetic and environmental variability to variability in alcohol consumption in the Finnish population appears to vary by birth cohort.

Keywords Abstinence, alcohol use, cohort study, environment, heritability, longitudinal, twins.

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INTRODUCTION

Studies in western countries such as the United Kingdom and the United States have noted higher levels of alcohol consumption in more recent birth cohorts [1–4]. Similar findings have been reported in Finland, as the large birth cohorts born after World War II started to drink more than cohorts preceding them [5]. Finnish alcohol policies have traditionally been strict since the ending of Prohibition in 1932, but a progressive liberalization began with a 1969 law allowing convenience stores to sell mild alcoholic beverages such as beer. Subsequently, alcohol consumption increased steadily with annual per capita consumption increasing from 3.6 litres of pure alcohol in 1968 to 10.8 litres in 2016 [6,7].

High abstinence rates have been associated with so-called dry drinking cultures, where alcohol is not a part of daily life and access is more restricted, such as in Finland [8]. However, abstinence rates have decreased in northern Europe, reaching lower levels than in the traditionally 'wet' southern Europe [9]. In Finland, the abstinence rate among men has decreased from 14% in 1955 to 6.6% in 2006, and among women from 31 to 9.5% [10,11]. Studies in Sweden and the United Kingdom suggest that people born in the

early 20th century are more likely to abstain than later born cohorts [4,12]. Ahacic *et al.* concluded that decreasing abstinence rates in Sweden appear to be attributable to decreasing abstinence in successive cohorts, rather than drinkers becoming abstainers as they age [12].

Genetic factors inferred from family and twin studies account for approximately 40–50% of individual differences in alcohol use [13,14]. The proportion of variation explained by genetic factors (i.e. heritability) may increase in an environment where alcohol is more readily available, or alcohol use is less restricted by the socio-cultural environment [15–17]. As more recent birth cohorts have had easier access to alcohol than earlier cohorts and drinking has become culturally more normative, heritability may have increased along with increasing consumption. However, existing literature is inconclusive on whether birth cohort moderates the heritability of alcohol use. An Australian study examined the frequency and quantity of alcohol use among adult twins in two age groups ($<$ 30 versus $>$ 30 years) and found no differences in heritability estimates [18]. Birth cohort differences controlling for the effect of age can be examined by comparing different birth cohorts at similar ages. However, using cross-sectional data, Heath *et al.* could not differentiate cohort effects from age effects [18]. Geels *et al.* compared two adolescent birth cohorts, studied in 1993 and 2008. While alcohol use initiation at the age of 13–15 years was more prevalent in the more recent cohort, there were no differences in heritability [19]. Furthermore, genetic and environmental influences on alcohol abstinence do not seem to be moderated by birth cohort [18,20–22]. Taken together, the few existing studies on birth cohort effects on the heritability of alcohol use have not been optimal for distinguishing cohort effects from age effects.

The current study focuses on changes in adult alcohol use in Finland over the period 1975–2011. The aims were to (1) estimate differences between birth cohorts in the quantity of monthly alcohol consumption and abstinence during the life course and (2) compare the relative contributions of genetic and environmental variance in alcohol consumption and abstinence between birth cohorts. Examining these research questions together is informative on possible gene–environment interaction effects: parallel changes in the prevalence and heritability of alcohol use would suggest a moderating effect of environmental conditions on genetic influences [23].

METHODS

Data

We used data from the Older Finnish Twin Cohort study, consisting of same-sex twin pairs born before 1958 with both co-twins alive in 1975. Data were collected in 1975, 1981, 1990 and 2011. Participants in the first two waves were born 1880–1957. Only participants born between 1930 and 1957 were invited to the 1990 follow-up, and only participants born 1945–57 received an invitation for the fourth follow-up in 2011. In the Older Finnish Twin cohort, the older age-groups are somewhat under-represented, whereas non-married individuals are over-represented compared to the general population. In its geographical distribution the sample is, to a large degree, representative of the population [24]. More details of the study design and data collection are provided elsewhere [25,26].

