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ORIGINAL ARTICLE

Alcoholism and alcohol drinking habits predicted from alcohol dehydrogenase genes

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Alcohol drinking habits and alcoholism are partly genetically determined. Alcohol is degraded primarily by alcohol dehydrogenase (ADH) wherein genetic variation that affects the rate of alcohol degradation is found in *ADH1B* and *ADH1C*. It is biologically plausible that these variations may be associated with alcohol drinking habits and alcoholism. By genotyping 9080 white men and women from the general population, we found that men and women with *ADH1B* slow vs fast alcohol degradation drank more alcohol and had a higher risk of everyday drinking, heavy drinking, excessive drinking and of alcoholism. For example, the weekly alcohol intake was 9.8 drinks (95% confidence interval (CI): 9.1–11) among men with the *ADH1B · 1/1* genotype compared to 7.5 drinks (95% CI: 6.4–8.7) among men with the *ADH1B · 1/2* genotype, and the odds ratio (OR) for heavy drinking was 3.1 (95% CI: 1.7–5.7) among men with the *ADH1B · 1/1* genotype compared to men with the *ADH1B · 1/2* genotype. Furthermore, individuals with *ADH1C* slow vs fast alcohol degradation had a higher risk of heavy and excessive drinking. For example, the OR for heavy drinking was 1.4 (95% CI: 1.1–1.8) among men with the *ADH1C · 1/2* genotype and 1.4 (95% CI: 1.0–1.9) among men with the *ADH1B · 2/2* genotype, compared with men with the *ADH1C · 1/1* genotype. Results for *ADH1B* and *ADH1C* genotypes among men and women were similar. Finally, because slow *ADH1B* alcohol degradation is found in more than 90% of the white population compared to less than 10% of East Asians, the population attributable risk of heavy drinking and alcoholism by *ADH1B · 1/1* genotype was 67 and 62% among the white population compared with 9 and 24% among the East Asian population.
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Keywords: genetic association study; alcoholism; population-based study; alcohol; alcohol dehydrogenase; acetaldehyde

Introduction

Alcoholism and alcohol drinking in general represent huge public health problems in most countries worldwide, preventing many individuals from successfully holding a job or looking after a family. In addition, excessive alcohol use leads to diseases such as liver cirrhosis, chronic pancreatitis, upper gastrointestinal cancers, cardiomyopathy, polyneuropathy and dementia. It has been shown in twin studies that heritability explains approximately 50% of alcoholism and problem drinking in the white population.[1,2]

The region surrounding the alcohol dehydrogenase (ADH) gene cluster is known to be associated with alcoholism from whole-genome scans.[3] Well-known

functional polymorphisms of *ADH1B* and *ADH1C* may explain this finding because the *ADH1B · 2* vs the *ADH1B · 1* allele confer a 38-fold increase in *in vitro* alcohol degradation rate (that is, the conversion of ethanol to acetaldehyde) and the *ADH1C · 1* vs the *ADH1C · 2* allele confer a 2.5-fold increase in *in vitro* alcohol degradation rate.[4]

During alcohol degradation, acetaldehyde is only found in low concentrations. If concentrations become high, for example, during treatment with disulfiram (used in some countries to prevent alcohol intake among alcoholics) or in individuals with a defective acetaldehyde dehydrogenase (found among Asians), individuals experience severe nausea and flushing and automatically abstain from drinking alcohol. It is possible that similar but less pronounced responses to alcohol are more likely to be produced among individuals carrying the fast alcohol degradation *ADH1B · 2* and *ADH1C · 1* alleles compared to individuals carrying the slow alcohol degradation *ADH1B · 1* and *ADH1C · 2* alleles. If so, individuals with slow alcohol degradation may be able to drink larger quantities of alcohol without experiencing discomfort due to elevated acetaldehyde levels, and consequently are more likely to use alcohol excessively and to develop alcoholism. This issue has been addressed in different populations in case–control settings where allele frequencies of *ADH1B* and *ADH1C* are compared between alcoholics and nonalcoholics.[5–28] To our knowledge, this has not been studied in a prospective setting in the general white population and it is unknown if the *ADH1B* and *ADH1C* polymorphisms are associated with alcohol drinking habits, such as amount of usual intake.

