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The Inheritance of Alcohol Consumption Patterns in a General Population Twin Sample: II. Determinants of Consumption Frequency and Quantity Consumed*

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ABSTRACT. Genetic models were fitted to self-report data on frequency of alcohol consumption and average quantity consumed when drinking, from 3,810 adult Australian twin pairs. Frequency of consumption is determined both by an abstinence dimension, which is strongly influenced by shared environmental effects but not by genetic effects, and by an independent frequency dimension, which is influenced by genetic effects in both sexes and possibly by shared environmental affects in men. Quantity of alcohol consumed is likewise determined by an environmental abstinence dimension and by an independent and partly heritable quantity dimension. The best-fitting model allowed for two routes to abstinence: those who were not abstainers by virtue of their position on the abstinence dimension could nonetheless become abstainers by their position on the second, frequency (or quantity) dimension. Heritability estimates were 66% in women and 42–75% in men, for frequency; and 57% in women and 24–61% in men, for quantity. (*J. Stud. Alcohol* **52:** 425–433, 1991)

COMPARATIVELY little is known about the influence of familial factors on alcohol consumption patterns. General population surveys of adult drinking practices have only rarely obtained information about consumption patterns of family members (Cahalan et al., 1969; Edwards et al., 1972). Twin studies have usually reported a genetic influence, although some studies have found the importance of genetic effects to vary with sex or age cohort, and several have suggested that the social environment may also have an important impact (see Heath et al., 1989a). An important area of uncertainty concerns the inheritance of different components of drinking behavior. Previous studies have not clearly addressed the question of whether family resemblance for abstinence or for frequency and quantity parameters (Cahalan and Cisin, 1968a,b; Knupfer, 1966; Straus and Bacon, 1953) is best explained by inheritance of a single continuum of consumption or by separate inheritance of abstinence, frequency and quantity.

In a previous article (Heath et al., 1991), we applied nonmetric multidimensional scaling to twin quantity/frequency/abstinence data. The results suggested separate determination of abstinence, frequency and quantity of alcohol consumption, but we cautioned that without parametric model-fitting analyses we could not reject alternative possibilities. In this article, we report the results of applying model-fitting methods to the same data in an attempt to confirm whether inheritance of abstinence is separate from that of quantity and frequency dimensions.

Method

Sample and measures

Subjects were 3,810 adult twin pairs, who were enrolled on the Australian National Health and Medical Research Council Twin Register and who had both completed and returned a health questionnaire. Since there was reason to believe that the genetic and environmental determinants of alcohol consumption pattern might interact with cohort (Jardine and Martin, 1984; Reich et al., 1988), twin pairs were subdivided into those pairs aged 30 years and under (young cohort) and those pairs aged over 30 years (older cohort). The breakdown of the sample by zygosity and age cohort is shown in Table 1. Further details of the sample are given elsewhere (Heath et al., 1989a, 1991; Jardine, 1985; Jardine and Martin, 1984).

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TABLE 1. Breakdown of sample by age cohort and zygosity group

Twin group	Young cohort (≤ 30)	Older cohort (> 30)
Monozygotic female	570	663
Monozygotic male	274	293
Dizygotic female	351	400
Dizygotic male	206	146
Opposite-sex dizygotic pair	510	397

Included in the questionnaire was an item about abstinence from alcohol use ("Have you EVER taken alcoholic drinks?") and items about average frequency of consumption ("Over the last year, about how often have you usually taken any alcoholic drinks?") and quantity consumed when drinking ("On average, how many GLASSES would you drink on each day that you take some alcohol?"). As in the previous article (Heath et al., 1991), we considered only quantity consumed on week-ends, since many respondents reported little or no weekday consumption, and rescaled this as a discontinuous variable. Life-long abstainers, and those drinking less than once a month, were included as the sixth and final category for both frequency and quantity variables. Other response categories are listed in Table 2. The format of the alcohol-related items in the questionnaire is reproduced in Jardine and Martin (1984).

Genetic and environmental models

Models were fitted separately to the frequency data and to the quantity data. We compared the results of fitting three different types of model, each making different assumptions about the relationship between the determinants of abstinence and the determinants of frequency of consumption (or quantity consumed) in those who were drinkers. We give a nontechnical overview of these models here, leaving a technical presentation for the model-fitting section. Critical assumptions of the three different models are summarized schematically, for quantity consumed, in Figure 1.