A total of 29 007 individuals participated in the study. Only participants who provided information on alcohol use on at least one occasion in addition to information on covariates were included in the individual-level analyses (n = 26 121). Zygosity was determined by a validated questionnaire which has more than 95% accuracy when compared with blood typing analysis [27]. All twins of unknown zygosity (n = 2943) were excluded from the quantitative genetic models, but contributed to the individual-level analyses. The study comprised 2395 twins without their co-twin.

Birth cohorts

The age of the participants at study baseline in 1975 ranged from 18 to 95 years. For the individual-level analyses, participants were grouped into seven 10-year cohorts based on their birth year. Due to the small number of participants born before 1900, the first birth cohort included individuals born 1880–1900.

In order to estimate the genetic and environmental variance components in alcohol use behaviours independently of age effects, we compared two larger birth cohorts at similar ages in the quantitative genetic analyses. The earlier-born cohort comprised twins born 1901–20, at the age of 55–74 [mean = 62.8, standard deviation (SD) = 5.3] in 1975. The more recent cohort was born 1945–1957, aged 54–66 (mean = 60.2, SD = 3.7) in 2011. Table 1 presents the final sample of full twin pairs (n = 11 608) for each wave by birth cohort and zygosity.

Measures

Assessment of alcohol use

All questionnaires included items on alcohol use frequency and quantity during an average week or month. Quantity was assessed on a seven-point scale separately for beer, wine and spirits. The upper limit for weekly and monthly consumption was anchored at $>$ 48 bottles of beer per week, $>$ 10 bottles of wine per week and $>$ 20 bottles of spirits per month.

A bottle of beer was defined as a standard 0.33-litre bottle with average alcohol content of 4.7%. A bottle of wine was defined as a 0.75-litre bottle with 11% average alcohol

Table 1 Number of complete twin pairs at each study year stratified by zygosity and birth cohort.

Study year	*1975*	*1981*	*1990*	*2011*
Birth cohort				
1880–1900				
MZ	34	17		
DZ	46	17		
1901–10				
MZ	149	102		
DZ	292	178		
1911–20				
MZ	271	214		
DZ	546	422		
1921–30				
MZ	439	393	44	
DZ	1027	869	84	
1931–40				
MZ	580	551	421	
DZ	1403	1262	973	
1941–50[a]				
MZ	1068	1010	732	469
DZ	2342	2177	1509	980
1951–57				
MZ	1114	1037	703	702
DZ	2293	2142	1385	1413

MZ = monozygotic twins, DZ = dizygotic twins, [a]1945–1950 in 2011.

content, and a bottle of spirits as being 0.5 litres with 35% alcohol content. For each beverage the reported amount was converted into grams of 100% alcohol per month and summed to form a composite measure of total alcohol consumption (continuous variable), as had been performed previously [28,29]. The variable was log-transformed due to its highly skewed distribution (in 1975: mean = 243.8; SD = 414.1; skewness = 4.8; kurtosis = 45.6).

Abstinence was determined from the composite alcohol consumption measure. Participants reporting consuming no alcohol (0 g/month) were classified as abstaining from alcohol during the given study year (binary variable).

Covariates

All individual-level analyses were adjusted for potential confounders. Time-variant covariates included marital status (1 = married, co-habiting or remarried, 0 = single, divorced or widowed), smoking status (1 = current smoker, 0 = non-smoker) and body mass index (BMI, kg/m^2). Time-invariant covariates were education (range from 0 = < 6 years to 3 = high school or more) and occupational status. Information on participants' education came from the 1975 and 1981 questionnaires, and educational level was defined as the highest degree reported in 1975 or 1981. Occupational status was coded from the 1975 data and participants were classified based on their reported occupation (for classification, see Table 2) [30].

Analyses

Multi-level analyses

The Older Finnish Twin Cohort study is structured as a longitudinal panel design. Thus, a multi-level analysis can be implemented to estimate between-individual variability in within-individual patterns of change over time. In a multi-level analysis framework, repeated measurements over time may be viewed as units (level 1) nested within individuals (level 2), which is specified as a two-level model of individual change. The non-independency of observations within twin pairs can be accounted for by adding a third level to the model, denoting individuals nested within pairs (level 3).