In the present study, we have genotyped a sample of 9080 men and women from the general white population to test the hypotheses that slow alcohol degradation *ADH1B · 1* and *ADH1C · 2* alleles are associated with alcohol drinking habits and with increased risk of alcoholism.

Results

The frequencies of the *ADH1B* and *ADH1C* alleles coding for slow alcohol degradation was 0.98 (*ADH1B · 1*) and 0.42 (*ADH1C · 2*) (Table 1). Genotypes were in Hardy–Weinberg equilibrium ($P = 0.8$ for *ADH1B* genotypes and $P = 0.7$ for *ADH1C* genotypes by χ^2-test). *ADH1B · 2* were associated with *ADH1C · 1* (linkage disequilibrium coefficients $D' = 0.90$ and $r^2 = 0.012$).

For *ADH1B*, we found that men and women who were homozygous for the slow alcohol degrading *ADH1B · 1* allele had a higher alcohol intake than men and women who were fast alcohol degrading *ADH1B · 2* heterozygotes or homozygotes. For example, men with the *ADH1B · 1/1* genotype drank on average 9.8 drinks per week (95% confidence interval (CI): 9.1–11) and men with the *ADH1B · 1/2* genotype drank on average 7.5 drinks per week (95% CI: 6.4–8.7) (Table 2). Furthermore, odds for any, daily, heavy and excessive alcohol drinking were 2–4 times higher among men and women who were *ADH1B · 1* homozygotes than among men and women who were *ADH1B · 2* hetero- or homozygotes.

Using the brief Michigan Alcoholism Screening Test (brief MAST) we found that men with the slow alcohol degradation *ADH1B · 1/1* genotype had a two- to fourfold risk of alcoholism compared to men with the fast alcohol degradation *ADH1B · 2/1* or *ADH1B · 2/2* genotypes (Table 2). The hazard ratio of hospitalization for alcoholism in men and women with the slow alcohol degradation *ADH1B · 1/1* genotype was 3.9 (95% CI: 1.0–16) and 2.7 (95% CI: 0.4–20).

For *ADH1C*, odds for heavy and excessive alcohol drinking were 40–70% higher among men who were hetero- or homozygous for the slow alcohol degrading *ADH1C · 2* allele than among men who were homozygous for the fast alcohol degrading *ADH1C · 1* allele (Table 3). Similar results were found in women; however, effect sizes were slightly smaller and only statistically significant for heavy drinking. *ADH1C* genotype was not associated with alcoholism (Table 3).

Analyses on daily, heavy and excessive drinking and on alcoholism (MAST score and hospitalizations) were repeated excluding consistent nondrinkers, that is, participants who reported no alcohol intake at every examination in which they participated (7.5% of the total study population). This restriction did not affect any of our results (data not shown).

Because of linkage disequilibrium between the *ADH1B · 2* and *ADH1C · 1* alleles, and the relatively large effect on enzyme activity of the *ADH1B* polymorphism, our results for *ADH1C* could be influenced by *ADH1B* genotype. Therefore, *ADH1C* analyses were repeated solely on individuals who were *ADH1B · 1* homozygotes (95% of the study cohort): we found similar results, indicating that the effect of *ADH1C* genotype was independent of *ADH1B* genotype (data not shown).

