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Alcohol Use, Smoking Habits and the Junior Eysenck Personality Questionnaire in Adolescent Australian Twins

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Abstract. In 1988, questionnaires were received from 1,400 twin pairs (17% MZM, 23% MZF, 17% DZM, 19% DZF, 24% DZO) aged 11 to 18, registered with the Australian NHMRC Twin Registry. Twins reported independently on themselves and on the perceived behaviour of their parents, siblings and friends. For smoking and for drinking in the previous month, the prevalence was modelled as a logistic function of age, sex, perceived smoking or drinking behaviour of family and friends, and the Junior Eysenck Personality Questionnaire (JEPQ) scales. Strenghts of association were: family behaviour, odds ratio (OR) ≤ 2; Extraversion and Psychoticism, interquartile OR $\approx$ 1.6; behaviour of friend, OR $\approx$ 3 to 6. Twin associations were represented by odds ratios. For smoking they were 16 in MZ and 7 in DZ same-sex pairs, and 3 in DZO pairs. Although the former is consistent with genetic factors determining adolescent smoking behaviour, the reduced association in DZO pairs and strong association with smoking by friends argue to the contrary. For drinking, twin odds ratios were 11 in MZM, MZF and DZF pairs, and 4 in DZM and DZO pairs, consistent with genetic factors determining alcohol use in male but not female, adolescents. Twin odds ratios were not influenced by adjustment for the JEPQ scales; this does not support the hypothesis that genetic factors which determine personality also determine smoking or drinking behaviour during adolescence.

Key words: Adolescents, Alcohol, Family, Genetics, Logistic regression, Maximum likelihood, Odds ratios, Personality, Smoking.

INTRODUCTION

With infectious disease now under 'control' in most Western societies, lifestyle factors have become a major cause of morbidity and premature mortality. Smoking, excessive

use of alcohol, excessive exposure to ultraviolet radiation, obesity and lack of physical activity are just a few examples. Unlike the causes of many infectious diseases, these factors are generally under the control of individuals. There is, therefore, the possibility that through behaviour change, the associated morbidity and mortality can be reduced. In order to develop appropriate interventions either to prevent habits developing, and/or to help individuals change their behaviour, it is desirable to understand the process by which these behaviours are developed.

Tobacco smoking and alcohol abuse account for more than 95% of drug-related deaths in Australia [10]. Adolescents experiment with tobacco and alcohol as part of growing up; it has been found that 35% of adult smokers began their habit while at school, while a further 14% began to smoke just after leaving school [9]. A substantial proportion of adolescents will go on to establish smoking and drinking habits that will lead to health problems in later years.

Predicting which children are most likely to smoke or drink heavily as adolescents and adults is, therefore, an important public health question. What psychological, family and social factors predict individuals at high risk for a sustained use of tobacco or for excessive use of alcohol during adolescence, or for continued use during early adulthood? What familial factors, such as genetic determinants or behaviours established while living in the family home, predict sustained use of tobacco and excessive use of alcohol in early adulthood? What are the relative predictive strenghts of these factors?

In 1988 a cohort of Australian adolescent volunteer twin pairs aged 11 to 18 years completed a questionnaire about their own use (and the perceived use by their family and friends) of alcohol and tobacco, and other features of their health and lifestyle. In this paper, the probability that an adolescent twin reports smoking or drinking during the previous month was examined as a function of age, sex, perceived family behaviour, perceived behviour of closest friends, and of the twin's score on the Eysenck Junior Personality Scale (JEPQ) [2,3]. In addition, the possible role of genetic factors in determining smoking and drinking behaviour during the formative adolescent years was studied by estimating the within-pair association in smoking or drinking behaviour separately for the five zygostiy types.