A basic multi-level model is composed of fixed and random effects. Fixed effects represent the mean of the trajectory pooling together all the individuals within the sample, and random effects represent the variation of individual trajectories around group means. Together they capture the general characteristics of growth for both the entire group and for individuals within the group [31].

The method employed in this study to estimate cohort effects is adapted from Yang & Land [32–34]. It incorporates cohort effects by including cohort indicator variables as well as age × cohort interaction terms in the multi-level models. The model specification is given in the Supporting information. Sex differences in alcohol consumption and abstinence were tested by including sex × cohort and sex × age interaction terms in the models.

Logistic regression was used in models for abstinence in which the outcome was binary (0 = not abstinent, 1 = abstinent). Equations for multi-level logistic regression models for binary outcomes are very similar to equations 1–5 in the Supporting information, with the exception of using a logit instead of a linear link. In order to simplify model estimation, the final logistic models included random intercepts but no random slopes for individuals [35]. Multi-level analyses were performed with Stata version 14.0 [36].

Quantitative genetic models

The total variance in alcohol use was decomposed into underlying genetic and environmental effects: additive genetic component (A), shared environmental component (C) and non-shared environmental component (E). Estimation of the genetic and environmental variance components is based on the assumptions that both monozygotic (MZ) and dizygotic (DZ) co-twins similarly share environmental factors and that while MZ co-twins share all their genes, DZ co-twins share, on average, half of their segregating genes.

Table 2 Descriptive statistics: proportions and means (standard deviation) by birth cohort and gender.

	1880–1900		*1901–10*		*1911–20*		*1921–30*		*1931–40*		*1941–50*[a]		*1951–57*	
	M	*W*	*M*	*W*	*M*	*W*	*M*	*W*	*M*	*W*	*M*	*W*	*M*	*W*
Education (1975–81) (%)														
< 6 years	39.0	39.6	24.2	23.8	11.0	7.4	4.7	3.8	2.2	2.0	1.2	0.6	0.8	0.6
6 years	41.6	39.0	56.9	52.7	58.3	61.6	59.9	60.1	50.8	49.0	36.0	34.1	25.9	22.9
Middle school	10.4	18.3	15.4	20.5	26.0	26.2	28.2	30.6	37.5	40.2	46.6	49.2	52.9	49.3
High school or more	9.0	3.1	3.6	3.0	4.7	4.8	7.2	5.6	9.5	8.8	16.2	16.1	20.4	27.1
Occupational status in 1975 (%)														
Other/ unknown	4.9	29.5	2.9	18.7	1.2	9.9	1.2	6.3	0.6	4.9	3.1	4.8	20.6	27.0
Farmer	47.5	25.5	30.3	25.6	25.3	23.6	17.0	22.6	8.9	10.3	5.0	4.5	4.7	1.2
Uskilled worker	14.8	13.4	14.2	17.3	15.8	19.1	13.1	15.3	10.5	14.9	6.8	10.9	13.0	11.1
Skilled worker	18.0	15.4	34.6	24.6	36.6	25.2	39.1	30.4	46.1	34.5	49.6	31.9	51.0	30.4
Lower white collar	8.2	10.1	13.5	12.0	14.9	18.9	20.4	21.2	21.3	30.2	23.9	40.9	9.4	29.1
Upper white collar	6.6	6.0	4.5	1.9	6.3	3.4	9.3	4.1	12.6	5.2	11.7	7.0	1.3	1.3
Abstinent from alcohol (%)														
1975	34.3	68.7	22.1	63.1	15.2	54.1	11.2	41.0	8.4	26.4	6.9	15.0	13.0	17.1
1981	39.5	66.2	26.9	64.9	20.5	59.8	14.2	45.3	10.1	28.8	7.7	17.9	8.0	15.8
1990	–	–	–	–	–	–	12.4	36.5	10.1	28.9	7.8	18.6	8.7	14.9
2011	–	–	–	–	–	–	–	–	–	–	4.1	10.3	5.7	8.5
Alcohol consumption[b]														
1975	157.1 (216.3)	60.5 (74.6)	303.9 (483.1)	96.8 (182.6)	342.5 (507.1)	94.5 (154.5)	403.6 (526.9)	108.9 (157.2)	474.3 (598.7)	141.8 (227.2)	457.4 (562.6)	170.2 (223.0)	410.6 (463.1)	176.1 (237.1)
1981	166.4 (192.7)	57.0 (46.8)	247.3 (391.9)	72.0 (120.0)	278.6 (424.9)	80.5 (128.9)	398.8 (537.9)	115.4 (194.5)	472.4 (572.9)	142.8 (233.5)	478.9 (564.2)	186.4 (259.2)	409.4 (484.9)	159.5 (196.6)
1990	–	–	–	–	–	–	442.2 (687.2)	119.2 (193.3)	476.9 (564.2)	162.0 (254.0)	506.7 (606.3)	219.8 (298.3)	476.6 (545.7)	203.7 (274.5)
2011	–	–	–	–	–	–	–	–	–	–	512.2 (698.0)	209.9 (329.8)	566.3 (749.0)	226.5 (333.5)