We also performed analyses on genotype combinations, ranking genotypes in order of expected total enzyme

Table 1 Distribution of *ADH1B* and *ADH1C* genotypes

ADH1B	*ADH1C*					
	Men (n = *4039*)			*Women* (n = *5041*)		
	1/1 (fast)	*1/2 (intermediate)*	*2/2 (slow)*	*1/1 (fast)*	*1/2 (intermediate)*	*2/2 (slow)*
1/1 (slow)	1272	1882	697	1570	2347	907
1/2 (intermediate)	110	70	5	130	81	3
2/2 (fast)	2	1	0	3	0	0

Table 2 Associations between *ADH1B* genotype and alcohol drinking habits and alcoholism

ADH1B alcohol degradation	*Men* (n = *4039*)		*Women* (n = *5041*)	
	1/2+2/2 (n = *188*) *(fast)*	*1/1* (n = *3851*) *(slow)*	*1/2+2/2* (n = *217*) *(fast)*	*1/1* (n = *4824*) *(slow)*
Alcohol drinking habits				
Weekly alcohol intake (*n* = 3784 M, 4052 W)[a]	7.5 (6.4–8.7)	9.8 (9.1–11)	3.0 (2.6–3.5)	4.0 (3.7–4.3)
Any alcohol intake (*n* = 3784 M, 4052 W)[b]	1.0 (reference)	2.1 (1.0–4.5)	1.0 (reference)	3.1 (1.8–5.3)
Daily drinking (*n* = 2015 M, 1259 W)[b]	1.0 (reference)	2.5 (1.5–4.1)	1.0 (reference)	1.9 (1.1–3.5)
Heavy drinking (*n* = 1262 M, 814 W)[b]	1.0 (reference)	3.1 (1.7–5.7)	1.0 (reference)	3.0 (1.4–6.4)
Excessive drinking (*n* = 507 M, 353 W)[b]	1.0 (reference)	2.7 (1.1–6.5)	1.0 (reference)	4.2 (1.3–13)
Alcoholism				
bMAST score ⩾5 (*n* = 351 M, 105 W)[b]	1.0 (reference)	2.9 (1.3–6.6)	1.0 (reference)	1.7 (0.5–5.2)
bMAST score ⩾10 (*n* = 217 M, 55 W)[b]	1.0 (reference)	2.6 (1.0–7.1)	1.0 (reference)	2.8 (0.4–20)
Hospitalization (*n* = 167 M, 74 W)[c]	1.0 (reference)	3.9 (1.0–16)	1.0 (reference)	2.7 (0.4–20)

Abbreviations: bMAST, the brief Michigan Alcoholism Screening Test; M, men; W, women.
Heavy drinking was defined as drinking more than 21 drinks per week for men and 14 drinks per week for women, and excessive drinking as drinking more than 35 drinks per week for men and 21 drinks per week for women. *n* indicates the number of men and women who are cases in respective analyses. Adjustment was made for *ADH1C* genotype, age, years of school education and examination year.
[a]Shown numbers are mean number of drinks per week (95% CI), which is estimated among those who report any alcohol intake.
[b]Shown numbers are OR (95% CI).
[c]Shown numbers are hazard ratios (95% CI).

Table 3 Associations between *ADH1C* genotype and alcohol drinking habits and alcoholism