METHOD

Australian Adolescent Twin Study

During 1987, questionnaires were mailed to 2,967 sets of parents of all twin pairs aged 11 to 18 years of age registered with the volunteer Australian NHMRC Twin Registry asking permission for their twin children to participate in a longitudinal study of the health and lifestyle of adolescent twins. Of these, 1,450 (49%) gave consent, 96 (3%) refused permission, 134 (5%) were returned to sender, and no reply was received from 1,287 (43%). It is likely that up to one half of the latter group were no longer living at the mailed address, given that this was the first mailing to this age group since recruitment onto the Registry, which occured for most during the early 1980s. As the major aim of study was to follow twins from adolescence to adulthood in order to determine

factors predictive of adult smoking and drinking behaviours, it was not critical from that viewpoint that the study sample was a 'random' population sample, although sample selection needs to be considered when interpreting analyses of twin associations. In any case, twins on the Registry are neither a random no population sample. No attempt was made to re-mail to non-respondents.

During 1988, questionnaires were mailed through the consenting parents and completed questionnaires were received from 2,836 twin individuals (98%). As 36 of these were from one memebr of the pair only, a total of 1,400 twin pairs comprised the study sample (97%). The questionnaire included items on zygosity, time spent with cotwin, self-reported alcohol use, smoking habits, and the JEPQ. The smoking and drinking items were those used in an anonymous Australia-wide survey conducted within classrooms during 1987, on 19,000 12- to 17-year old schoolchildren, namely, the Secondary School Alcohol and Smoking Survey (SSASS) [8]. In addition, twins reported independently on the smoking and drinking behaviour of their parents, siblings, cotwin and on that of up to four of their closest friends. The amount of time spent with each relative and friend was also reported.

For the statistical analysis below, smoking and drinkng in the previous month were defined by a positive answer to the questions: "Have you smoked cigarettes/had an alcoholic drink in the last four weeks?" Perceived smoking and drinking status was defined by the individual twin recording the relative or friend as being an occasional, light, heavy or chain smoker, or as an occasional, light, party or heavy drinker, as distinct from a non-smoker or a non-drinker. For brothers, sisters and friends, a variable of smoking and drinking was defined for each group depending whether there was at least one member of the group perceived to be a smoker or drinker respectively. For example, a twin would be recorded as 'having a friend who smoked' if at least one of the nominated closest friends was perceived by the twin to be a smoker by the above definition.

Modelling Association in a Binary Trait Between a Pair of Twins

There are numerous measures of association for 2×2 tables, which can be applied to twin data on a binary trait. During the early twentieth century there were many intense debates among statisticians about the 'right' measure eg. [18]. It is now clear that different measures have different uses, and the choice of which to use should be guided by practical considerations. For example, if one is interested in making inferences about the genetic and environmental causes of variation in a theoretical and bivariate normal distributed 'liability' for a binary trait, the polychoric correlation coefficient is applicable. On the other hand, a descriptive measure of association in a pair of ordered binary variables is given by the usual correlation coefficient. As previoulsy illustrated [5,6,13], when applied to twin data this can be used to make inferences about the genetic and environmental determinants of the binary trait itself, (as distinct from the unmeasured and hypothesised, yet untestable, liability). With the latter approach it is possibile to model flexibly the influence of measured covariates on the prevalence of the trait.

Let Y_i, $i = 1,2$, be a binary trait for individual i of a (twin) pair, and write $\pi_i = P(Y_I = 1)$, so that $P(Y_i = 0) = 1 - \pi_i$, and $Var(Y_i) = \pi_i(1 - \pi_i)$. The correlation coefficient is

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$$\varrho = [E(Y_1Y_2) - E(Y_1)E(Y_2)] / [Var(Y_1)Var(Y_2)]^{1/2}$$
$$= [P(Y_1 = 1, Y_2 = 1) - \pi_1\pi_2] / [\pi_1(1-\pi_1)\pi_2(1-\pi_2)]^{1/2}.$$

Let $D = [\pi_1(1-\pi_1)\pi_2(1-\pi_2)]^{-1/2}$. The four joint probabilities for j, k = 0, 1 can be expressed as

$$P(Y_1 = j, Y_2 = k) = P(Y_1 = j)P(Y_2 = k) + \delta_{jk}\varrho D, \tag{1}$$

where $\delta_{jk} = 1$ if j = k, else −1. The log likelihood of n independent pairs, $\{(Y_{1\ell}, Y_{2\ell})\}$ of observations is given by

$$LL = \sum_{\ell=1}^{n} \log[P(Y_{1\ell} = y_{1\ell}, Y_{2\ell} = y_{2\ell})] \tag{2}$$

and can be expressed as a function of the parameters ϱ, π_1 and π_2. There are, however, problems in using the parameter ϱ. Its magnitude is difficult to interpret as its association with the tetrachoric coefficient depends on the marginal probabilities, π_1 and π_2; see Fig. 1 of [11], as does the range of permissible values of ϱ; see eg. [12].