The single liability dimension (SLD) model (Eaves and Eysenck, 1980; Eaves et al., 1978) postulated that there is a single liability continuum that determines quantity or frequency of consumption (including abstinence), with those predisposing factors that distinguish regular drinkers from less frequent drinkers (or heavy drinkers from light drinkers) and those factors that distinguish drinkers from abstainers differing only in degree rather than in kind. The independent liability dimensions (ILD) model (Eaves and Eysenck, 1980) was a two-process model that postulated that there are two independent liability dimensions that together influence quantity or frequency of consumption: the first is an abstinence dimension and the second is a frequency (or quantity) dimension that determines frequency of consumption (or quantity consumed) in those

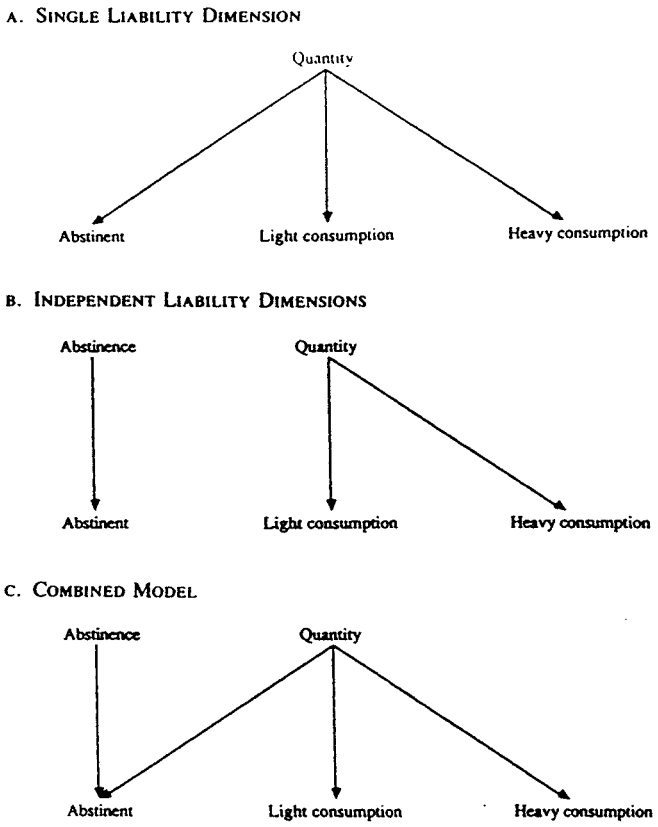

FIGURE 1. Schematic representation of single liability dimension, independent liability dimensions and combined models, for differences in quantity consumed

who are not abstainers. Unlike the single liability dimension model, the ILD model predicts that the drinking co-twins of abstinent twins will not differ in their average frequency of alcohol consumption (or quantity consumed) from the drinking co-twins of drinking twins, since abstinence and frequency (or quantity) dimensions are assumed independent.

We also considered a third, combined (CM) model that combined features of both SLD and ILD models. Like the ILD model, it postulated that there are two independent liability dimensions influencing frequency of consumption (or quantity consumed), and that those in the lower tail of the liability distribution for the first dimension, abstinence, would be abstainers. Like the SLD model, it postulated that those in the lower tail of the second dimension, frequency (or quantity), would also be abstainers. In other words, the combined model postulated that there are two different routes to abstinence from alcohol. By comparing the results of fitting the SLD and ILD models and the combined model, we were able to determine whether either of the two former models was sufficient to explain our data, or whether the more general model gave a significant improvement in fit over both. We would not necessarily expect the same model to fit both the frequency data and the quantity data. We considered it

possible that the SLD model would fit the frequency data, but that either ILD or CM models would be needed to explain the quantity data.

For each of the SLD, ILD and CM models, we compared the effects of making different assumptions about the influence of genetic and shared environmental effects on the postulated liability dimension(s), and about the interactions of these genetic and environmental effects with sex and cohort. We considered three basic models for each liability dimension: (1) influence of environmental effects shared by members of a twin pair, as well as of nonshared environmental effects (that make one twin differ from his/her co-twin), but no influence of genetic effects (environmental model); (2) influence of additive genetic effects and nonshared environmental effects (genetic model); (3) influence of additive genetic effects and both shared and nonshared environmental effects (full model). We also tested for differences in the magnitude of these effects between sexes and between cohorts.

