Pergamon

PII: S0191-8869(97)00094-9

Person. individ. Diff. Vol. 23, No. 4, pp. 535–542, 1997

Printed in Great Britain
0191-8869/97 $17.00+0.00

WHAT ARE THE CAUSES OF INDIVIDUAL DIFFERENCES IN FOOD CONSUMPTION AND ARE THEY MODIFIED BY PERSONALITY?

Margaret A. Yeo,[1] Susan A. Treloar,[1] Geoffrey C. Marks,[2] Andrew C. Heath[3] and Nicholas G. Martin[1]*

[1]Queensland Institute of Medical Research, P.O. Royal Brisbane Hospital, Brisbane, Queensland 4029, Australia, [2]Nutrition Program, University of Queensland, Brisbane, Queensland, Australia and [3]Department of Psychiatry, Psychology and Genetics, Washington University School of Medicine, St Louis, MO U.S.A.

(Received 17 February 1997)

Summary—This paper reports, for 1613 Australian twin pairs aged 18–26 yr, the components of variance in self-reported high fat, salt and fibre food consumption, and the influence of personality on this consumption. Individual environment is the most influential factor and most of the familial influence on variance in consumption of high fat and salt food was explained by a common set of genes. Weak associations were found between personality and food consumption variables. The set of genes influencing variance in the social conformity (EPQ-R-S lie scale) personality trait made a small contribution to the genetic effects influencing consumption of fatty and salty food. We did not find evidence to support recently reported positive association between extraversion and salt consumption nor negative associations between neuroticism and psychoticism, and fat consumption. © 1997 Elsevier Science Ltd

Keywords: food consumption, family environment, personality, Eysenck Personality Questionnaire, structural equation modelling, LISREL, twin study

INTRODUCTION

A range of factors may influence food choice, including food-related factors (nutrient content, physical/chemical properties, and physiological effects), economic and social factors (food price and availability, and social/cultural attitudes) and person-related factors (sensory and psychological factors) (MacFie & Thomson, 1994). Person-related factors in food choice models have traditionally been considered in terms of sensory, attitudinal and physiological factors, neglecting the direct effects of individual differences in personality on food consumption behaviour.

Recently, the Eysenckian personality factors of psychoticism, extraversion, and neuroticism were examined in a population-based Australian study of the relationship between personality and nutrient densities (Falconer, Baghurst & Rump, 1993). Sex and nutrient specific correlations were found between personality dimensions of psychoticism and neuroticism, and sodium and fibre densities. A larger proportion of dietary variance was explained by personality factors (15–30%) than demographic factors (6–17%) including education and occupational status (Falconer *et al.*, 1993).

In the context of a twin study we sought to replicate these findings and to extend them by investigating the underlying genetic and environmental factors contributing to any correlation between personality and food consumption.

SAMPLE AND METHODS

Survey design and administration

Australian twins aged 18–26 yr in 1989 were the target population for the Australian Twins and Families Health and Lifestyle Questionnaire (HLQ). The twins were enrolled in the volunteer Australian National Health and Medical Research Council Twin Register (ATR) which contains

*To whom all correspondence should be addressed. Tel.: (07)3362 0278; fax: (07)3362 0101; e-mail: nickm@qimr.edu.au.

approximately 25,000 pairs throughout Australia, i.e. between 10 and 20% of twins living in Australia (Jardine, Martin & Henderson, 1984).

The HLQ was mailed in May 1990 to 4269 pairs born 1964–1971. The study participants represent the younger of two cohorts participating in the HLQ. Most of these twins had been recruited at school, up to 10 yr earlier, so it was not possible to reestablish contact with 1000 pairs although extensive telephone follow-up procedures were implemented. Because of address changes in the interim and name changes in females, we did not expect a high recontact rate. The contactable sample size was therefore 3269 pairs. The HLQ focused on personality, lifestyle and diseases. The food consumption measures had not been incorporated in any of the previous longitudinal studies.

Measures

The study instrument used for measuring consumption of high fat, salt and fibre food was based on a semi-quantitative short dietary questionnaire (Hopkins, Williams, Kuida, Stults, Hunt, Barlow & Ash, 1989), with the three lists of foods modified to be representative of commonly consumed high fat, salt and fibre foods in Australia. The following items provided qualitative measures of food consumption:

- 'When was the last time you ate any food that is high in fat, salt or fibre, such as those in the list above?'; and
- 'How often do you eat any food that is high in fat, salt or fibre, such as those in the list above?'