The odds ratio

$$\psi = P(Y_1 = 1, Y_2 = 1)P(Y_1 = 0, Y_2 = 0) / P(Y_1 = 1, Y_2 = 0)P(Y_1 = 0, Y_2 = 1) \tag{3}$$

has become a major concept in epidemiology. It has a natural interpretation for rare traits, ie. π_1, π_2 small, in which case it is approximately equal to the risk ratio, the proportional increase in disease risk of an individual given that the other member of the pair is affected. For statistical reasons it is preferable to use the natural logarithm of the odds ratio, log (ψ), as it is unbounded and related to logistic regression coefficients.

The influence of covariates on trait prevalence can be easily accommodated. For example, let $\pi_1 = P(Y_{1\ell} = 1 \mid X_{1\ell} = x_{1\ell})$ and $\pi_2 = P(Y_{2\ell} = 1 \mid X_{2\ell} = x_{2\ell})$ be logistic functions of observed values, x, of covariates, X, with parameters $\{\alpha_j\}$. That is,

$$\text{logit}[E(Y_{i\ell} \mid X = x_{i\ell})] = \alpha_0 + \alpha_1 x_{1\ell} + \ldots + \alpha_p x_{p\ell}$$

where logit(x) = log[x/(1−x)].

For a given pair, write $\pi_{jk} = P(Y_{1\ell} = j, Y_{2\ell} = k \mid X_{1\ell} = x_{1\ell}, X_{2\ell} = x_{2\ell})$ for j,k = 0,1,

$$\psi = \pi_{11}\pi_{00} / \pi_{10}\pi_{01}, \tag{4}$$

$$\pi_1 = \pi_{10} + \pi_{11}, \text{ and } \pi_2 = \pi_{01} + \pi_{00}$$

Therefore,

$$\varrho^2 D(\psi - 1) - \varrho[(\psi - 1)(\pi_1 + \pi_2 - 2\pi_1\pi_2) + 1] + D(\psi - 1) = 0. \tag{5}$$

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For given ψ, covariates, π_1 and π_2, if equation (5) has real roots then, as the product of roots must be 1, only one of them is between -1 and 1. The four joint probabilities $\{\pi_{jk}\}$ can be calculated using equation (1), and the log likelihood given by equation (2) expressed in terms of ϱ, π_1 and π_2. Furthermore, the latter two parameters can be expressed as functions of the logistic regression coefficients, $\{\alpha_j\}$, while the parameter ϱ can be parameterised in terms of measured characteristics of the pair such as zygosity, age, sex, cohabitational status, etc. Parameters can be estimated by maximum likelihood, using a numerical optimisation routine such as SEARCH [16], and model fitting carried out by reference to asymptotic likelihood theory using the likelihood ratio test, or by Akaike's criterion [1]. Confidence intervals based on standard errors will be more appropriate if ψ is estimated on a logarithmic scale.

RESULTS

Table 1 shows the age-by-sex distribution of the twin sample. The self-reported zygosity breakdown was 235 (17%) MZM, 321 (23%) MZF, 240 (17%) DZM, 265 (19%) DZF and 339 (24%) DZO. The proportion of DZO pairs was significantly less than the proportion of DZ like-sexed pairs ($p<0.01$).

Table 1 - Age-by-sex distribution of the sample of 1,400 adolescent twin pairs, 1988.