If there were sex differences in genetic and environmental effects, we tested whether the correlation between shared environmental effects in the two sexes, or between genetic effects, was significantly less than unity. This might arise, for example, if some shared environmental effects were influencing only one sex, or some genetic influences were being expressed in only one sex. In either case, the opposite-sex dizygotic correlation would be lower than would otherwise be predicted from the four same-sex twin correlations. Thus, if both additive genetic effects and shared environmental effects were important, and the opposite-sex correlation were too low, we would be unable to determine whether this was because of a genetic correlation less than unity or a shared environmental correlation less than unity (Eaves, 1977). If there were differences in the magnitude of genetic and environmental effects between age cohorts, the absence of intergenerational or longitudinal data made it impossible for us to determine whether some genetic effects or some shared environmental influences were restricted to one cohort.

For each of the SLD, ILD and CM models, we also fitted a general model which estimated, for each liability dimension, a separate polychoric correlation (Olsson, 1979) for each of the twin groups. Fitting this model would give the same results as the full model with sex-dependent and cohort-dependent effects, and a genetic or shared environmental correlation between sexes less than unity, unless the assumptions of the full model are violated (e.g., because the dizygotic twin correlations are higher than the monozygotic correlations).

Model fitting

For each of the 10 twin groups (2 cohorts × 5 sex/zygosity groups), a two-way 6 × 6 contingency table was computed, cross-classifying alcohol consumption frequency, or quantity consumed, of the first twin (or male twin in the case of opposite-sex pairs) by that of the second twin (or female twin). Models were fitted separately to the five contingency tables for each cohort, and then jointly to the full set of 10 tables. Models were fitted by the method of maximum likelihood (Eaves et al., 1978; Eaves and Eysenck, 1980). We shall discuss model fitting with explicit reference to the quantity variable. All methods apply equally to frequency of consumption.

For each of the three models, we assumed that the underlying liability dimension(s) were normally distributed and that their distribution in twin pairs was multivariate normal. For the SLD model, and for the quantity dimension of the ILD and CM models, we assumed that abrupt thresholds, t_o, t_1 . . . t_n, were superimposed upon the underlying liability distribution, dividing it into discrete categories: $t_0 = \infty$, $t_n = +\infty$, and thresholds t_1 . . . t_{n-1} were to be estimated by model fitting. Thus a twin with liability lying between t_0 and t_1 would fall into quantity category (i), a twin with liability lying between t_1 and t_2 would fall into quantity category (ii), and so on. For both SLD and CM models, there were six discrete quantity categories and $n = 6$; but for the ILD model the quantity dimension had only five categories (i.e., no abstinence category), giving $n = 5$. For both ILD and CM models, we further assumed that the abstinence dimension was subdivided into two categories by thresholds s_0, s_1 and s_2 where $s_0 = -\infty$, $s_2 = +\infty$, and s_1 was an additional model parameter. Those twins with liabilities greater than s_1 on the abstinence dimension would be abstainers, the rest would be drinkers (ILD model) or potential drinkers (CM model). In all analyses, we allowed thresholds to vary with both sex and age cohort. Thus there were 10 thresholds to be estimated when the SLD and ILD models were fitted to each cohort, and 12 thresholds when the CM model was fitted; and twice these numbers when all 10 contingency tables were analyzed jointly.

Let f_{ijk} denote the frequency of twin pairs from the k-th twin group falling into the i,j-th cell of the two-way observed contingency table, and p_{ijk} denote the corresponding expected probability under a given model with given parameter values (including thresholds). The log-likelihood of a set of observations under the model is given by

$$L = \ln (c) + \Sigma\Sigma\Sigma\, f_{ijk} \ln (p_{ijk}) \qquad (1)$$

and hence maximum-likelihood estimates of model parameters are obtained by maximizing (1) with respect to the parameter values. Our task, therefore, is to find a function relating p_{ijk} to the model parameters.

The single liability dimension model assumes a direct one-to-one correspondence between the categories into which the underlying liability distribution is divided and the observed quantity categories. The probability that a

TABLE 2. Frequency of alcohol consumption and average quantity consumed when drinking at weekends, broken down by sex and age cohort