Response categories are shown in Table 1. The variables were recoded so that high scores reflected low fibre and high fat and salt consumption according to national dietary goals to reduce fat and salt intake and increase fibre intake (Nutbeam, Wise, Bauman, Harris & Leeder, 1993).

Instruments used for measuring personality dimensions were: (i) the revised short scale Eysenck Personality Questionnaire (EPQ-R-S) (Eysenck, Eysenck & Barrett, 1985); and (ii) a short form of Cloninger's Tridimensional Personality Questionnaire (TPQ) (Cloninger, Przybeck & Svakic, 1991; Heath, Cloninger & Martin, 1994). The EPQ-R-S contained 48 'yes/no' items (12 items per personality dimension) from which scores were derived for the four personality dimensions of psychoticism, extraversion, neuroticism and social conformity (lie scale). Seven impulsiveness items supplement the EPQ-R-S extraversion scale which measures only sociability, since impulsiveness and sociability are thought to be genetically distinct traits (Eaves & Eysenck, 1975). The TPQ contains 54 'true/false' items (18 items per personality dimension) from which scores are derived for the three personality dimensions of harm avoidance, novelty seeking and reward dependence (Heath *et al.*, 1994).

Zygosity of twins was determined from their responses to standard items in the HLQ about physical similarity and being mistaken for each other. These items have been found to be at least 95% accurate compared with blood-testing results (Kasriel & Eaves, 1976; Martin & Martin, 1975).

Data analysis

Polychoric correlations. The statistical package SAS 6.09 (Statistical Analysis Systems Institute Inc., 1989) was used for preliminary descriptive analyses. PRELIS 2.12 (Jöreskog & Sörbom, 1993) was used to estimate polychoric correlations for the ordinal food consumption variables. The calculation of polychoric correlations assumes that categorical variables arise from the arbitrary imposition of thresholds on to a normally distributed continuous distribution of liability and that the joint distribution of liabilities of two such variables is bivariate normal with polychoric correlation r. PRELIS treats variables as ordinal if there are fewer than 15 categories. Since PRELIS estimates thresholds for ordinal variables to minimise skewness and kurtosis, the personality variables harm avoidance and reward dependence, with 18 categories and quite non-normal distributions, were recoded to 15 or fewer categories, so that all variables in our analysis were treated as ordinal.

Univariate genetic analysis. We now use LISREL to fit structural equation models, by the method of weighted least squares (WLS), to the polychoric correlations and their accompanying asymptotic covariance matrices (calculated by PRELIS) to test hypotheses about the causes of individual differences. Our hypotheses encompass the possibility that an individual's food consumption may be influenced by individual environmental exposures (E), environmental exposures common to

other family members (C), and also additive (A) and dominant (D) genetic factors. Sensible models, which include one or more of these parameters, are fitted to test the following hypotheses:

1. that there is no family resemblance (E alone);
2. that any family resemblance is due to shared family environment alone (C + E);
3. that any family resemblance is due to additive genetic factors alone (A + E);
4. that any family resemblance is due to both additive genetic and shared environmental factors (A + C + E);
5. that any family resemblance is due to additive and dominant genetic factors (A + D + E).

The fit of the models was assessed in terms of how well the model explained the data and the parsimony of the fit, i.e. the extent to which the model can predict phenomena with a smaller set of parameters. Parsimony of fit was measured by the Akaike information criterion (AIC) which is calculated as:

$$\chi^2 - 2(\text{d.f.})$$

and is interpreted such that the model with the lowest AIC value provides the most parsimonious fit (Neale & Cardon, 1992).

Multivariate genetic analysis. To test hypotheses about the genetic and environmental causes of covariation between variables we need the full cross-twin, cross-trait polychoric correlation matrices for monozygotic and dizygotic pairs. An initial atheoretical multivariate parameterisation employs the Cholesky decomposition which absorbs all genetic and environmental covariance in an arbitrary factorisation (Neale & Cardon, 1992).

For the first multivariate genetic analysis, a full Cholesky model was fitted containing latent additive genetic, shared environmental and individual environmental variables influencing phenotypic variance in six observed food consumption variables ('frequency' and 'last time of consumption' of high fat, salt and fibre foods). A reduced Cholesky model was also fitted which allowed for a common salt and fat factor, and specific fibre factor. The second multivariate genetic analysis was based on correlations between the social conformity personality measure (EPQ lie scale) and 'frequency', and 'last time of, high fat and salt consumption' variables. The full Cholesky model contained latent factors for the four observed food consumption variables and one personality variable. Thus, for each twin, there are five factors for each of the latent additive genetic, shared environmental and individual environmental variables.