	Age								
Sex	11	12	13	14	15	16	17	18	TOTAL
Males	47	186	145	172	214	199	214	114	1,291 (46%)
Females	62	145	176	199	275	269	214	169	1,509 (54%)
TOTAL	109	331	321	371	489	468	428	283	2,800
	4%	12%	11%	13%	17%	17%	15%	10%	

More than 94% of twins under the age of 18 and 80% of 18-year-old twins lived with their family. Ninety-nine percent of twins under the age of 16 and 95% of those aged 16 to 18 lived with their cotwin. The proportion of pairs reporting spending 'almost all' of their time with each other decreased from around 80% in 11- to 12-years-olds to 53% in 17- and 35% in 18-year -olds. Only 3% of 17-year-old and 6% of 18-year-old pairs reported 'rarely or never' spending time with one another, and less in the younger age groups. The proportion of pairs reporting spending 'almost all' of their time with their parents decreased from around 70% for mother and 60% for father in 11- to 12-year-olds to 30% for 17- and 20% for 18-year-olds, independent of the sex of the twin and of the parent. Less than 1% of twins under the age of 18 'rarely or never' spent time with their mother, compared to 6% for fathers, while for 18-year-olds these figures increased to 5% and 14% respectively, independent of the sex of the twin.

Table 2 compares the percentage of twins who reported smoking or drinking in the previous month with results from the large Australian secondary school survey. It can be seen that rates in the twins were lower in early adolescence, but more comparable at older ages.

Table 2 - Percentage of twins reporting smoking cigarettes, or having an alcoholic drink, in the previous month, and the same percentages from the Australian secondary school survey (SSASS: n = 19,000). After Hill et al [8].

		Age							
Sex	Study	11	12	13	14	15	16	17	18
Smoking:									
Males	Twins	0	0	8	9	22	27	29	32
	SSASS	n.a.	7	13	22	28	31	29	n.a.
Females	Twins	0	3	5	12	20	26	29	31
	SSASS	n.a.	7	15	27	33	36	32	n.a.
Drinking:									
Males	Twins	9	14	22	22	31	48	68	81
	SSASS	n.a.	27	34	43	58	70	72	n.a.
Females	Twins	10	7	13	24	37	53	67	86
	SSASS	n.a.	19	31	41	54	65	69	n.a.

Twins classified their parents' smoking as: non- (58%), ex- (15%), occasional (3%), light (10%), heavy (14%) and chain (1%), with a tendency for fathers to be more often classified as heavy or chain smokers than mothers (14% vs 7%; $p < 0.05$). For drinking the breakdown was: non- (22%), occasional (38%), light (24%), party (10%) and heavy (8%), with fathers more likely to be classified as party or heavy drinkers than mothers (17% vs 7%; $p < 0.05$).

Table 3 shows the mean and standard deviations of the four JEPQ scales by age and sex. The median standard deviations were: 2.7 for Psychoticism, 4.0 for Extraversion, 4.8 for Neuroticism, and 3.8 for the Lie scale.

To explore the relationship between smoking and drinking and the JEPQ scales, two indices SMINDEX and DRINDEX [7] were created. These take into account reported lifetime experience of smoking and drinking, respectively, and usage during the last year, month and week, with more recent experiences given greater weight. Correlations between SMINDEX/DRINKEX and each of the four JEPQ scales were calculated for each age-by-sex category. SMINDEX was positively associated with Psychoticism and Extraversion (median correlation 0.22), and negatively associated with the Lie scale (median correlation −0.22). In females alone there was evidence of a weak association with

Table 3 - Mean and standard deviation (in parentheses) of the JEPQ scales for Psychoticism, Extraversion, Neuroticism and Lie (Social Conformity), by age and sex.

