Genetic Influences on Alcohol Consumption Patterns and Problem Drinking: Results from the Australian NH&MRC Twin Panel Follow-up Survey

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The role of genetic factors in the etiology of alcoholism, particularly alcoholism in women, remains controversial. Findings consistent with an important genetic influence in males on alcoholism risk, or other measures of alcohol problems, have come from studies of adoptees,1-4 half-siblings,5 and monozygotic and dizygotic twin pairs.6-13 Negative findings or findings suggesting at most a modest genetic contribution to male alcoholism risk have emerged from three adoption samples14 (see also Cadoret, this volume) and three twin samples.15-17 Whilst the majority of these studies supports a genetic contribution to male alcoholism risk, the extent to which inconsistencies between studies yielding positive and negative results can be explained by differences in sociocultural environment, differences in assessment procedure (e.g., reliance on official records of hospitalization or temperance board contact versus medical records versus self-report questionnaire versus structured psychiatric interviews), differences in sample ascertainment procedure (epidemiologic sampling versus ascertainment through probands identified through treatment facilities or official records), or merely differences in sample size and consequent statistical power remains uncertain.

For alcoholism in females, findings have been even less consistent than those in males. An early consensus emerged that genetic factors were less important in females than in males, supported by the relatively weak evidence for a genetic influence on female alcoholism risk both from adoption studies18,19 and from twin studies.9-11 A recent interview study of an epidemiologic sample of young adult female twin pairs, however, yielded heritability estimates, varying as a function of narrowness or breadth of diagnostic criteria, in the range of 50–60%.20 A mailed questionnaire assessment of problem drinking by an older, predominantly female, twin panel, likewise found evidence for substantial genetic influence in females.13,21 Once again, there are many possible reasons for these inconsistencies; however, findings indicating a substantial heritability in females have been restricted to those studies which used an epidemiologic sampling strategy, rather than identifying alcoholic probands through treatment facilities or official records.

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In contrast to these findings for alcoholism risk, surveys of drinking practices in general population twin samples, unselected for alcoholism risk, have consistently found evidence for a genetic contribution to individual differences in drinking pattern (average quantity consumed per drinking occasion) in both males and females. Self-report drinking practices assessed in general community (rather than treatment) samples have good reliability and validity and, at least beyond age 25, considerable longitudinal stability. Twin studies of alcohol consumption patterns in the United States, Scandinavia (Finland, Sweden, Australia, and the United Kingdom) have all yielded results consistent with a substantial genetic influence on alcohol consumption patterns in both sexes, with estimates of heritability (i.e., proportion of variation in alcohol consumption levels attributable to genetic factors) in most studies falling in the range of 30–60%. Prospective studies indicate that a substantial proportion of the longitudinally stable component of variation in drinking practices may be determined by genetic factors. In contrast to findings for alcoholism, studies from Finland, Sweden, and Australia, which used sample sizes sufficiently large to give good statistical power for resolving sex differences in the genetic and environmental determinants of drinking practices, yielded heritability estimates that were at least as high in females as in males.

Given the robust evidence for genetic influences on alcohol consumption patterns, it is natural to question whether these genetic influences on consumption can explain at least part of the genetic variation in alcoholism risk. If genetic factors play a major role in determining an individual’s “alcohol consumption set-point,” the level of consumption at which an individual’s drinking practices will settle by age 25, it seems likely that because of genetic influences, some individuals will maintain a level of consumption too low to put them at risk of drinking problems, whilst other individuals will maintain consumption levels that may put them at much higher risk. High risk studies comparing adult offspring of alcoholics and controls in an alcohol challenge paradigm have found differences in such measures as subjective reactions to alcohol and objective measures of increase in body sway, after a standard body-weight adjusted dose of alcohol. Data from the Australian alcohol challenge twin study confirm a significant genetic contribution to differences in performance after alcohol challenge, but they also show a significant and substantial correlation between genetic effects on alcohol challenge performance and genetic effects on normal variation in alcohol consumption patterns. It would therefore be surprising if genetic influences on consumption were not also having an influence on risk of alcohol problems. In the present paper, we attempt to address more directly the question of whether genetic influences on alcohol consumption pattern can explain in part genetic influences on alcoholism risk, using self-report data on consumption and history of problem drinking obtained in the 1988–1989 follow-up survey of the Australian National Health and Medical Research Council twin panel.