Consumption measure	Respondents falling in each class (%)			
	Young women (n = 2,352)	Young men (n = 1,470)	Older women (n = 2,523)	Older men (n = 1,275)
Frequency				
(i) every day	2.7	5.6	14.5	18.4
(ii) 3–4 times each week	10.0	18.7	12.7	18.3
(iii) about twice a week	17.3	19.4	8.4	12.8
(iv) about once a week	21.1	19.4	10.3	11.0
(v) once or twice a month	20.0	16.0	13.7	12.1
(vi) less often/never	28.9	20.9	40.4	27.5
Quantity				
(i) 9 + drinks/occasion	10.0	22.3	3.6	10.9
(ii) 7–8 drinks/occasion	4.6	7.2	2.4	7.5
(iii) 5–6 drinks/occasion	12.0	13.8	5.7	11.9
(iv) 3–4 drinks/occasion	19.7	15.4	14.5	16.5
(v) 2 or fewer drinks/occasion	24.8	20.3	33.4	25.6
(vi) abstainer [a]	28.9	20.9	40.4	27.5

[a] Category (vi) is defined identically for the frequency and quantity measures.

twin pair from the k-th group falls into the i,j-th cell of the observed contingency table will be simply

$$p_{ijk} = \Phi(t_i,t_j) - \Phi(t_{i-1},t_j) - \Phi(t_i,t_{j-1}) + \Phi(t_{i-1},t_{j-1}) \quad (2)$$

where Φ is the bivariate normal distribution function with correlation ρ_k, and ρ_k is the liability correlation between twin pairs from the k-th twin group. The twin correlations may in turn be expressed as a function of genetic and environmental parameters (Eaves et al., 1978), or alternatively a separate correlation may be estimated for each twin group (Olsson, 1979).

Equation 2 also gives the conditional probability under ILD and CM models, replacing p_{ijk} by x_{ijk}, that a twin pair will fall into the i,j-th cell, conditional upon the fact that they both fall into the category of drinkers (ILD) or potential drinkers (CM) on the abstinence dimension. Under these models, ρ_k will be the liability correlation of the k-th twin group on the quantity dimension. Likewise, substituting s_m for t_i, s_n for t_j, y_{mnk} for p_{ijk}, and correlation r_k (the liability correlation of the k-th twin group on the abstinence dimension) for correlation ρ_k, Equation 2 gives the probability that the first and second members of a twin pair will fall into the m-th and n-th categories of the abstinence dimension. Under the ILD model, the unconditional probabilities p_{ijk} are given by

$$p_{ijk} = y_{11k}\, x_{ijk},\ i = 1, \ldots 5,\ j = 1, \ldots 5 \quad (3)$$
$$p_{ijk} = y_{12k}\, x_{i.k},\ i = 1, \ldots 5,\ j = 6 \quad (4)$$
$$p_{ijk} = y_{21k}\, x_{.jk},\ j = 1, \ldots 5,\ i = 6 \quad (5)$$
$$p_{ijk} = y_{22k},\quad i = 6,\ j = 6 \quad (6)$$

where y_{11}, y_{22}, y_{12} and y_{21} denote the probabilities that twin pairs from the k-th twin group both fall in the drinking category, both fall in the abstinent category, or are discordant with the first twin a drinker and the co-twin an abstainer, or vice versa; and $x_{i.k}$ denotes the conditional probability of the first twin from the k-th group falling into the i-th category of the quantity dimension, and $x_{.jk}$ denotes the conditional probability of the second twin falling into the j-th category. Equation 3 corresponds to the concordant drinking twin pairs, Equation 6 to the concordant abstinent twin pairs, and Equations 4 and 5 give probabilities for twin pairs where the first twin is a drinker and the second an abstainer, or vice versa.

Under the combined model, y_{11k}, y_{12k} etc. will give the probabilities that twins fall into the abstinent or drinking category on the abstinence dimension, but twins in the drinking category may still become abstainers because of their position on the quantity dimension. Unconditional probabilities for concordant-drinking pairs will be given by Equation 3 above. Other expressions for unconditional probabilities for pairs where one or both twins are abstainers will be:

$$p_{ijk} = y_{11k}\, x_{ijk} + y_{12k}\, x_{i.k},\ i = 1, \ldots 5,\ j = 6 \quad (7)$$
$$p_{ijk} = y_{11k}\, x_{ijk} + y_{21k}\, x_{.jk},\ j = 1, \ldots 5,\ i = 6 \quad (8)$$
$$p_{ijk} = y_{22k} + y_{11k}\, x_{66k} + y_{12k}\, x_{6.k} + y_{21k}\, x_{.6k},\ i = 6,\ j = 6 \quad (9)$$

These correspond to the cases of concordant abstinent pairs (9) and discordant pairs where either the first twin (7) or the second twin (8) is a drinker. Here x_{66k} denotes the probability of twin pairs being concordant for abstinence on the quantity dimension (given that they are drinkers on the abstinence dimension); and $x_{6.k}$, $x_{.6k}$ denote the probabilities of first or second twins from the k-th group who are drinkers on the abstinence dimension being abstainers on the quantity dimension.