		Age							
Scale	Sex	11	12	13	14	15	16	17	18
Psychoticism									
	Male	4.02	4.84	5.14	4.95	5.13	5.09	4.48	4.27
		(2.66)	(3.31)	(3.13)	(3.16)	(3.20)	(3.41)	(2.98)	(3.16)
	Female	3.60	3.66	3.44	3.67	3.73	3.55	2.57	2.80
		(1.96)	(2.50)	(2.33)	(2.70)	(2.60)	(2.63)	(1.94)	(2.35)
Extraversion									
	Male	15.21	15.40	15.15	15.31	15.06	15.11	15.12	14.80
		(3.31)	(3.75)	(3.82)	(3.38)	(4.20)	(4.04)	(3.77)	(4.32)
	Female	14.48	14.94	15.16	14.73	15.61	15.11	14.41	15.24
		(4.24)	(3.59)	(3.93)	(4.41)	(3.76)	(4.50)	(4.88)	(4.51)
Neuroticism									
	Male	9.96	10.66	11.66	10.81	10.53	10.99	10.45	10.59
		(4.12)	(4.66)	(4.73)	(4.78)	(5.05)	(5.15)	(4.81)	(5.08)
	Female	12.00	11.29	12.60	12.68	12.78	13.50	12.92	12.18
		(4.57)	(5.16)	(4.43)	(4.83)	(4.54)	(4.83)	(4.94)	(5.16)
Lie (Social Conformity)									
	Male	8.70	7.31	6.51	7.36	6.31	6.86	7.40	6.70
		(4.60)	(4.15)	(3.88)	(3.80)	(3.68)	(3.60)	(4.02)	(3.68)
	Female	8.50	8.13	7.94	7.27	7.01	7.30	7.73	7.30
		(4.35)	(4.27)	(3.76)	(4.28)	(3.70)	(3.76)	(3.93)	(3.29)

Neuroticism (median correlation about 0.18). DRINDEX was positively associated with Psychoticism (median correlation 0.27), and with Extraversion in twins 14 and older (median correlation 0.30), and negatively associated with the Lie scale (median correlation −0.20). There was no evidence of an association with Neuroticism.

The statistical model described above was applied separately to the binary traits, smoking in the previous month and drinking in the previous month. The proportion of smokers (drinkers) was modelled as a logistic function of each twin's age (as a quadratic function), sex, perceived smoking (drinking) behaviour of family members, perceived smoking (drinking) behaviour of friends, and of the JEPQ scales. Interactions with sex were examined for all covariates. The association between twins was modelled as a log odds ratio, $\log(\psi)$, by the five zygosity groups (MZM, MZF, DZM, DZF, DZO), and by age.

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Table 4 - Estimates (standard errors in parentheses) for analysis of 'smoking in the previous month'. (The proportion was adjusted for age, age^2 and, although not significant, sex)

	Model I	Model II	Model III	Model IV
Mother Smokes		0.274 (0.159)		0.277 (0.163)
Father Smokes		0.524 (0.148)		0.490 (0.152)
Sister Smokes		0.396 (0.159)		0.299 (0.163)
Brother Smokes		0.603 (0.149)		0.620 (0.151)
Friend Smokes		2.013 (0.139)		1.843 (0.140)
Psychoticism			0.1422 (0.0194)	0.1252 (0.0218)
Extraversion			0.1106 (0.0152)	0.0985 (0.0170)
Neuroticism			0.0319 (0.0117)	0.0239 (0.0132)
Lie (Social Conformity)			−0.0331 (0.0158)	−0.0364 (0.0179)
Log odds ratio MZM	3.561 (0.523)	2.622 (0.601)	3.421 (0.581)	2.968 (0.708)
Log odds ratio MZF	3.415 (0.428)	2.892 (0.498)	3.191 (0.455)	2.761 (0.510)
Log odds ratio DZM	2.167 (0.404)	1.810 (0.510)	2.231 (0.459)	1.817 (0.535)
Log odds ratio DZF	2.175 (0.365)	1.575 (0.436)	2.215 (0.391)	2.063 (0.529)
Log odds ratio DZO	1.101 (0.296)	0.990 (0.338)	1.117 (0.330)	0.975 (0.369)

Tables 4 and 5 show the estimates from model fits on adjustment for age and sex alone (model I), and in addition on adjustment for perceived behaviour of family and friends (models II and IV) and for the JEPQ scales (models III and IV). For both smoking and drinking, adjusting for JEPQ scales made virtually no difference to estimates of log odds ratios (cf. model I with III; II with IV). Adjusting for perceived behaviour

of family and friends led to substantially lower estimates of log odds ratios (cf. model I with II; III with IV). There were no significant interactions with sex, and the log odds ratio estimates were independent of age.