**METHOD**

**Sample**

The Australian National Health and Medical Research Council (NH&MRC) twin panel is a volunteer twin panel, recruited through the media, schools, and a variety of other sources. In 1980–1981, a mailed questionnaire survey of adult twin pairs enrolled on the panel was conducted, questionnaires being mailed to 5,967 twin pairs
aged 18 or older. Completed questionnaires were received from both members of 3,808 twin pairs (64% pairwise response rate) and from one twin only from an additional 576 pairs (69% individual response rate). The median age of respondents was 30. The questionnaire contained questions about personality, health, and lifestyle, including self-report drinking patterns, but no questions about alcohol-related problems. Although a volunteer sample, the "1981 survey" sample consists of individuals from a broad range of socioeconomic backgrounds. Among twins who returned both questionnaires, 33% of the female respondents and 19% of the male respondents had completed 10 or fewer years of education, and 27.1% and 23.7%, respectively, reported unskilled occupations. Eight percent of female respondents and 20% of male respondents were university graduates. Self-report drinking practices of this sample were broadly comparable to figures reported for the general population of Australia by the Australian Bureau of Statistics, modestly lower for male respondents and higher for female respondents.

As is commonly found in twin studies, however, female twins and monozygotic (Mz) twins were over-represented in the sample; 1,232 MZ female, 567 MZ male, 747 DZ female, 350 DZ male, and 912 unlike-sex twin pairs returned questionnaires.

In 1988–1989, a three-phase follow-up survey of the 1981 survey sample was begun, to explore the changes in drinking practices and history of alcohol problems in this sample. A follow-up questionnaire was mailed to all 3,808 complete twin pairs participating in the 1981 survey who were still living and for whom a current address could be traced. Twins who did not return a mailed questionnaire were given the option of an abbreviated telephone interview. In subsequent phases of the follow-up (still in progress), twins receive a telephone screening interview to identify pairs in which at least one twin has a history of alcohol problems, for more intensive follow-up by in-person interview. In the first phase, mailed questionnaire or abbreviated telephone interview data were obtained from both members of 2,997 twin pairs (82% pairwise response rate if we exclude from the target sample those 139 twin pairs in which one or both twins were deceased by the time of follow-up). The same self-report questionnaire was remailed in 1990 to 500 male and 500 female twin individuals who had returned follow-up questionnaires, to provide 2-year test-retest data, with completed returns being returned by 427 male and 442 female respondents. Data on self-report drinking practices and drinking problems from this first questionnaire phase are analyzed in this paper. Inasmuch as data from lifetime abstainers were excluded, as were data from twins whose cotwin either had not responded or had omitted to answer either problem-drinking or alcohol consumption questions, final sample sizes for the analyses presented in this paper were: 726 MZ female pairs, 406 DZ female pairs, 321 MZ male pairs, 178 DZ male pairs, and 454 unlike-sex pairs.

Measures

Drinking problems were assessed using items selected from a self-report questionnaire based on Feighner criteria for alcoholism, which had been used for a national survey in the United States, with the original hope of improving discrimination between type I and type II alcoholics. Questions about alcohol withdrawal symptoms and alcoholic blackouts were not used because of concern about whether respondents would understand these items when given in a self-report questionnaire. Three items relating to self-perceived excessive consumption of alcohol, objections about one's drinking from others, and guilty feelings about one's
drinking appeared to have low specificity when used to assess problem drinking in this sample, particularly in female respondents, and these items are not used in the analyses presented here. Table 1 (in Results section) summarizes the items used to assess alcohol problems and the corresponding Feighner group for each item. Respondents were asked to indicate whether they had ever experienced any of these problems, and if so, whether the problem had occurred during each of the following time periods: (1) last 12 months; (2) 1–3 years ago; (3) 4–6 years ago; and (4) 7 or more years ago (which would overlap with the time of the 1981 mailing). For each time period the number of reported problems was summed, and a lifetime problem-drinking score was computed as the maximum score from any time period. Whilst a self-report questionnaire format does not allow detailed probing to permit accurate identification of symptoms that occurred together in time, the scoring procedure used at least provides some limited information about clustering of symptoms in the same period of the respondent’s life. Self-report average weekly alcohol consumption was assessed by a single question which asked respondents to rate their own typical weekly consumption on a 9-point scale, with response categories (1) none, (2) 1–3 drinks, (3) 4–6 drinks, (4) 7–12 drinks, (5) 13–18 drinks, (6) 19–24 drinks, (7) 25–42 drinks, (8) 43–70 drinks, and (9) 70+ drinks per week, with one drink defined as “a can/stubby of beer, a glass of wine or nip of spirits.”