Both the SLD and the ILD models are special cases of the combined model. When $s_1 = +\infty$, the CM reduces to the SLD model. When $t_5 = +\infty$, the CM reduces to the

TABLE 3. Goodness-of-fit of models estimating a separate correlation for each twin group, for each dimension

Model	Young cohort			Older cohort			Joint analysis		
	χ^2	*df*	*p*	χ^2	*df*	*p*	χ^2	*df*	*p*
Frequency									
Single liability dimension	230.71	160	< .001	158.98	160	.51			
Independent liability dimensions	258.19	155	< .001	242.35	155	< .001			
Combined	177.36	153	.11	133.61	153	.87	316.92	316	.47
Quantity									
Single liability dimension	210.02	160	< .01	197.40	160	.02			
Independent liability dimensions	205.62	155	< .01	219.15	155	< .01			
Combined	164.73	153	.24	170.34	153	.16	341.05	316	.16

ILD model. Thus the fit of either model can be compared to that of the more general combined model by likelihood-ratio test (see below).

Assessment of goodness-of-fit

To assess the goodness-of-fit of a model, we calculated the likelihood-ratio statistic,

$$C = 2\ (L_0 - L),$$

where L is the log-likelihood obtained at the maximum-likelihood solution for a given model, and L_0 is the log-likelihood obtained when a separate probability p_{ijk} is estimated for every cell of each contingency table. This statistic is approximately distributed as chi-square, with $35n\text{-}p$ degrees of freedom, where p is the number of model parameters (including threshold values) estimated and n is the number of contingency tables analyzed. To compare the fit of different nested models (e.g., SLD versus CM genetic models; or SLD genetic versus SLD full models), we likewise computed the likelihood-ratio statistic

$$C = 2\ (L_1 - L_2),$$

where L_1 is the log-likelihood of the more general model, L_2 is that of the reduced model that fixes some of the values of the parameters of the former model, and the number of degrees of freedom is equal to the number of parameters of the former model that have been fixed in the latter. Where two models were not nested (e.g., SLD genetic versus ILD genetic models), it was not possible to compare the fit of each directly, but we could still compare each model to the more general model that included both as special cases (e.g., CM genetic model).

Results

Table 2 gives the distribution of frequency of consumption, and average quantity consumed when drinking at week-ends, broken down by sex and age cohort. In both sexes, the older cohort includes a higher proportion of regular drinkers (drinking at least 3–4 times per week), but a smaller proportion of heavy drinkers (e.g., drinking 5 or more alcoholic drinks when alcohol is taken). The older cohort also includes a higher proportion of abstainers. Men report heavier and more frequent alcohol consumption than do women of the same age cohort, but the average quantity consumed by the young female twins is quite comparable to that of the older men.

Frequency of consumption

Table 3 gives the results of fitting single liability dimension, independent liability dimensions and combined models, estimating a separate polychoric correlation for each twin group, for each dimension, and allowing threshold values to vary as a function of sex and age cohort. For the frequency data, for the young cohort, both SLD and ILD models were rejected by chi-square test of goodness-of-fit, but the combined model gave an adequate fit to the data. For the older cohort, the ILD model was again rejected. The SLD model gave an adequate fit to the data, but the combined model gave a significantly better fit, by likelihood-ratio chi-square ($\chi^2 = 25.37$, 7 df, $p < .001$). We did not attempt to fit either SLD or ILD models jointly to both young and older cohorts, since the combined model gave a better fit than these in each cohort. When we fitted the combined model jointly to both cohorts, constraining the genetic and environmental parameters to be the same in both cohorts, but allowing for differences in threshold values between cohorts, this model gave an excellent fit to the data. The chi-square for testing the heterogeneity of genetic and environmental parameters across cohorts was nonsignificant ($\chi^2 = 5.95$, 10 df, $p = .82$). Thus we can conclude that the combined model gives the best fit to the frequency of consumption data and that there is no evidence that genetic and environmental influences on the abstinence and frequency dimensions interact with age cohort.