[a]1945–1950 in 2011. [b]Mean value (grams of alcohol per month) for those currently using alcohol; M = men, W = women.

Cohort effects on the magnitude of genetic and environmental influences on alcohol consumption were analysed by fixing A, C and E (ACE) variance components to be equal in the two defined larger cohorts and then comparing the fit of this model with the fit of the model having separate estimates for the cohorts. If the model with separate estimates has a better fit, this implies that the groups have heterogeneous variance components [37]. Before the cohort analysis, gender differences were tested by fitting a sex-limitation model separately in both cohorts, fixing variance components equal for men and women.

Abstinence was analysed using a liability-threshold model, where the categories (abstinent versus not abstinent) were assumed to reflect an imprecise measurement of an underlying normal distribution of liability [38]. Cohort differences were analysed in the same manner as for alcohol consumption: by comparing the model with separate estimates with a model with estimates fixed equally in the two cohorts.

Model parsimony was assessed by the Akaike information criterion (AIC) [39] and model fit using the log-likelihood function (−2LL). The difference between the log-likelihoods of two models is asymptotically distributed as χ^2, with the number of degrees of freedom reflecting the difference in parameters between models. Structural equation modelling was performed using OpenMx version 2.9.9. within R 3.0.2 [40,41]. Age was controlled for in the quantitative genetic models. Twins without their co-twin in the study provided information for means and variances.

RESULTS

Descriptive statistics for the sample stratified by sex and birth cohort are presented in Table 2 and Supporting information, Table S1.

Birth cohort differences in trajectories of alcohol consumption

We found significant interaction effects for sex and the main explanatory variables (cohort: $\chi^2_{(6)} = 119.14$, $P < 0.001$; age: $\chi^2_{(6)} = 76.58$, $P < 0.001$), and thus conducted the multi-level analyses stratified by sex. Multi-level analyses suggested that the more recent cohorts had higher and the earlier-born cohorts lower levels of alcohol use than the reference group (1921–30 cohort) on average (Table 3).

In addition to the weak linear main effect of age, models showed significant age × cohort interaction effects, indicating that alcohol consumption decreased with age in the earlier cohorts but increased in more recent cohorts. In addition, age had a significant quadratic effect. Figure 1 illustrates model-predicted alcohol use trajectories for men and women in four of the later-born cohorts, and shows that the more recent cohorts had higher predicted levels of alcohol consumption.

Birth cohort differences in trajectories of abstinence

Abstinence analyses where stratified by sex, because a significant interaction effect was found between cohort and sex ($\chi^2_{(6)} = 140.41$, $P < 0.001$). Logistic models revealed cohort differences in the odds of being abstinent as well as in the abstinence growth rates. Table 4 shows that, compared to the reference cohort (1921–30), the earlier-born cohorts of women had at least thrice the odds of being abstinent and the later-born cohorts were increasingly less likely to be abstinent. A similar, albeit not quite as evident, effect was found among men. Linear growth rate estimates suggested that as age increased the odds of abstaining also increased. However, age × cohort interaction effects reflected that the most recent cohorts showed a decrease in abstinence throughout the four waves of data. The quadratic term was non-significant and was dropped from the final model.