Data Analyses

Initial exploratory analyses of the psychometric properties of the problem-drinking scale (computation of Cronbach’s alpha45; principal factor analysis46) ignored the twin structure of our data, that is, the nonindependence of observations on twin pairs. Under random sampling (which we assume is at least approximately achieved here), this will still lead to unbiased estimates of population parameters, although the sampling variance of those parameters will be underestimated. Exploratory analyses also used product-moment correlations between items, ignoring the complication that these were dichotomous and their joint distribution therefore highly non-normal. As a consequence, estimates of factor loadings and Cronbach’s alpha will be lower than would have been the case had we used tetrachoric correlations, but they will be more comparable to publications on other scales in which a similar approach was typically used. For these exploratory analyses, alcohol problem items were recoded as lifetime presence or absence of symptoms, without regard for clustering of symptoms in the same period of the respondent’s life. In computing 2-year test-retest correlations for average weekly consumption and maximum lifetime problem score, polychoric correlations were used.47,48

For genetic analyses, we computed 4 × 4 matrices of polychoric correlations between problem-drinking and average weekly alcohol consumption scores of first and second twins, separately for each zygosity group. Genetic models were fitted by asymptotic weighted least squares using LISREL.46–53 We fitted a general bivariate genetic model,51,54 allowing for sex-dependent common factor genetic, shared environmental and within-family environmental effects on both weekly consumption and history of problems as well as residual or specific-factor genetic and environmental effects on problem drinking. Parameters of the full bivariate genetic model53,54 are summarized in Figure 1. In the most general model, separate parameters were estimated for males and females (distinguished by subscripts m and f). We also fitted submodels of the general bivariate genetic model, deleting one or more parameters of the full model (e.g., fixing the genetic common factor effect on problems to
**COMMON-FACTOR** EFFECTS

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<tr>
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**SPECIFIC FACTOR** EFFECTS

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**CONSUMPTION**

**PROBLEMS**

FIGURE 1. Bivariate genetic path model\(^{53,54}\) for genetic and environmental contributions to the variation and covariation of alcohol consumption levels and problem drinking. \(E_C, C_C,\) and \(A_c\) denote within-family environmental, shared environmental, and additive genetic common factor effects, which influence both consumption and problems (paths \(e, c,\) and \(h\) for consumption, \(e', c',\) and \(h'\) for problems); and \(E_S, C_S,\) and \(A_s\) denote residual or specific-factor within-family environmental, shared environmental, and additive genetic effects on problems (paths \(e'', c'',\) and \(h''\), respectively).

**zero \([h'=0]\),** to test the hypothesis that there is no correlation between genetic effects on consumption and genetic effects on problem drinking, or fixing the genetic specific factor effect on problems to zero \([h''=0]\), to test the hypothesis that the same genes that determine level of consumption also determine risk of problem drinking.

For each model we obtained weighted least squares estimates of model parameters as well as an overall chi-square test of the goodness-of-fit of the model.\(^{50,51,53}\) The overall goodness-of-fit of nested models was compared by likelihood-ratio chi-square ("chi-square difference") test, with number of degrees of freedom equal to the number of free parameters of the more general model fixed to zero in the full model.