For smoking in the previous month, the log odds ratio estimates were similar for MZM and MZF pairs. They were equivalent to the odds for a monozygotic twin to be

Table 5 - Estimates (standard errors in parentheses) for analysis of 'drinking in the previous month'. (The proportion was adjusted for age, age^2 and, although not significant, sex)

	Model I	Model II	Model III	Model IV
Mother Drinks		0.484 (0.131)		0.509 (0.134)
Father Drinks		0.257 (0.151)		0.269 (0.153)
Sister Drinks		0.259 (0.112)		0.189 (0.114)
Brother Drinks		0.150 (0.116)		0.080 (0.118)
Friend Drinks		1.278 (0.113)		1.153 (0.116)
Psychoticism			0.0756 (0.0169)	0.0757 (0.0183)
Extraversion			0.0963 (0.0121)	0.0889 (0.0129)
Neuroticism			0.0148 (0.0098)	0.0093 (0.0106)
Lie (Social Conformity)			−0.0450 (0.0130)	−0.0339 (0.0140)
Log odds ratio MZM	2.520 (0.343)	2.290 (0.377)	2.457 (0.359)	2.305 (0.390)
Log odds ratio MZF	2.968 (0.334)	2.520 (0.347)	2.902 (0.359)	2.520 (0.365)
Log odds ratio DZM	1.650 (0.335)	1.240 (0.358)	1.713 (0.373)	1.291 (0.386)
Log odds ratio DZF	2.279 (0.336)	2.137 (0.374)	2.460 (0.371)	2.345 (0.415)
Log odds ratio DZO	1.831 (0.289)	1.358 (0.306)	1.742 (0.301)	1.295 (0.316)

a smoker when the cotwin is a smoker, being about 16-fold greater than the odds when the cotwin is a non-smoker. Likewise, for DZM and DZF pairs the association was similar, and equivalent to about a 7-fold increase in odds for smoking. The association between DZO pairs, however, was equivalent to at most a 3-fold increase in odds, marginally lower than in DZ like-sexed pairs combined ($\chi_1^2 = 3.4$; $p < 0.07$). Effects on smoking associated with perceived behaviour were present if the relative was male, but not if the relative was female, independent of the sex of the twin. Overall, the perceived family behaviour associations were small (less than a 2-fold increase in odds for smoking) compared to those associated with the friends' perceived behaviour. The latter was similar in magnitude to that associated with the cotwin's actual behaviour, particularly if the twin was one of a dizygotic like-sexed pair. The odds that a twin was a smoker in the previous month was about 6 times higher if it was reported that one or more of the closest friends smoked, compared to reporting none smoked, and the strength of this association was independent of the sex and zygosity of the twin.

Psychoticism and Extraversion exhibited highly significant and strong associations with smoking in the previous month, with an increase in odds ratio of 0.12 and 0.10 respectively for every unit increase on these scales. By noting that the interquartile range for a normally distributed variable covers approximately 1.35 standard deviations, a rough interpretation of these estimates can be made as follows. In Table 3 the standard deviation of Psychoticism for a given age was about 2.7 units. Therefore the interquartile range for age-adjusted Psychoticism is approximately 3.6 units. The estimate of 0.123 for the logistic regression coefficient associated with Psychoticism (last column Table 4) can thus be interpreted as a predicted increase of log odds of 0.45, or a 1.6 times increase in odds for smoking, going from the lower to the upper quartile on this variable. Similar calculations for Extraversion give an interquartile increase in odds for smoking of about $\exp(1.35 \times 4.0 \times 0.1) = 1.7$. For Neuroticism, the association was of marginal significance, equivalent to an interquartile increase in odds for smoking of about $\exp(1.35 \times 4.8 \times 0.024) = 1.2$. For the Lie scale there was a significant interquartile decrease associated in odds for smoking of about $\exp(1.35 \times 3.8 \times 0.04) = 1.2$.