ADH1C alcohol degradation	*Men* (n = *4039*)			*Women* (n = *5041*)		
	1/1 (n = *1384*) *(fast)*	*1/2* (n = *1953*) *(intermediate)*	*2/2* (n = *702*) *(slow)*	*1/1* (n = *1703*) *(fast)*	*1/2* (n = *2428*) *(intermediate)*	*2/2* (n = *910*) *(slow)*
Alcohol drinking habits						
Weekly alcohol intake (*n* = 3784 M, 4052 W)[a]	9.8 (9.1–11)	10.4 (9.4–12)	10.5 (9.4–12)	4.0 (3.7–4.3)	4.1 (3.7–4.5)	4.1 (3.7–4.6)
Any alcohol intake (*n* = 3784 M, 4052 W)[b]	1.0 (reference)	1.3 (0.9–1.8)	0.7 (0.5–1.2)	1.0 (reference)	1.1 (0.9–1.4)	1.0 (0.7–1.4)
Daily drinking (*n* = 2015 M, 1259 W)[b]	1.0 (reference)	1.1 (0.9–1.4)	1.1 (0.8–1.5)	1.0 (reference)	1.2 (0.9–1.5)	1.2 (0.9–1.7)
Heavy drinking (*n* = 1262 M, 814 W)[b]	1.0 (reference)	1.4 (1.1–1.8)	1.4 (1.0–1.9)	1.0 (reference)	1.3 (1.0–1.7)	1.2 (0.8–1.7)
Excessive drinking (*n* = 507 M, 353 W)[b]	1.0 (reference)	1.6 (1.1–2.3)	1.7 (1.1–2.6)	1.0 (reference)	1.4 (0.9–2.1)	1.0 (0.6–1.6)
Alcoholism						
bMAST score ⩾5 (*n* = 1076 M, 597 W)[b]	1.0 (reference)	1.0 (0.8–1.3)	1.0 (0.7–1.4)	1.0 (reference)	0.8 (0.5–1.2)	0.8 (0.5–1.5)
bMAST score ⩾10 (*n* = 290 M, 65 W)[b]	1.0 (reference)	1.0 (0.7–1.3)	1.0 (0.7–1.6)	1.0 (reference)	0.6 (0.3–1.0)	0.7 (0.3–1.5)
Hospitalization (*n* = 209 M, 90 W)[c]	1.0 (reference)	1.2 (0.9–1.7)	0.9 (0.6–1.5)	1.0 (reference)	1.4 (0.8–2.6)	2.2 (1.2–4.2)

Abbreviations: bMAST, the brief Michigan Alcoholism Screening Test; M, men; W, women.
Heavy drinking was defined as drinking more than 21 drinks per week for men and 14 drinks per week for women, and excessive drinking as drinking more than 35 drinks per week for men and 21 drinks per week for women. *n* indicates the number of men and women who are cases in the respective analyses. Adjustment was made for *ADH1B* genotype, age, years of school education and examination year.
[a]Shown numbers are mean number of drinks per week (95% CI), which is estimated among those who report any alcohol intake.
[b]Shown numbers are OR (95% CI).
[c]Shown numbers are hazard ratios (95% CI).