Fitting a bivariate genetic model allowed us to estimate the correlation between genetic effects on problem drinking and genetic effects on alcohol consumption level, estimated as

\[
\rho_g = h' h'' \left( h''^2 + h^2 \right)^{-1},
\]

and to estimate in similar fashion correlations between shared environmental effects on consumption level and on problem drinking and correlations between within-family environmental effects on consumption level and on problem drinking. However, since we were analyzing current drinking patterns, but lifetime history of problem drinking, significant and substantial genetic and environmental correlations might arise because of a causal effect of problem drinking on alcohol consumption pattern. To test this hypothesis, we also fitted strong causal models (FIG. 2) which assumed that a positive genetic correlation between consumption and problem drinking arises because of the causal influence of problem drinking on consumption pattern \((i_p>0)\) or because of the causal influence of consumption pattern on problem drinking \((i_p>0)\). Parameters of the most general reciprocal causation submodel are summarized in FIGURE 2. We have shown elsewhere\(^{53,54}\) that the strong unidirectional causation and reciprocal causation models are a submodel of the general bivariate genetic model of FIGURE 1.
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**FIGURE 2.** Reciprocal causation path model allowing for the reciprocal causal influence of alcohol consumption level on problem drinking and of problem drinking on consumption pattern. ALC and PROB denote true phenotypes for consumption level and for problem drinking, with reciprocal causal paths $i_p$ and $i_s$ denoting the causal influence of consumption on problems, and of problems on consumption, respectively; and CONSUMPTION and PROBLEMS denote the corresponding observed variables, with error variances $\theta_c$ and $\theta_p$. Variables $E_A$, $C_A$, and $A_A$ denote direct within-family environmental, shared environmental, and additive genetic effects on consumption (i.e., excluding indirect effects mediated through the causal influence of problems on consumption), with corresponding paths $e_a$, $c_a$, and $h_a$; and $E_p$, $C_p$, and $A_p$ denote direct within-family environmental, shared environmental, and additive genetic effects on alcohol problems, excluding those effects mediated through the causal influence of consumption on problems.

**RESULTS**

Table 1 summarizes factor loadings under a one-factor model, estimated separately for males and for females, for items of the problem-drinking scale. In females ($n = 3,426$), the item “Drinks in morning to get rid of hangover” had only a modest factor loading (0.21), but loadings of all other items were substantial, ranging from a high of 0.75 (“treated for a drinking problem”) to a low of 0.53 (adverse effects of drinking on health). In males ($n = 1,819$), several items had modest loadings of about 0.4 (“drunk driving”; “treated for a drinking problem”; “drinks in the morning to get rid of a hangover”), but the remaining items had substantial loadings in the range of 0.5–0.6. In two-factor solutions, the second factor in females had moderately high loadings only on the two items relating to cutting down on drinking, and the second factor in males had no clear interpretation, implying that the scale used is assessing a single global factor of alcohol-related problems. Internal consistency was high in both sexes: Cronbach’s alpha for lifetime problems was 0.87 in females and 0.83 in males; and for current problems, that is, problems during the preceding 12 months, was 0.92 in females and 0.84 in males. In the reliability substudy, 2-year test-retest correlations for lifetime total problems were also high, with polychoric correlations of 0.72 in females ($n = 373$) and 0.85 in males ($n = 350$) being obtained. Thus, psychometric properties of the problem-drinking scale appear satisfactory. The test-retest correlation for average weekly consumption was 0.83 in females ($n = 414$) and 0.84 in males ($n = 408$).
TABLE 1. Factor Loadings and Endorsement Frequencies of Items of the Problem-Drinking Scale: Items were Recorded as Lifetime Presence or Absence of Problems

| Feighner Group | Item Content                          | Factor Loadings | Endorsement Frequency (%) |
|               |                                     | Females | Males | Females | Males |
| A             | Adverse effects on health            | 0.53    | 0.54  | 4.8     | 11.5 |
| A             | Binge drinking                       | 0.70    | 0.57  | 1.0     | 7.2  |
| A             | Neglected responsibilities while drinking | 0.68    | 0.61  | 1.9     | 9.0  |
| B             | Inability to cut down on drinking    | 0.59    | 0.56  | 2.2     | 6.0  |
| B             | Failure to stick to plan to stop drinking | 0.57    | 0.57  | 3.2     | 6.5  |
| B             | Drinks in morning to get rid of hangover | 0.21    | 0.39  | 1.5     | 8.8  |
| C             | Adverse effects on work/employment opportunities | 0.73    | 0.63  | 0.9     | 4.9  |
| C             | Physical fights while drinking        | 0.61    | 0.50  | 1.3     | 8.4  |
| C             | Drunk driving                         | 0.54    | 0.38  | 1.5     | 11.0 |
| D             | Adverse effects on friendships/social life | 0.65    | 0.65  | 2.5     | 11.1 |
| D             | Adverse effects on marriage/home life | 0.65    | 0.60  | 2.4     | 11.4 |
| —              | Treatment for drinking problem        | 0.75    | 0.41  | 0.7     | 1.7  |