RESULTS

Respondents

Response rate by this younger cohort to the Australian Twins and Families Health and Lifestyle Questionnaire (HLQ) was 68.8% (3862/5614) of contactable individuals. Telephone follow-up

Table 1. Response distributions (%) for frequency (FRQ) and last time (LT) of consumption of high fat, salt and fibre foods for male (n = 1419–1442) and female (n = 2198–2212) individuals

Response category	FRQFAT		FRQSALT		FRQFIBRE	
	Males	Females	Males	Females	Males	Females
Almost never	1.5	2.8	2.9	6.6	5.9	5.8
Less than once a week	5.3	12.2	10.0	15.7	40.4	40.8
1–5 times a week	36.7	40.8	37.3	41.3	33.7	33.7
1–4 times a day	49.4	39.8	44.7	33.4	13.3	14.2
5 or more times a day	7.2	4.4	5.1	3.0	6.6	5.5
Response category	**LTFAT**		**LTSALT**		**LTFIBRE**	
	Males	Females	Males	Females	Males	Females
Over a year ago	0.3	0.3	1.3	1.6	2.8	2.6
Over a week ago	1.7	2.4	4.2	6.7	9.9	8.7
A week ago	1.5	2.1	3.5	5.2	5.4	6.0
2–5 days ago	13.0	16.2	18.1	23.2	18.8	20.0
Within the last day	83.5	79.0	72.9	63.3	63.1	62.7

Table 2. Polychoric twin-pair correlations (and asymptotic standard errors) for food consumption variables by sex/zygosity category

	MZF 421[a]		MZM 241		DZF 286		DZM 155		DZFM 160		DZMF 149	
	r	s.e.	*r*	s.e.	*r*	s.e.	*r*	s.e.	*r*	s.e.	*r*	s.e.
LTFAT	**0.20**	0.09	**0.27**	0.14	0.12	0.11	0.07	0.15	0.01	0.15	−0.07	0.17
LTSALT	**0.24**	0.07	**0.23**	0.11	0.17	0.09	0.15	0.12	0.12	0.12	0.22	0.14
LTFIBRE	**0.36**	0.06	**0.36**	0.08	**0.31**	0.08	**0.29**	0.10	**0.24**	0.11	0.02	0.11
FRQFAT	**0.40**	0.06	**0.27**	0.08	0.13	0.08	0.15	0.10	0.09	0.10	0.04	0.10
FRQSALT	**0.30**	0.06	**0.28**	0.08	**0.18**	0.07	**0.20**	0.09	−0.07	0.08	0.06	0.09
FRQFIBRE	**0.33**	0.06	**0.36**	0.07	**0.24**	0.07	**0.18**	0.09	0.14	0.08	0.11	0.10

[a]Number of pairs.
Note: bold correlations are significant at the 0.05 level.

enabled demographic information to be acquired for a further 45 pairs and 474 single twins, yielding an individual co-operation rate of 83% of contactable individuals.

A total of 3862 individuals aged 18–26 yr responded to the HLQ. Both members of 1613 pairs (71.7% of 2249 pairs) and only one member of 636 pairs (28.3% of 2249 pairs) returned questionnaires. Complete pairs ($n=1613$) contained 6.5% more 'never-married' respondents, 13% more college and university educated respondents and 10% fewer current smokers, than incomplete pairs ($n=636$). The sample had 20% more university and senior secondary school educated persons than in the general Australian population. However, for both complete and incomplete pairs, median scores for the food consumption variables were identical so this would not seem to be a source of information bias.

Food consumption: frequency distributions and correlations

Response frequency distributions are shown in Table 1. Responses to the 'last time consumed high fat, salt and fibre food' variables were concentrated on two of the five response categories while a more diverse range was used for the 'frequency of consumption' variables. We did not observe any sex differences in the median scores for these variables.

Table 2 details, for complete pairs, estimated co-twin polychoric correlations for food consumption variables after listwise deletion of non-respondents, for each of the six sex/zygosity groups (monozygotic females (MZF), monozygotic males (MZM), dizygotic females (DZF), dizygotic males (DZM), dizygotic opposite-sex female first-born (DZFM), dizygotic opposite-sex male first born (DZMF)). Correlations are generally low, but greater among monozygotic twins and females than dizygotic twins and males.

Demographic variables analysed but found not to be significantly associated at the 0.05 level with food consumption were: marital status, education level, employment status and occupation.