Quantitative genetic models of alcohol consumption

The two birth cohorts were compared with one another at similar ages in the quantitative genetic analyses. Within-pair correlations with 95% confidence intervals (CI) and corresponding sample sizes are presented in the Supporting information, Table S2. Sex-limitation models suggested heterogeneous variance estimates (Supporting information, Table S3); thus, we conducted the cohort comparison analyses separately for men and women.

In the cohort comparison in men, the model with homogeneous estimates fitted the data better than the heterogeneity model ($\chi^2_{(3)} = 2.5$, $P = 0.475$; AIC: 4116.8 versus 4112.6). For women, the model with heterogeneous variance component estimates between the earlier and more recent cohort was supported ($\chi^2_{(3)} = 39.3$, $P < 0.001$; AIC: 3553.6 versus 3586.9). Standardized variance component estimates for the full ACE models are presented in Table 5. Heritability was 39% (95% CI = 27–45%) for the earlier and the more recent cohort of men. For women, the earlier-born cohort had a heritability estimate of 21% (95% CI = 30–58%), whereas for the more recent cohort it was 51% (95% CI = 45–56%). The shared environmental component explained 21% (95% CI = 0.00–0.45) of the variation in the earlier cohort and 0% (95% CI = 0.00–0.12) in the more recent cohort.

Quantitative genetic models of abstinence

Because no gender differences in variance component estimates were found (Supporting information, Table S3),

Table 3 Multi-level model estimates of birth cohort and age effects on alcohol consumption.

	Men			*Women*		
Fixed effects	*Coefficient*	*SE*	*P-value*	*Coefficient*	*SE*	*P-value*
For intercept						
Intercept	4.519	0.072	< 0.001	4.384	0.057	< 0.001
Birth cohort						
1880–1900	−0.692	0.172	< 0.001	−0.385	0.124	0.002
1901–10	−0.302	0.065	< 0.001	−0.251	0.058	< 0.001
1911–20	−0.207	0.049	< 0.001	−0.197	0.043	< 0.001
1921–30	ref.			ref.		
1931–40	0.190	0.034	< 0.001	0.237	0.032	< 0.001
1941–50	0.239	0.032	< 0.001	0.432	0.030	< 0.001
1951–57	0.136	0.035	< 0.001	0.409	0.031	< 0.001
For linear growth rate						
Age	−0.007	0.003	0.037	−0.009	0.004	0.017
Birth cohort × age						
1880–1900	−0.021	0.031	0.486	0.015	0.024	0.526
1901–10	−0.037	0.010	< 0.001	−0.018	0.010	0.076
1911–20	−0.018	0.007	0.008	−0.010	0.007	0.166
1921–30	ref.			ref.		
1931–40	0.014	0.004	< 0.001	0.012	0.004	0.003
1941–50	0.018	0.003	< 0.001	0.017	0.004	< 0.001
1951–57	0.024	0.003	< 0.001	0.021	0.004	< 0.001
For quadratic growth rate						
Age^2	−0.0003	0.00004	< 0.001	−0.0003	0.00003	< 0.001
Control variables						
Smoker (yes)	0.383	0.013	< 0.001	0.366	0.013	< 0.001
BMI (unit kg/m^2)	0.043	0.002	< 0.001	0.006	0.002	< 0.001
Married (yes)	−0.216	0.014	< 0.001	−0.089	0.011	< 0.001
Education						
< 6 years	−0.022	0.069	0.751	−0.201	0.070	0.004
6 years	−0.082	0.033	0.013	−0.188	0.026	< 0.001
Middle school	−0.048	0.029	0.101	−0.164	0.023	< 0.001
High school or more	ref.			ref.		
Social class in 1975						
Other/unknown	−0.246	0.046	< 0.001	−0.214	0.040	< 0.001
Farmer	−0.539	0.046	< 0.001	−0.428	0.048	< 0.001
Uskilled worker	−0.070	0.044	0.110	−0.183	0.042	< 0.001
Skilled worker	−0.087	0.037	0.017	−0.173	0.039	< 0.001
Lower white collar	−0.128	0.037	0.001	−0.076	0.037	0.040
Upper white collar	ref.			ref.		
Random effects: variance components						
Level 1: within-person	0.362	0.005		0.328	0.004	
Level 2: between individuals	0.342	0.011		0.173	0.007	
In growth rate	0.0005	0.00003		0.0004	0.00002	
Level-3: between twin pairs	0.287	0.012		0.185	0.008	
Goodness-of-fit						
BIC	72 230.46			61 522.59		
AIC	71 947.93			61 269.01		