Also shown in Table 1 are endorsement frequencies for the individual items of the problem-drinking scale. These emphasize that the Australian twin sample is a general community sample, in which only a small minority of individuals have received treatment for alcohol problems (0.7% of women, 1.7% in men) and in which the most common alcohol problems relate to adverse social and health consequences of the respondent’s drinking and (particularly in men) drunk driving.

Table 2 gives the 4 x 4 matrices of polychoric correlations between lifetime problem-drinking score and current consumption pattern of first and second twins from each zygosity group. In female like-sex twin pairs, twin pair correlations for average weekly alcohol consumption and for reported history of drinking problems are approximately twice as high in monozygotic as in dizygotic twin pairs (0.59 vs. 0.28 and 0.43 vs. 0.24), consistent with important additive genetic influences on both consumption and problem drinking. Furthermore, cross-correlations between problem drinking by one twin and alcohol consumption patterns of the cotwin are on average substantially higher in monozygotic than dizygotic pairs, implying a genetic contribution to the covariance of consumption pattern and problem drinking. In male like-sex pairs, for whom sample sizes are much smaller, monozygotic and dizygotic correlations for problem drinking are approximately equal in magnitude. However, before we interpret this as evidence against a genetic influence on problem drinking in Australian males, two complications must be noted. A significant correlation is observed between opposite-sex pairs for problem drinking (0.31), instead of the zero correlation which would be predicted if there was a genetic but no shared environmental influence on female problem drinking, and a shared environmental but no genetic influence on male problem drinking. Furthermore, a higher MZ than DZ male like-sex correlation is observed for current consumption, and substantially higher MZ than DZ cross-correlations between current consumption by one twin and problem drinking by the cotwin, suggesting that there may in fact be genetic influences on problem drinking by males, and that our failure to observe higher MZ than DZ male like-sex correlations for problem drinking may be a consequence of sampling variability, given the rather small sample sizes for the male like-sex groups.
### TABLE 2. Twin Pair Correlations for Current Average Weekly Alcohol Consumption and Lifetime Problem Drinking Score

<table>
<thead>
<tr>
<th></th>
<th>MZ Female Pairs</th>
<th>DZ Female Pairs</th>
<th>MZ Male Pairs</th>
<th>DZ Male Pairs</th>
<th>DZ Unlike-Sex Pairs</th>
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<tr>
<td></td>
<td>(n = 726)</td>
<td></td>
<td>(n = 406)</td>
<td></td>
<td>(n = 454)</td>
</tr>
<tr>
<td></td>
<td>I</td>
<td>II</td>
<td>III</td>
<td>IV</td>
<td>I</td>
</tr>
<tr>
<td>I. Problem drinking—twin A</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td>1.00</td>
</tr>
<tr>
<td>II. Current consumption—twin A</td>
<td>0.43</td>
<td>1.00</td>
<td></td>
<td></td>
<td>0.22</td>
</tr>
<tr>
<td>III. Problem drinking—twin B</td>
<td>0.43</td>
<td>0.19</td>
<td>1.00</td>
<td></td>
<td>-0.02</td>
</tr>
<tr>
<td>IV. Current consumption—twin B</td>
<td>0.25</td>
<td>0.59</td>
<td>0.18</td>
<td>1.00</td>
<td>0.25</td>
</tr>
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</table>