Food consumption and personality

Statistically significant (0.01 level) but weak associations were found between personality and the food consumption variables (see Table 3). Findings for females were:

1. negative association between psychoticism and salty food consumption ($r=-0.10$);
2. negative association between extraversion and fatty ($r=-0.14$) and salty ($r=-0.12$) food consumption;
3. positive association between neuroticism and high fibre food consumption ($r=0.14$) and high fat food consumption ($r=0.18$);
4. positive association between harm avoidance and high fat food consumption ($r=0.12$). Increasing fibre consumption was associated with increasing impulsiveness scores ($r=0.11$).

The strongest correlations between personality and food consumption variables were found for males between the lie scale (EPQL) personality trait and the variables 'last time consumed high fat and high salt foods', so these variables were selected for multivariate analysis. The correlation coefficients found were −0.23 and −0.21 for males, and −0.12 and −0.11 for females (see Table 3).

Table 3. Polychoric correlations for food consumption and personality variables for male ($N=1383$) and female ($N=2133$) individuals

		LTFAT	LTSALT	LTFIBRE	FRQFAT	FRQSALT	FRQFIBRE
Psychoticism	Males	0.02	0.00	**0.07**	0.01	0.02	**0.06**
	Females	**−0.08**	**−0.07**	**0.06**	−0.03	−0.04	0.02
Extraversion	Males	−0.06	−0.05	−0.05	−0.02	0.01	−0.01
	Females	**−0.12**	**−0.08**	**−0.07**	**−0.05**	−0.03	**−0.09**
Neuroticism	Males	0.01	0.02	**0.10**	−0.01	0.02	0.05
	Females	**0.06**	0.02	**0.13**	0.02	0.04	**0.10**
Social conformity	Males	**−0.22**	**−0.20**	0.01	**−0.11**	**−0.13**	0.01
	Females	**−0.11**	**−0.12**	0.03	**−0.11**	**−0.09**	0.01
Impulsiveness	Males	−0.02	−0.02	0.06	**−0.06**	0.01	0.03
	Females	**−0.06**	−0.04	**0.07**	−0.03	−0.01	0.03
Novelty seeking	Males	0.06	**0.07**	0.05	0.00	0.03	0.04
	Females	−0.01	−0.01	0.01	−0.02	−0.01	−0.01
Harm avoidance	Males	0.07	**0.08**	**0.08**	**0.07**	**0.06**	0.05
	Females	**0.08**	**0.06**	**0.11**	**0.06**	**0.06**	**0.10**
Reward dependence	Males	**−0.08**	**−0.07**	−0.08	−0.02	−0.05	−0.07
	Females	−0.02	0.02	**−0.11**	**−0.06**	−0.02	**−0.09**

Note: bold correlations are significant at the 0.05 level. No adjustment has been made for multiple testing.

Food consumption: univariate genetic modelling results

In all cases, the hypothesis of no family resemblance (E model) could be rejected (at the 0.05 significance level) although individual environmental variance (including measurement error) was consistently found to be the major influence on variance in food consumption. For the 'last time consumed' variables we did not find significant heterogeneity in the causes of individual differences between males and females but neither could we unequivocally decide between additive genes or shared environment as the cause of familial resemblance. Heritability estimates for the variables 'last time consumed high fat food' (LTFAT), 'last time consumed high salt food' (LTSALT), and 'last time consumed high fibre food' (LTFIBRE) were 0.18 ($\chi^2_5=1{,}98$, $p=0.85$), 0.24 ($\chi^2_5=1.23$, $p=0.94$) and 0.38 ($\chi^2_5=4.48$, $p=0.48$), respectively.

For the 'frequency of consumption' variables, no sex heterogeneity was found for the variables 'frequency of consuming high fat food' (FRQFAT) and 'frequency of consuming high fibre food' (FRQFIBRE) and in both cases the CE model could be rejected and the AE model accepted, heritabilities being respectively 0.32 ($\chi^2_5=5.07$, $p=0.41$) and 0.35 ($\chi^2_5=1.46$, $p=0.92$). For 'frequency of consuming high salt food' (FRQSALT) there was strong evidence for differences in the causes of familial aggregation in males and females since the opposite-sex twin correlations were close to zero while the dizygotic same-sex correlations are greater than zero. The data are most consistent with modest but qualitatively different genetic influences in both sexes.

Food consumption: genetic and environmental covariation

Our multivariate genetic analysis of 'frequency' and 'last time consumed high fat, salt and fibre food' variables confirmed that both additive genetic and shared environmental common fat/salt factors explained familial resemblance in fat and salt consumption since the ACE model ($\chi^2_{105}=129.09$, $p=0.06$) best fitted the female data when they were modified to reflect the higher correlation between fat and salt variables compared with fibre variables (see Table 4). We found that genetic factors had more influence on fat consumption than salt consumption.