For drinking in the previous month, the log odds ratio estimates were also similar for MZM and MZF pairs and equivalent to about an 11-fold increase in the odds for drinking if the monozygotic cotwin is a drinker. Again, for DZM and DZO pairs, the association was similar and equivalent to about a 4-fold increase in odds for drinking. The association between DZF pairs, however, was higher than in the other DZ pair categories, equivalent to a 10-fold increase in odds for drinking and no different to the effect found in MZF pairs.

Effects on drinking associated with perceived behaviour were present for the mother and equivalent to a 1.7-fold increase in odds, but not for any other type of relative. Overall, the perceived family behaviour associations were small compared to those associated with the friends' perceived behaviour. The latter was associated with a more than 3-fold increase in odds for drinking and similar in magnitude to that found for twin's actual drinking behaviour in dizygotic male-female pairs.

As for smoking in the previous month, Psychoticism and Extraversion exhibited highly significant and strong asosciations with drinking in the previous month. The strength of association with Psychoticism was marginally lower, with an increase in odds for drinking going from the lower to the upper quartile of about exp

$(1.35 \times 2.7 \times 0.08) = 1.3$. For Extraversion, the interquartile increase in odds for drinking was about exp $(1.35 \times 4.0 \times 0.09) = 1.6$. There was no association with Neuroticism. The Lie scale was associated with a significant interquartile decrease in odds for drinking of about exp $(1.35 \times 3.8 \times 0.03) = 1.2$.

Qualitatively similar results were found when the same analyses were applied to smoking and to drinking in the previous week. In none of the analyses was there evidence that the twin pair odds ratios were dependent on the age of the pair, but only strong associations, however, could have been detected with this sized sample.

DISCUSSION

Simplistic application of the classic twin argument to the observation of higher twin associations in MZ pairs than in DZ same-sexed pairs would suggest that genetic factors, in part, determine adolescent smoking behaviour. Further analyses showed a reduced association in DZO pairs and a strong effect associated with smoking by friends. Both these argue to the contrary, although the former could reflect different genes acting in males and females. The latter indicates that 'peer' influences are important determinants of smoking behaviour, although, of course, from this cross-sectional data it is impossibile to differentiate between the direct influence of peers on an individual's behaviour and an individual's tendency to select or report that they have friends who share behaviours and characteristics associated with smoking. Nevertheless, the difference in smoking association between MZ and DZ pairs, and between DZ like-sex and DZ opposite-sex pairs, could be explained by differences in the strength of peer influences within a twin pair. The relatively weak associations with perceived family behaviour also appear to argue against a role for genes, but misclassification errors due to the indirect nature of these measures could have diluted any true associations with the actual behaviour of first-degree relatives or friends.

For drinking, the odds ratios can be interpreted by the classic twin argument as being consistent with genetic factors playing a role in determining alcohol use in male, but not female, adolescents. An important observation was that twin pair odds ratios, both for smoking and for drinking in the previous month, were not influenced by adjustment for the JEPQ scales. This was despite the finding that Extraversion and Psychoticism were strongly associated with these smoking and drinking behaviours, and despite separate analyses which have shown that these scales are positively correlated in twin pairs [17]. Peto [19], Khoury et al [15] and others have considered, in theory, how the strength of association between a binary trait and a covariate, and the strength of familial aggregation in that covariate, determine familial association in the binary trait. They all concluded that both these factors must be strong in order to have a non-negligible influence on familial association, but as Hopper and Carlin [14] pointed out, this should not be over-interpreted, as the covariate could be merely a poor surrogate for an underlying familial risk factor of some consequence.

Nevertheless, our data do not support the hypothesis that genetic factors which determine personality also determine smoking and drinking behaviour during adolescence. As regards smoking, this hypothesis was put forward by Eysenck and Eaves [4] and can be summarised as: (i) smoking behaviour is, in part, determined by genetic factors;

(ii) smoking behaviour is associated with personality, in particular, with those aspects measured by the scales developed by Eysenck; (iii) these personality dimensions are themselves, in part, under genetic control; and (iv) this explains why genetic factors, in part, determine smoking. This study addresses adolescence, and it could be that during this turbulent period of life, social and peer-related factors are strong enough to obscure underlying genetic influences.