Results of fitting bivariate genetic models to the alcohol consumption and problem-drinking data are summarized in TABLE 3. The full model allowing for sex-dependent parameters gives an acceptable fit to the data ($p = 0.25$). Models which specify no genetic influences on consumption (model 2) or no common factor genetic influences on problems (model 4) are rejected by chi-square test of goodness-of-fit. Models which specify no shared environmental effects on consumption (model 3), no specific factor shared environmental effects on problems (model 6), no specific factor genetic effects on problems (model 5), or neither specific factor genetic nor shared environmental effects (model 7) all give acceptable fits to the data, and fits which are not significantly worse than that of the full model, by likelihood-ratio chi-squares test ($\chi^2 < 2.5$ in all cases). However, a model which specifies no shared environmental effects on problems or consumption and no specific factor genetic effects on problems is rejected by chi-square test of goodness of fit (model 8: $p < 0.001$). We must retain in the model either specific factor genetic effects (model 9) or specific factor shared environmental effects (model 10), with the latter model giving a slightly (though not significantly) better fit. Constraining the parameters of model 9 or model 10 to be the same in both sexes in each case leads to a significant worsening of fit, by likelihood-ratio chi-square test (model 9 vs 11: $\chi^2 = 9.47$, d.f. = 4, $p = 0.05$; model 10 vs 12: $\chi^2 = 9.79$, d.f. = 4, $p < 0.05$).

**FIGURE 3** summarizes parameter estimates under models 9 and 10, the two best-fitting models. Model 9 yields a total heritability for problem drinking of 44%...
TABLE 3. Results of Fitting Bivariate Genetic Models to Twin Pair Correlation Matrices for Problem Drinking and Alcohol Consumption Patterns

<table>
<thead>
<tr>
<th>Model Description</th>
<th>Goodness-of-Fit</th>
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<tbody>
<tr>
<td></td>
<td>d.f.</td>
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<tr>
<td>1. Full sex-dependent bivariate genetic model</td>
<td>16</td>
</tr>
<tr>
<td>2. No genetic effects on consumption ($hm=hm'=hf=hf'=0$)</td>
<td>20</td>
</tr>
<tr>
<td>3. No shared environmental effects on consumption ($cm=cm'=cf=cf'=0$)</td>
<td>20</td>
</tr>
<tr>
<td>4. No specific factor genetic effects on problems ($hm'=hf'=0$)</td>
<td>18</td>
</tr>
<tr>
<td>5. No specific factor genetic effects on problems ($hm'=hf'=0$)</td>
<td>18</td>
</tr>
<tr>
<td>6. No specific factor shared environmental effects on problems ($cm'=cf'=0$)</td>
<td>18</td>
</tr>
<tr>
<td>7. No specific factor genetic or shared environmental effects ($hm'=hf'=cm'=cf'=0$)</td>
<td>20</td>
</tr>
<tr>
<td>8. Model 1 with $hm'=hf'=cm=cm'=cf=cf'=0$</td>
<td>24</td>
</tr>
<tr>
<td>9. No shared environmental effects ($cm=cm'=cf=cf'=0$)</td>
<td>22</td>
</tr>
<tr>
<td>10. Model 1 with $hm'=hf'=cm=cm'=cf=cf'=0$</td>
<td>22</td>
</tr>
<tr>
<td>11. Model 9 with no sex-dependent parameters</td>
<td>26</td>
</tr>
<tr>
<td>12. Model 10 with no sex-dependent parameters</td>
<td>26</td>
</tr>
</tbody>
</table>

in females, 50% in males; a heritability for average weekly alcohol consumption of 58% in females, 45% in males; and a genetic correlation (i.e., correlation between genetic effects on risk of problem drinking and genetic effects on alcohol consumption) of 0.42 in females, 0.45 in males. Under model 10, heritability estimates for average weekly alcohol consumption remain the same, but the heritability of risk of problem drinking is reduced to 8% in females, 10% in males, with shared environmental effects accounting for an additional 29% and 35% of the variance, respectively. Under model 10, there is a perfect correlation between genes which affect consumption and genes which determine risk of problem drinking, because there are no specific factor genetic effects on consumption.