Table 4 gives estimates of the twin polychoric correlations and their standard errors for the two liability dimensions under the combined model. All correlations are less than unity, showing that nonshared environmental effects

TABLE 4. Twin polychoric correlations (± SEs) under combined model

	Frequency of consumption		Quantity consumed	
	Abstinence r (± SE)	Frequency r (± SE)	Abstinence r (± SE)	Quantity r (± SE)
Monozygotic female	0.82 ± 0.05	0.66 ± 0.03	0.85 ± 0.05	0.56 ± 0.04
Monozygotic male	0.85 ± 0.07	0.74 ± 0.03	0.90 ± 0.05	0.58 ± 0.04
Dizgotic female	0.75 ± 0.12	0.32 ± 0.06	0.71 ± 0.10	0.32 ± 0.06
Dizygotic male	0.84 ± 0.12	0.52 ± 0.06	0.91 ± 0.08	0.43 ± 0.07
Opposite-sex dizygotic	0.78 ± 0.13	0.27 ± 0.05	0.74 ± 0.11	0.20 ± 0.05

are important for both dimensions. For the abstinence dimension, polychoric correlations for each twin group were all comparable in magnitude, giving little evidence for genetic effects. Familial environmental effects were very important, with the estimated twin correlations all lying in the range 0.75–0.85. For the frequency dimension, the monozygotic correlations were significantly higher than the corresponding dizygotic correlations. In female same-sex pairs, the dizygotic correlation was roughly one-half the monozygotic correlation, consistent with additive gene action but no shared environmental effects on the frequency dimension. In male same-sex pairs, the dizygotic correlation for this dimension was greater than one-half the monozygotic correlation, suggesting both additive gene action and shared environmental effects.

Table 5 compares the results of fitting different genetic and environmental combined models to the frequency of consumption data, analyzing the two cohorts jointly and assuming that genetic and environmental effects are homogeneous across cohorts. We do not give results for the SLD and ILD models since these gave worse fits than the corresponding combined models. Models in the table are identified by their differing assumptions about the causes of family resemblance for each dimension, since all models allowed for nonshared environmental effects. All other models were compared to the most general model, which estimated separate polychoric correlations for each twin group for each dimension.

Estimating a single shared environmental parameter instead of five polychoric correlations for the abstinence dimension (model 4 in Table 5) gave a very slight and nonsignificant worsening of fit, compared to the general model (1). This confirmed that family resemblance for this first dimension could be explained by shared environmental effects, and that there was no evidence for either genetic effects or sex-dependent effects. A model that ignored sex-dependent effects for the frequency dimension (model 9) gave a significantly worse fit than the most general model. So, too, did a sex-dependent environmental model (8). A sex-dependent additive genetic model, fixing the correlation between gene effects in the two sexes to unity, gave an adequate fit to the data (model 7); but adding sex-dependent shared environmental parameters to this model gave a highly significant improvement in fit (model 5: $\chi^2 = 10.53$, 2 df, $p < .005$). This latter model gave a fit that was not significantly worse than the most general model. However, a sex-dependent genetic model that allowed for a correlation between gene effects in the two sexes of less than unity (model 6) also gave an adequate fit to the data, and a fit that was not significantly worse than the most general model. These two models, therefore, could not be resolved by our data.

From the results of model fitting, therefore, we concluded that frequency of alcohol consumption is determined by at least two independent dimensions which show strong familial aggregation. The first dimension, which we labeled abstinence, was environmentally determined. We labeled the second dimension frequency, but it must be remembered that under the combined model individuals who would be drinkers on the abstinence dimension

TABLE 5. Results of fitting genetic and environmental models and combined model to frequency data

Model		Goodness-of-fit			Likelihood-ratio vs full model		
Abstinence	Frequency	χ^2	df	p	χ^2	df	p
1. Separate r's	Separate r's	316.92	316	.47	–	–	–
2. Full	Separate r's	317.26	319	.52	0.34	3	.75
3. Genetic	Separate r's	334.52	320	.28	17.60	4	.001
4. Environmental	Separate r's	317.40	320	.53	0.48	4	.98
5. Environmental	Full, sex-dependent	317.71	321	.60	0.79	5	.98
6. Environmental	Genetic, sex-dependent[a]	324.21	322	.45	7.29	6	.29
7. Environmental	Genetic, sex-dependent	328.24	323	.41	11.32	7	.13
8. Environmental	Environmental, sex-dependent [a]	382.15	322	.01	65.23	6	< .001
9. Environmental	Full model, no sex effects	335.46	323	.30	18.54	7	< .001

[a] Indicates correlation between genetic effects in men and women allowed to take values less than unity.

Note: Genetic and environmental parameters are sex-independent unless otherwise indicated. Separate r's indicate that separate polychorics were estimated for each twin group.