As discussed earlier, these data give limited support to proposition (i) and strong support to proposition (ii). Proposition (iii) has been addressed independently on these data [17] and only limited support was evident. In brief, for Psychoticism the MZM and MZF covariances were similar, and the DZM, DZF and DZO covariances were also similar and equal to about one-half the pooled MZ covariance. All variances and covariances were independent of age. This is consistent with genetic factors determining trait variation. The total variance, however, was greater in males than in females, suggesting that environmental factors are more important among adolescent boys. For Extraversion, in males the MZ covariance was about twice the DZ covariance, and the variance and covariances were independent of age. For females, however, the total variance and covariance in MZ pairs increased markedly with age. The covariance in DZO pairs was about one-half that of DZM and DZF pairs and independent of age. This data does not appear to fit a simple genetic model. For Neuroticism, variances and covariances were independent of age, and within zygosity of sex, although the DZO covariance was about 25% below that of DZM and DZF pairs. These covariances were consistent with genetic factors explaining less than half of variation. For the Lie scale, the striking feature was that variances and covariances decreased with age, independent of sex. There was little difference between MZ and DZ covariances, which accounted for only a small proportion of total variance by age 18. This is not consistent with a classic genetic model.

Young et al [20] reported analyses of Eysenck Personality Questionnaire scale data which included JEPQ responses from 262 twin pairs aged 7 to 17 years (59 MZM, 50 MZF, 40 DZM, 37 DZF, 76 DZO). They augmented this with adult twin and parent-offspring data, and found that the data for Extraversion and Neuroticism were on the whole consistent with a simple additive genetic model with random mating. There were spouse associations on both the Psychoticism and Lie scales. There was evidence of marked inconsistency of gene action between juveniles and adults, with the exception of the Neuroticism dimension. For the Lie scale, social interaction between juvenile cotwins was detected, and the authors suggested this 'as a paradigm for a trait for which environmental interactions between relatives have a major role in the causes of individual variability'. As to whether or not variation in JEPQ scales is genetically determined, the observation that the twin associations in smoking and drinking in the previous month did NOT alter after adjustment for JEPQ scales, argues stronlgy against the hypothesis that genetic variation in personality explains putative 'genetic' causes of smoking and drinking.

Inferences about genetic and environmental causes of trait variation in the population implicitly presume that the trait coavariation in the twin sample studied accurately reflects that in the population. This would be the case if the study sample was random with respect to the traits of interest. The twin subjects in our study were not from a random sample of the twin population, as they had been volunteered by their parents and had volunteered themselves. We were partly able to address whether they were random

with respect to the traits of interest by comparing the responses of our sample with those of the anonymous SSAASS survey of children studied in their (randomly selected) school classrooms. Smoking and drinking rates were similar in the older ages. The lower rates in the younger aged twin subjects could reflect the different mode of questionnaire administration (home vs classroom). Nevertheless, there is the potential that our estimates of twin association in smoking and drinking behaviour might be biased. It is possible that our sample was more likely to include twin pairs more similar in their behaviours. This may not necessarily invalidate conclusions on the existence of genetic causes of variation if the sampling bias is independent of zygosity. Our sample was deficient in its representation of DZO pairs, compared to DZM and DZF pairs. If this is an indication that the sampling bias is stronger for DZO pairs, then differences between our estimates of smoking and drinking associations in DZO pairs compared with other categories of twin pairs will be underestimated. In addition, this would suggest that the associations between sampled DZO pairs are higher than in the population, thus casting further doubts on the role of genetic factors on adolescent smoking and drinking.

The cohort was followed up in 1991 with an 80% pairwise response rate and further follow-up studies are intended. In time, it will be of interest to examine adult smoking and drinking in this cohort to assess whether adolescent scores on the JEPQ scales predict adult smoking and drinking, and to recalculate the twin associations in these behavioural traits.

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