Alcohol consumption variable was log-transformed. SE = standard error; BIC = Bayesian information criterion; AIC = Akaike information criterion; BMI = body mass index.

men and women were combined in the cohort comparison analyses. The birth cohort heterogeneity model provided a significantly better fit for the data than the homogeneous variances model ($\chi^2_{(3)} = 17.8$, $P < 0.001$; AIC: −12856.4 versus −12844.6). The variance component estimates for the earlier and more recent cohort are presented in Table 5. Comparing same-aged (54–74 years) people, the shared environment explained 43% of the variation in the earlier-born cohort, but merely 6% in the more recent cohort.

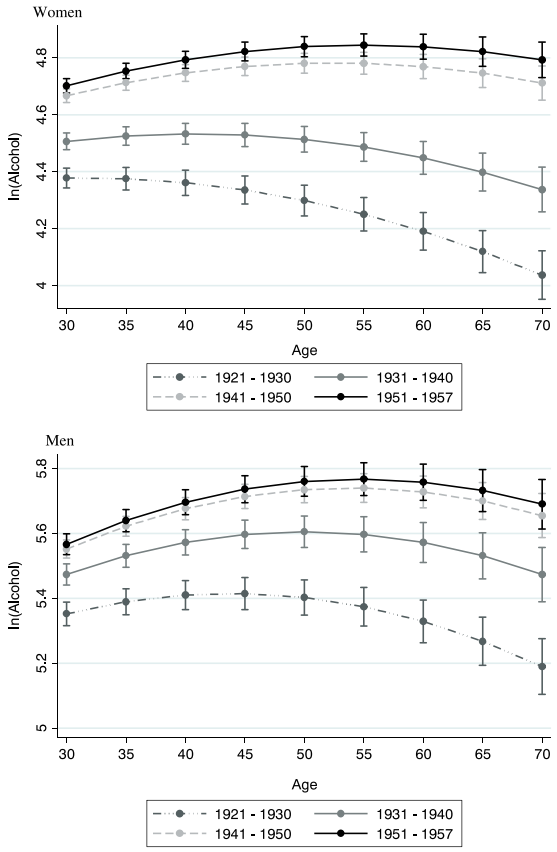

Figure 1 Model predicted age trajectories of alcohol consumption for women (top) and men (bottom) stratified by birth cohort

DISCUSSION

Our first aim was to examine the role of birth cohort in adult alcohol use in Finland during the period 1975–2011. The second goal was to investigate birth cohort differences in genetic and environmental sources of variation in alcohol consumption and abstinence by comparing variance component estimates for two birth cohorts at similar ages.

Mean levels of alcohol consumption were systematically higher in more recent cohorts in both men and women, with effects most evident in cohorts born since 1941. These results confirm earlier findings from Finland in a larger sample and controlling for the confounding effects of education, marital status, smoking and BMI [5]. Higher levels of alcohol consumption in more recent cohorts have also been noted in other western countries [1–4].

Age effects on quantity of alcohol use were relatively small once birth cohort was taken into account. Importantly, we also found age × cohort interaction effects: age-related decline in alcohol consumption was present in the earlier-born cohorts, whereas an opposite effect was found in more recent cohorts, not inconsistent with earlier findings [5]. Jointly, our results of cohort and age effects suggest that the observed increase in levels of per capita alcohol consumption may be attributable to population-level changes, where earlier-born cohorts with lower levels of alcohol consumption have been replaced by more recent cohorts exhibiting higher alcohol use.

Trajectories of alcohol abstinence revealed similar trends, with odds of abstaining becoming lower across successive birth cohorts. More recent cohorts were less likely to become abstinent as they aged: results consistent with findings from Sweden [12].