When we fitted unidirectional causation models (FIG. 2), to test whether the significant genetic correlations observed between problem-drinking history and current consumption levels could be explained by the effects of problem drinking on consumption pattern rather than of consumption pattern on problem drinking, results were rather mixed. When we fixed $h_{pm} = h_{pf} = 0$ (for comparison with model 10), and instead estimated causal parameters $i_{pm}$ and $i_{pf}$ representing the causal influence of problem drinking on consumption patterns, this model gave a much worse fit to the data ($\chi^2 = 31.89$, d.f. = 24, $p = 0.13$), and this fit was little improved by estimating male and female error variances for problem drinking as free parameters ($\chi^2 = 31.36$, d.f. = 22, $p = 0.09$). However, if instead we allowed for a causal influence of consumption pattern on risk of problem drinking (i.e., $i_{pm} = i_{pf} = 0$ and $i_{mp}, i_{mf} > 0$), this model gave an excellent fit to the data ($\chi^2 = 24.34$, d.f. = 24, $p = 0.44$), consistent with the hypothesis that genetically determined differences in drinking style are contributing to risk of problem drinking. However, when we tested direction-of-causation models using the assumptions about mode of inheritance of model 9 (i.e., $h_{pm}, h_{pf} > 0$), there was no power to resolve alternative causal hypotheses. For
example, under this model, the hypothesis that problem-drinking history is affecting current consumption pattern yielded a chi-square of 27.67, and the alternative hypothesis that consumption pattern is influencing risk of problem drinking yielded an almost identical chi-square of 27.58.

**DISCUSSION**

We have analyzed self-report questionnaire data on history of alcohol problems and current alcohol consumption patterns obtained in a follow-up survey of the Australian NH&MRC twin register, to explore the relation between genetic influences on consumption and genetic influences on risk of problems. Our measure of problem drinking, based on Feighner criteria, had good psychometric properties, though the much higher loadings of items such as “treatment for a drinking problem” in females than in males is consistent with the possibility that the scale is a better measure of alcohol problems in women (the primary focus of our study) than in men. Model-fitting confirmed significant genetic influences on both consumption and problem drinking. However, whilst estimates of the heritability of average weekly consumption were consistent across the two best-fitting models (58% in females, 45% in males), estimates of the heritability of alcohol problems, though significantly different from zero, differed markedly according to whether we allowed for specific-factor genetic effects on problems (model 9: heritability estimates of 44% in females, 50% in males) or specific-factor shared environmental effects on problems (model 10: 8% in females, 10% in males). The imprecision of our heritability estimates for alcohol problems is undoubtedly a consequence of the reduced power for resolving genetic and shared environmental effects on dichotomous variables, particularly
when the degree of familial resemblance is relatively modest. As interview data become available, allowing for assessment of tolerance and withdrawal, we anticipate that higher twin-pair correlations will be observed, allowing for a much more powerful resolution of genetic and nongenetic effects.

Interpretation of our results is highly model dependent. Under model 10, which yielded a very modest heritability for alcohol problems in both sexes, all of the genetic variance in risk for alcohol problems could be explained by genetic factors that also influenced alcohol consumption patterns. Under model 9, which yielded more substantial heritability estimates for alcohol problems, genetic correlations were more modest (0.42 in females, 0.45 in males), though significantly different from zero. Furthermore, under model 9 we could not exclude the possibility that these genetic correlations were the consequences of the influence of history of drinking problems on current consumption patterns. Inclusion of baseline data on alcohol consumption patterns from the 1981 survey, and interview data on history of alcoholism from phases two and three of the follow-up survey, may allow these ambiguities to be resolved with considerably greater statistical power.

SUMMARY

Self-report questionnaire data from 3,000 adult twin pairs participating in the 1988–1989 follow-up survey of the Australian NH&MRC twin panel were analyzed to determine (1) the contribution of genetic factors to risk of problem drinking in males and females; and (2) the magnitude of the correlation between genetic effects on problem drinking and genetic effects on alcohol consumption level. Significant genetic contributions were found both for average weekly consumption of alcohol and for problem-drinking history. For level of consumption, genetic factors accounted for approximately 58% of the variation in females and 45% of the variation in males. Heritability estimates for problem drinking, though significantly greater than zero, were variable in magnitude, ranging (under different models) from 8–44% in females and 10–50% in males. Likewise, estimates of the magnitude of the genetic correlation, whilst in all cases significantly greater than zero, ranged from 0.42–1.00 in females and 0.45–1.00 in males under different models.

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