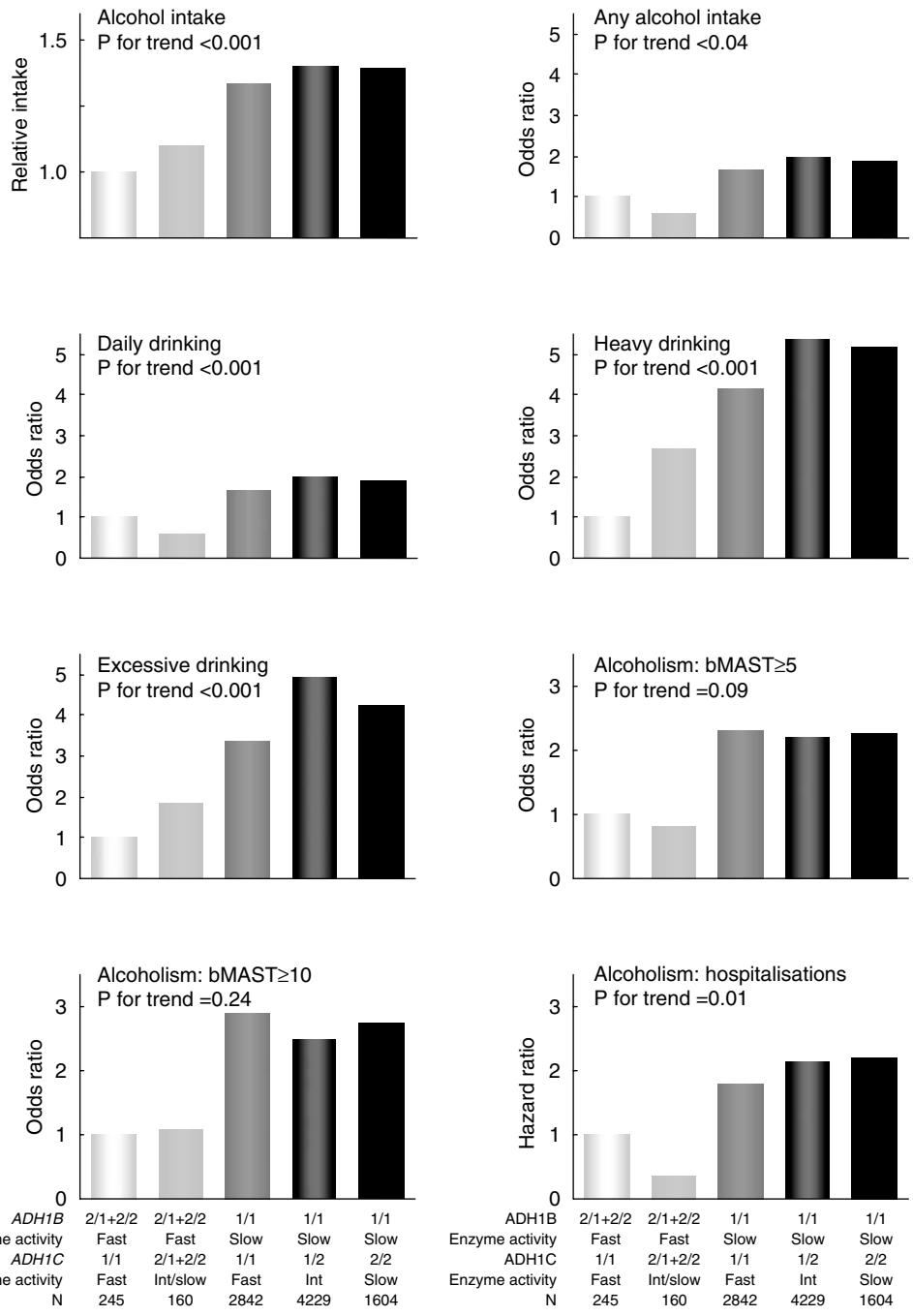

Figure 1 Association between the combined *ADH1B* and *ADH1C* genotypes and alcohol drinking habits and alcoholism. The combined *ADH1B* and *ADH1C* genotypes are ranked according to expected enzyme activity ((*ADH1B · 2/1 + ADH1B · 2/2*, *ADH1C · 1/1*) >(*ADH1B · 2/1 + ADH1B · 2/2*, *ADH1C · 2/1 + ADH1C · 2/2*) >(*ADH1B · 1/1*, *ADH1C · 1/1*) >(*ADH1B · 1/1*, *ADH1C · 1/2*) >(*ADH1B · 1/1*, *ADH1C · 2/2*)). *ADH1C · 1/2* and *ADH1C · 2/2* were combined because of few subjects in these categories among the *ADH1B · 2* hetero- and homozygotes. Analyses are for men and women combined and *P*-values are for linear trend tests.

activity, and tested for linear trend in each of the variables for alcohol drinking habits and alcoholism (Figure 1). For most end points, there was a statistically significant trend test in the expected direction: individuals with slow vs fast alcohol degradation drank more alcohol and more often, and had a higher risk of alcoholism.

ADH1B genotypes are very differently distributed among whites and East Asian populations (Figure 2). Among the white population, more than 90% carry the *ADH1B · 1/1* genotype, coding for slow alcohol degradation, whereas among the East Asian population less than 10% carry this genotype. Hence, the population attributable risk of heavy