Quantitative genetic analyses suggested a lower heritability of alcohol consumption in the earlier-born cohort of women. Genetic factors explained an estimated 21% of the variance of alcohol consumption in the earlier and 51% in the more recent cohort at the age of 54–74. Variation explained by shared environment decreased from 21 to 0%. These findings are novel, as the few previous studies found no evidence of birth cohort differences [18–22]. However, previous studies compared cohorts that were born in close proximity to one another, probably explaining why no such effects were found. Societal changes (e.g. changes in availability of alcohol and social acceptability of alcohol use) impact birth cohorts in ways that may be difficult to detect in comparisons made between two successive cohorts. Our findings of parallel changes in the quantity and heritability of alcohol use in women suggest a moderating effect of the environment on genetic influences [23].

These results are in line with the social control and the social push theories [23,42]. In a society with strong social control, genetic factors have less influence on phenotypical variation, as a large proportion of the population will exhibit the same socially desirable phenotype. This may have been the case among the earlier cohort of women. After the 1960s, the normative sanctions on alcohol use began to lift, allowing freer expression of genetic predispositions in the later-born cohort.

In contrast to our results in women, variance component estimates for the earlier and more recent cohort of men could be set equal. Heritability of alcohol consumption quantity was 39%, and non-shared environments explained the remaining variation. The shared environment does not seem to have influenced men's alcohol use in the same manner as in women. Differences in social control exerted by the environment may explain this finding. Among earlier and more recent cohorts in Finland, men have been relatively free to consume alcohol, while women's alcohol use remained socially restricted only for the earlier-born cohort.

The main finding concerning genetic and environmental influences on abstinence was the steep decline in the importance of shared environmental influences. Shared environment explained nearly half of the variation in the

Table 4 Logistic multi-level model estimates of cohort and age effects on abstinence from alcohol.

	Men			*Women*		
Fixed effects	*Odds ratio*	*SE*	*P-value*	*Odds ratio*	*SE*	*P-value*
For intercept						
Intercept	0.017	0.008	< 0.001	0.054	0.016	< 0.001
Birth cohort						
1880–1900	4.975	30.980	0.045	30.730	10.640	0.003
1901–10	3.251	10.125	0.001	30.360	0.748	< 0.001
1911–20	1.517	0.436	0.147	20.640	0.470	0.000
1921–30	ref.			ref.		
1931–40	0.567	0.123	0.009	0.331	0.051	< 0.001
1941–50	0.360	0.074	< 0.001	0.140	0.021	< 0.001
1951–57	0.542	0.118	0.005	0.130	0.020	< 0.001
For linear growth rate						
Age	1.062	0.023	0.007	10.054	0.015	< 0.001
Birth cohort × age						
1880–1900	1.015	0.148	0.917	0.906	0.066	0.173
1901–10	1.092	0.058	0.100	0.975	0.033	0.441
1911–20	1.065	0.044	0.134	10.022	0.026	0.375
1921–30	ref.			ref.		
1931–40	0.980	0.025	0.414	0.972	0.016	0.086
1941–50	0.940	00.021	0.004	0.941	0.014	< 0.001
1951–57	0.920	0.021	< 0.001	0.920	0.013	< 0.001
Control variables						
Smoker (yes)	0.111	0.013	< 0.001	0.159	0.015	< 0.001
BMI (unit kg/m^2)	0.959	0.015	0.007	10.022	00.008	0.006
Married (yes)	0.470	0.046	< 0.001	0.751	0.045	< 0.001
Education						
< 6 years	9.267	30.390	< 0.001	70.165	10.863	< 0.001
6 years	2.724	0.592	< 0.001	30.304	0.483	< 0.001
Middle school	1.454	0.285	0.056	20.354	0.312	< 0.001
High school or more	ref.			ref.		
Social class in 1975						
Other/unknown	6.185	10.890	< 0.001	40.730	10.110	< 0.001
Farmer	7.394	20.218	< 0.001	90.782	20.435	< 0.001
Uskilled worker	2.950	0.892	< 0.001	30.270	0.787	< 0.001
Skilled worker	1.682	0.445	0.049	10.890	0.434	0.006
Lower white collar	1.750	0.466	0.035	10.080	0.239	0.742
Upper white collar	ref.			ref.		
Random effects: variance components						
Random intercept: individuals	4.961	0.462		3.566	0.232	
Random intercept: twin pairs	8.754	0.650		5.557	0.300	
Goodness-of-fit						
BIC	14 739.54			27 272.66		
AIC	14 514.29			27 044.49		