TABLE 6. Results of fitting genetic and environmental models and combined model to quantity data

Model		Goodness-of-fit			Likelihood-ratio vs full model		
Abstinence	Quantity	χ^2	*df*	*p*	χ^2	*df*	*p*
1. Separate *r*'s	Separate *r*'s	341.05	316	.16	–	–	–
2. Full	Separate *r*'s	343.88	319	.16	2.83	3	.41
3. Genetic	Separate *r*'s	367.70	320	.03	26.65	4	< .001
4. Environmental	Separate *r*'s	345.07	320	.16	4.02	4	.40
5. Environmental	Full, sex-dependent	345.26	321	.17	4.21	5	.52
6. Environmental	Genetic, sex-dependent[a]	350.13	322	.13	9.08	6	.17
7. Environmental	Genetic, sex-dependent	357.62	323	.09	16.57	7	.02
8. Environmental	Environmental, sex-dependent[a]	375.12	322	.02	26.07	6	< .001
9. Environmental	Full model, no sex effects	358.38	323	.09	17.33	7	.01

[a] Indicates correlation between genetic effects (or shared environmental effects) in men and women allowed to take values less than unity.
Note: Genetic and environmental parameters are sex-independent unless otherwise indicated. Separate *r*'s indicate that separate polychorics were estimated for each twin group.

may still end up as abstainers because they fall in the lower tail of the frequency dimension. There were significant genetic effects on the frequency dimension, and these genetic effects interacted with sex. However, we were unable to determine whether there were also shared environmental effects on frequency in men (model 5), or whether the correlation between gene effects in the two sexes was less than unity (model 6). Either of these two models gave an adequate fit to the data.

From the parameter estimates obtained under the two best fitting models, we calculated that shared environmental effects accounted for 81–83% of the variance in liability on the abstinence dimension, with the remaining variance attributable to nonshared environmental effects. Under model 5, the heritability of frequency of consumption was 66% in women and 42% in men. In men shared environmental effects were also important, accounting for an additional 32% of the variance; but in women the shared environmental variance component was estimated as zero. Under model 6, the heritability of frequency of consumption was estimated as 65% in women and 75% in men, and the correlation between gene effects was estimated as 0.74.

Quantity consumed

The results obtained for the quantity variable were broadly consistent with those for frequency of consumption. The SLD and ILD models were rejected in both age cohorts, but the combined model gave an adequate fit in each case (Table 3). When the results of the joint analysis and the separate cohort analyses were compared, there was no significant evidence for heterogeneity of genetic and environmental effects across cohorts ($\chi^2 = 5.98$, 10 df, $p = .82$). Estimates of polychoric correlations for the abstinence dimension under the combined model were comparable to those obtained in the analyses of the frequency of consumption data (Table 4). For the monozygotic twin groups, estimated correlations for the quantity dimension were lower than was the case for the frequency dimension. However, there was again evidence for significant genetic effects on quantity in both sexes, and perhaps also shared environmental effects in men.

Table 6 summarizes the results of fitting different genetic and environmental combined models to the quantity data, analyzing the two age cohorts jointly. A model that estimated separate twin correlations for the quantity dimension, but estimated a single shared environmental parameter for the abstinence dimension (model 4 in Table 6), gave almost as good a fit as the most general model estimating separate correlations for each dimension (model 1). Models that ignored sex-dependent effects (9) or genetic effects (8) on the frequency dimension could be rejected. Once again, however, it was not possible to choose between a full model with sex-dependent effects (5) and a genetic model with sex-dependent effects and a correlation between gene effects in the two sexes of less than unity (6).

As with the analyses of the frequency of consumption data, therefore, the results indicated shared environmental effects, but no genetic effects, on the abstinence dimension, with no evidence that these environmental effects interact with sex; and, for the quantity dimension, either sex-dependent genetic effects, with a correlation less than unity between gene effects in the two sexes, or else sex-dependent genetic and shared environmental effects, with the latter being important only in men. Shared environmental effects accounted for 86% of the variance in the abstinence dimension. For the quantity dimension, additive genetic effects accounted for 57% of the variance in women, and in men either 61% (model 6) or 24% (model 5) of the variance. Under model 6, the genetic correlation was estimated as 0.56; under model 5, shared environmental effects accounted for 35% of the variance in men (but 0% of the variance in women).