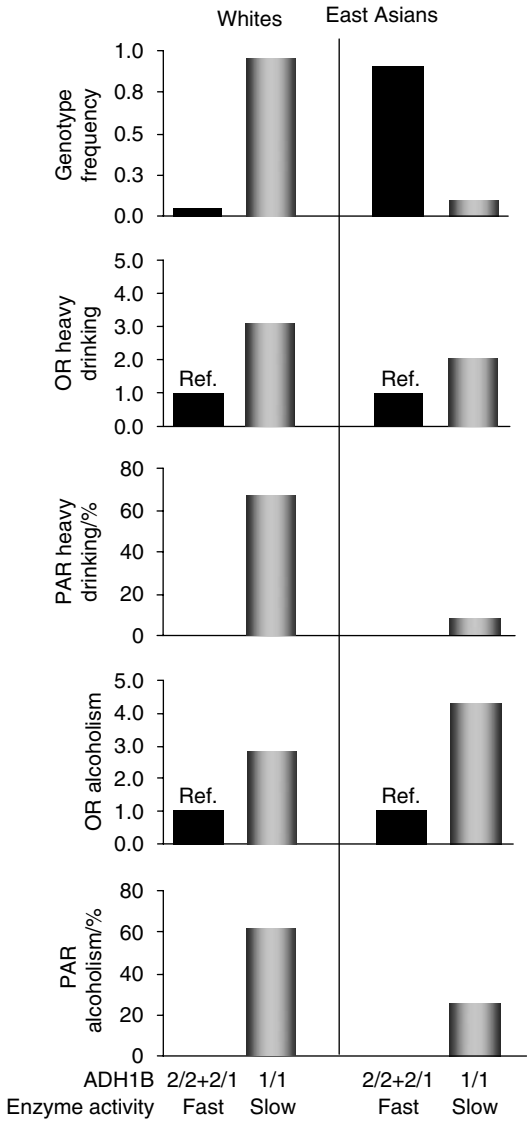

Figure 2 Genotype frequencies, OR and PARs of heavy drinking and alcoholism according to *ADH1B* genotype among white and East Asian populations. Slow alcohol degradation *ADH1B·1/1* genotype is predominant among Caucasians, but rare among East Asian population. Risks of heavy drinking and alcoholism according to *ADH1B·1/1* among white and East Asian populations are comparable, but because of the different genotype distributions in the two populations, PARs are much higher among white population. For East Asian population, genotype frequencies, OR and PARs are calculated from previous studies.[29,30] Ref, reference group; OR, odds ratio; PAR, population attributable risk.

drinking and alcoholism according to the *ADH1B·1/1* genotype is 67 and 62% among white population compared with 9 and 24% among the East Asian population.

Discussion

Our results suggest that *ADH1B* and *ADH1C* genotypes are associated with alcoholism and alcohol drinking habits. Men and women with *ADH1B* slow vs fast alcohol degradation drank more alcohol, were more often daily, heavy and excessive drinkers and had higher risks of alcoholism. Men and women with *ADH1C* intermediate and slow vs fast alcohol degradation were more often heavy and excessive drinkers. As expected, effect sizes were smaller for *ADH1C* than for *ADH1B*, but given the high frequency of the *ADH1C·2* allele, it is nevertheless a very interesting finding. Moreover, the impact of the *ADH1C·2* allele on cumulative lifetime alcohol intake may be significant. Our results also suggest that *ADH1B* and *ADH1C* genotypes may partly explain why white people generally drink more alcohol than East Asian. The population risk of heavy drinking and alcoholism attributable to the *ADH1B·1/1* genotype was 67 and 62% among white population and only 9 and 24% among the East Asian population.

Among men, we found relative estimates for alcoholism from two to three among *ADH1B·1* homozygotes, which is comparable to results from a recent meta-analysis consisting predominantly of East Asian studies.[29] Also, the odds ratio (OR) for heavy drinking for men was 3.1 which agrees with what is previously found among Asian men.[30] Separate estimates for women were not available for any of the end points in previous studies. Our results were remarkably similar for men and women indicating that the investigated associations are not sex-specific.