SE = standard error; BIC = Bayesian information criterion; AIC = Akaike information criterion; BMI = body mass index.

earlier-born cohort, but merely 6% in the more recent cohort. One potential explanation for this finding is the strong tradition of educating and parenting children and youth to embrace the values of the temperance movement, an influential ideology from the mid-1800s onwards that began to decline during the 1960s [43]. Changes in youth education regarding alcohol use may have resulted in the declining importance of shared environmental influences on abstinence. Genetic factors explained 25% of the variation in the earlier-born cohort and 40% in the more recent cohort. Most previous studies reported no genetic influences on alcohol abstinence [20–22]. However, many earlier studies investigated the initiation of alcohol use versus abstinence in adolescence or early adulthood, while our estimates are for older adults. Other factors, such as operationalization of the abstinence variable, birth cohort differences or cultural contexts, may also explain this difference.

Table 5 Standardized estimates (95% confidence intervals) of genetic and environmental variance components for alcohol use in two birth cohorts of men and women.

Age		*54–74*	
Birth cohort	*A*	*C*	*E*
Alcohol consumption			
Men			
1901–20	0.39 (0.27–0.45)	0.00 (0.00–0.08)	0.61 (0.55–0.68)
1945–57	0.39 (0.27–0.45)	0.00 (0.00–0.08)	0.61 (0.55–0.68)
Women			
1901–20	0.21 (0.00–0.56)	0.21 (0.00–0.45)	0.58 (0.43–0.76)
1945–57	0.51 (0.36–0.56)	0.00 (0.00–0.12)	0.49 (0.44–0.55)
Abstinence			
1901–20	0.25 (0.01–0.50)	0.43 (0.23–0.63)	0.32 (0.23–0.43)
1945–57	0.40 (0.13–0.61)	0.06 (0.00–0.30)	0.54 (0.39–0.72)

A = additive genetic; C = shared environmental; E = non-shared environmental influences.

Our findings should be considered in the context of some limitations. First, the data did not permit isolating the effects of cohort, age and period. As a result, period effects could not be explicitly estimated. Historical events during the study period may have caused fluctuations in alcohol consumption trajectories and thus had an impact on the average alcohol use of all birth cohorts. Another notable limitation was that the earliest cohorts did not have sufficient follow-up measurements to confidently estimate the effects of aging in the multi-level models. Thus, the results of the earliest cohorts should be interpreted with caution. A further limitation is that we did not specifically analyse cohort differences in the genetic and environmental influences on the overlap in the liability for any alcohol use and the amount of consumption, as would be possible with the common-causal-contingent approach [44]. Further studies are needed to model more comprehensively the possible cohort effects in transitions between different stages of alcohol use. The data in this study cannot answer whether the lower heritability of alcohol use in the earlier-born cohort was stable, or whether heritability had decreased with age, as the earlier-born cohorts were not followed-up from early adulthood. The most parsimonious hypothesis is that heritability remains somewhat stable during adulthood, but longitudinal studies of similar birth cohorts with an earlier start of follow-up would be necessary for confirmation.

In conclusion, the quantity of alcohol consumption as well as its heritability have increased in more recent birth cohorts in Finland. Heritable influences have been moderated by environmental conditions, so that the socio-cultural changes leading to increased consumption also allowed individuals to express their genetic predispositions more freely for alcohol use. Future research is encouraged to address whether our findings will replicate in birth cohorts born after the 1950s and in other countries.

Declaration of interests

None.

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Table S1 Mean age (SD) for each birth cohort by gender and study year.

Table S2 Pairwise and tetrachoric correlations in alcohol consumption and abstinence between MZ and DZ twin pairs by birth cohort and gender.

Table S3 Sex-limitation model fit statistics for alcohol use in the earlier and more recent cohort.