Conclusions

The breakdown, by age cohort and sex, of frequency of consumption and average quantity consumed (Table 2)

confirms that important information may be lost by using only an overall measure of average total alcohol consumption. In this sample, older respondents reported drinking more frequently, but younger respondents more heavily, on those occasions when they consumed alcohol. Without either follow-up data on the younger respondents or data on a new cohort of young adult twins we cannot be certain whether these represent separate developmental stages in the natural history of alcohol use (cf., Vaillant, 1983) or cohort-related differences in drinking style. In a previous article (Heath et al., 1991) we speculated that Cloninger's Type I and Type II alcoholics (Cloninger, 1987; Cloninger et al., 1981, 1985, 1988) might represent those individuals with extreme liability values on frequency and quantity dimensions. It is noteworthy that Type I alcoholics are more likely to report late onset (i.e., at an age when regular drinking would be at its peak in this sample) and Type II alcoholics, early onset (when heavy drinking would be highest).

Our results confirm that the inheritance of abstinence is separate from that of frequency or quantity dimensions. We found no evidence for genetic effects on the abstinence dimension but a major effect of the shared environment. Although there have been comparatively few characters found for which monozygotic and dizygotic twin correlations are similar but substantial, religious affiliation appears to be one such case (Eaves et al., 1989). Since groups with different religious beliefs have been found to show different levels of abstinence (Cahalan et al., 1969; Clark and Midanik, 1982; Encel et al. 1972; Heath and Martin, 1988; Mulford, 1964; Riley and Marden, 1947), such beliefs may prove to play an important role in the inheritance of the abstinence dimension.

For both frequency and quantity dimensions, we found a very similar pattern of inheritance. There was evidence for an important influence of genetic effects in both sexes. It is possible that these genetic effects influence only alcohol consumption pattern. Alternatively, they may reflect inherited, temperamental (Tarter et al., 1985) or personality (Cloninger, 1987) differences having broader effects on behavior. It also appeared that there were shared environmental influences on these dimensions in men, but not women. However, we could not reject the possibility that some of the genes influencing consumption pattern are sex-specific. The striking similarity of the results for quantity and frequency variables suggests the possibility that there are genetic or shared environmental influences that are common to both dimensions. More complex multivariate genetic analyses (Heath et al., 1989b; Martin and Eaves, 1977; Martin et al., 1985) would be needed to test this hypothesis.

We found no evidence for the interaction of genetic and environmental effects with age cohort. There were cohort-related mean differences in frequency and quantity of consumption, which we took into account by estimating separate thresholds for each cohort. The genetic and environmental causes of variability about these means did seem to be consistent across cohorts. These findings contradict an earlier report of analyses of these data which combined quantity and frequency variables to yield a total consumption measure, and which assumed a single liability dimension model, including abstainers in the analysis (Jardine and Martin, 1984).

For male same-sex pairs, Jardine and Martin found no significant evidence for heritable influences on alcohol consumption by older men. This probably resulted from the confounding in that article of the inheritance of abstinence and frequency and quantity dimensions. For the former dimension we, too, found nongenetic inheritance, and we would predict from the increased frequency of abstainers that it would be in the older cohort that the evidence for heritable influences on consumption would be hardest to detect. For the female same-sex pairs, Jardine and Martin reported an increase in nonstandardized genetic and environmental variance components between younger and older age cohorts. Much of this apparent heterogeneity can be explained by the overall increase in variability with age (Jardine and Martin, 1984), which in our analyses will be taken into account by the estimation of separate threshold values for each cohort. However, a second article, which analyzed the effects of Genotype × Environment interaction on total consumption by non-abstinent female twins (Heath et al., 1989a) and took account of overall variability differences, also found evidence for a change in genetic effects between age cohorts which we were unable to confirm in the present analyses. Since the analysis of discontinuous rather than continuous variables in the present article will lead to an inevitable loss of statistical power, we cannot exclude the possibility that interactions of genetic and environmental effects with age cohort are occurring (cf., Cloninger et al., 1988; Reich et al., 1988) but are too weak to be detected in our analyses.

The independent liability dimensions and combined models that we have used in this article may have other applications in the analysis of the inheritance of vulnerability to substance abuse. Our results for alcohol consumption patterns suggest that there are at least two paths to abstinence: those who abstain because of their religious beliefs and those who are not abstainers by belief (i.e., who are potential drinkers on the nongenetic abstinence dimension) but nonetheless become abstainers by temperament (or whatever else characterizes the partly genetic frequency or quantity dimensions). For other abused substances, where there may be between-family differences in access to abused drugs as well as differences in vulnerability amongst those with access, we might expect similar two-process models to apply.

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