The various end points were probably subject to some misclassification, that is, sensitivity and/or specificity less than 100%. Measures of heavy and excessive drinking will be misclassified if amount of alcohol intake is under- or overreported, which it in many instances probably is. The MAST score is not a perfect screenings tool for alcoholism either. However, these errors occur most likely independently of genotype, and because end points are binary, will lead to bias toward the null.[31] Hence, we do not consider misclassification of end points to have caused our results. For alcoholism defined by hospital registry information, sensitivity is likely considerably less than 100% because many alcoholics are untreated or treated at private clinics that are not included in the national registers. However, specificity could be close to perfect; few nonalcoholics are presumably diagnosed as alcoholics. In this scenario, nondifferential misclassification is not affecting the hazard ratio.[32]

Never-drinkers have not been exposed to alcohol and hence, their drinking status cannot have been affected by *ADH1B* and *ADH1C* genotypes. It was not possible to separate never-drinkers from nondrinkers in this study, so performing analyses without never-drinkers was not an option. Potentially, this could have caused bias, but since excluding consistent nondrinkers from analyses had virtually no impact on our results, we do not consider this a major limitation.

We modeled the amount of alcohol intake in five different ways and alcoholism in three different ways. Hence, several statistical tests have been performed which in some instances call for caution. However, outcomes in this study were not independent but merely represent similar

outcomes modeled differently and we chose not to adjust for multiple comparisons.

A likely explanation for our findings is that differences in enzyme activity from the *ADH1B* and *ADH1C* polymorphisms result in intraindividual differences in alcohol degradation rate and that, for a given level of alcohol intake, individuals with fast alcohol degradation have higher levels of acetaldehyde and thus more unpleasant symptoms compared with individuals with slow alcohol degradation. However, an effect of the polymorphisms on alcohol degradation rate *in vivo* has been difficult to demonstrate,[33] which may have been due to insufficiently sensitive laboratory methods. In a more recent study which applied a more refined method for measuring the rate of alcohol degradation, results showed a significant difference in degradation rate according to the *ADH1B* genotype.[34] In further support of an *in vivo* effect of the *ADH1B* genotype is that individuals with the most active enzymes are consistently reported to experience more unpleasant symptoms such as flushing when drinking alcohol compared to individuals with the less active enzymatic forms.[35–38]

Our study had several strengths. First of all, sample size was large and provided adequate power to study associations between the relatively rare *ADH1B · 1/2* genotype and several end points, and to detect the small effect sizes associated with the *ADH1C* genotypes. Furthermore, participants were men and women from the general population, all of Danish descent. Hence, population stratification is unlikely to have affected our results. Alcohol drinking habits were described in several dimensions and information on alcoholism was obtained from two independent sources (questionnaire and hospital registry information). All end points were assessed independently from genotyping and participants were unaware of the purpose of this study when enrolled.

In conclusion, our data suggest that alcoholism and alcohol drinking habits are partly predictable from *ADH1B* and *ADH1C* genotypes. Results for men and women were comparable and, as expected, effects of *ADH1B* were larger than effects of *ADH1C*.

Methods

Study population

Our data originate from The Copenhagen City Heart Study, which is a series of studies conducted in the Danish general population. Examinations consisted of interview and physical examination, and more especially, blood was given for DNA purification at the examination that was performed during 1991–1994. All participants gave written consent and the ethics committee for Copenhagen and Frederiksberg approved the study (no. 100.2039/91). Enrolment and examination procedures have been described in more detail elsewhere.[39,40] Of the 17 180 individuals who were invited to the 1991–1994 examination, 10 135 participated, 9259 gave blood and 9222 were successfully genotyped. Eligibility criterion for participation was Danish citizenship and therefore, the Copenhagen City Heart Study does not reflect the ethnic admixture of Copenhagen (the proportion of inhabitants with foreign citizenship was 8% in 1994). However, even a few participants of foreign ethnicity could potentially confound our results since the fast alcohol degradation *ADH1B · 2* allele is rare among white population and quite frequent in other populations. Information on ethnicity was not assessed at the examinations, and hence information on birthplace was obtained from the Civil Registration System. Participants born in Asia, Africa, the Middle East, South America or Greenland were excluded from further study ($n = 211$). In all, 9080 individuals were eligible for analyses, some of whom also participated in the examinations during 1981–1983 ($n = 6615$) and during 2001–2003 ($n = 4684$).

Genotyping procedures

The *ADH1B · 2* allele (rs1229984, Arg47His in exon 3) and *ADH1C · 2* allele (rs698, Ile3490Val in exon 8) were identified by means of duplex polymerase chain reaction followed by Nanogen microelectronic chip technology (Nanogen NMW 1000 Nanochip Molecular Biology Workstation)[41] using standard conditions (details available from authors). In a validation study, the accuracy of the Nanogen method was found to be comparable to restriction fragment length polymorphism.[42]

End points

Questions on drinking habits were included in the questionnaire at the examinations during 1981–1983, 1991–1994 and 2001–2003. Amount of alcohol intake was reported as usual intake of weekly beers, wine and spirits. Assuming one drink to be equal to 12 g of pure alcohol, a measure of total weekly intake was calculated. We defined heavy drinking as drinking more than 21 drinks per week for men and 14 drinks per week for women, and excessive drinking as drinking more than 35 drinks per week for men and 21 drinks per week for women.[43] Participants were defined as daily drinkers if they reported to drink alcohol every day.

We defined alcoholism from questionnaire as well as from hospital discharge information. The former definition was taken from the 1991–1994 questionnaire, which included a screening test for alcoholism (10 question version of the brief MAST[44]). The test included questions such as 'Do you feel you are a normal drinker?' and 'Have you ever gone to anyone for help about your drinking?' Test scores of $\geqslant 5$ and of $\geqslant 10$ were used as dichotomized end points. Information on hospitalizations for alcoholism was obtained from the Danish Hospital Discharge Register where all hospitalizations in Denmark, classified according to the World Health Organization's International Classification of Diseases (ICD) are registered.[45] The following diagnoses indicative of hospitalization due to alcoholism were obtained: ICD-8 codes 303.09–303.99 and ICD-10 codes F10.1–F10.4.

Statistical analyses

All statistical models included *ADH1B* and *ADH1C* genotypes, age and years of school education using the SAS/Stat

software (version 8.02). *ADH1B·2* heterozygotes were combined with *ADH1B·2* homozygotes ($n=6$). Estimated haplotype frequencies were calculated by Hplus.[46,47] Linkage disequilibrium was expressed as r^2 and D'.[48,49]

To study the association between *ADH1B* and *ADH1C* genotypes and amount of alcohol intake, the correlated mixed distribution model was applied (Mixcorr macro[50]). This model handles data with clumping at zero and a lognormal distribution for nonzero values, and contains components to model the probability of a nonzero value and the mean of nonzero values, allowing for repeated measurements using random effects.[50] This means that if a variable affects the mean amount by affecting both the probability of occurrence of a nonzero value and also the mean of a nonzero value, these effects can be separated and quantified. Hence, two estimates are produced from this model: the OR for having a nonzero alcohol intake (that is, for not being a non-drinker) and the mean amount of alcohol intake among those with a nonzero intake.

To study the association between *ADH1B* and *ADH1C* genotypes and daily, heavy and excessive drinking, we applied logistic regression allowing for repeated measurements using random effects (proc nlmixed). We applied unconditional logistic regression to study associations between *ADH1B* and *ADH1C* genotypes and dichotomized brief MAST score (proc genmod).

Risk estimates for alcoholism defined from hospitalizations were computed by means of Cox proportional hazard regression (proc phreg). Age was used as the time axis and analyses were corrected for delayed entry. Vital status of the participants was obtained from the National Central Person Register. The observation time for each participant was the period from participation in the Copenhagen City Heart Study, until date of alcoholism, death, emigration outside Denmark or January 1, 2004, whichever came first. We had 100% follow-up.

Population attributable risk was calculated as (proportion of exposed in the population $\cdot$ (OR$-$1))/(proportion of exposed in the population $\cdot$ (OR$-$1) $+$ 1).[51]

Abbreviations

ADH	*alcohol dehydrogenase*
bMAST	*brief Michigan Alcoholism Screening Test*
OR	*odds ratio*
PAR	*population attributable risk*
Ref	*Reference group*

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Duality of interest

None declared.

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