Statistically significant heterogeneity differences in additive genetic and shared environmental

Table 4. Correlation matrix for male (below diagonal, $n=1535$) and female (above diagonal, $n=2151$) individuals for 'last time' and 'frequency of high fat, salt and fibre consumption' variables

	LTFAT	LTSALT	LTFIBRE	FRQFAT	FRQSALT	FRQFIBRE
LTFAT	**1.00**	0.60	−0.11	0.57	0.40	0.06
LTSALT	0.71	**1.00**	−0.13	0.49	0.69	0.04
LTFIBRE	−0.19	−0.16	**1.00**	0.00	−0.03	0.75
FRQFAT	0.56	0.43	0.02	**1.00**	0.68	−0.10
FRQSALT	0.46	0.66	0.01	0.72	**1.00**	−0.12
FRQFIBRE	−0.02	0.01	0.80	−0.16	−0.16	**1.00**

Table 5. Multivariate genetic analysis of lie scale personality variable and 'last time' and 'frequency of high fat and salt consumption' variables: four group AE model

	Additive genetic variance				
	A1	A2	A3	A4	
Lie scale	0.44				
LTFAT	0.03*	0.25			
FRQFAT	0.02*	0.20	0.18		
LTSALT	0.04*	0.25	0.00	0.03	
FRQSALT	0.04*	0.19	0.07	0.08	
	Individual environmental variance				
	E1	E2	E3	E4	E5
Lie scale	0.56				
LTFAT	0.01*	0.71			
FRQFAT	0.00*	0.18	0.42		
LTSALT	0.00*	0.23	0.00	0.45	
FRQSALT	0.00*	0.07	0.23	0.19	0.13

Factor loadings shown are the squared path coefficient estimates.
Factor loadings denoted by * have negative path coefficient estimates.

effects were found for males and females ($\chi^2_{27}=40.24$, $p=0.05$). For males (where statistically significant results were not obtained) common additive genetic effects explained much more of the variance in high fat and salt food consumption than they explained for females. Most of the familial resemblance in high fibre food consumption seemed to be explained by specific (i.e. not influencing variance in consumption of high fat or salt food) additive genetic effects for males and specific shared environmental effects for females.

Personality and food consumption: any genetic or environmental covariation?

Our multivariate analysis of the influence of personality on the genetic and environmental components of variance in food consumption indicated that the set of genes influencing variance in the social conformity (EPQ lie scale) personality trait made a small contribution to the genetic effects influencing consumption of high fat and salt food (see Table 5).

The additive genetic and individual environmental model (AE) best fitted the data for both males ($\chi^2_{65}=77.98$, $p=0.13$) and females ($\chi^2_{65}=74.46$, $p=0.20$). The first additive genetic factor absorbs all genetic effects from the personality dimension of social conformity (lie scale). Factors (and labels) in Table 5 are: additive genetic lie scale factor (A1), additive genetic fat factors (A2, A3), additive genetic salt factor (A4), individual environmental lie scale factor (E1), individual environmental fat factors (E2, E3), and individual environmental salt factors (E4, E5). The fifth factor has been set to zero for additive genetic effects because it was zero in the full Cholesky AE model.

The four group (same-sex dizygotic and monozygotic pairs) AE model gave a borderline fit ($\chi^2_{176}=205.42$, $p=0.064$), as is common with most complex multivariate models. Table 5 shows that less than 5% of additive genetic variance in high fat and salt food consumption was common to additive genetic variance in the lie scale personality trait.

Most familial resemblance was explained by common additive genetic fat factors: 25–38% of total variance in the 'last time' and 'frequency of high fat consumption' variables, and 25% of total variance in the salt consumption variables. Therefore, the additive genetic factors influencing variance in the lie scale personality trait are largely different from those influencing variance in the fat and salt consumption variables.

DISCUSSION

The weak co-twin correlations found for our food consumption measures indicated that only modest familial aggregation would be found in high fat, salt and fibre food consumption. We found that additive genetic effects explained most of the familial resemblance in fatty and salty food consumption and that these effects were stronger for males than for females. Our multivariate genetic analyses showed that a common set of genes influenced high fat and salt food consumption and that these were independent of the set of genes and shared environmental effects influencing

high fibre food consumption. Both additive genetic and shared environmental effects contributed to familial resemblance in high fibre food consumption.

Our analyses of the qualitative food consumption measures in the national Health and Lifestyle Questionnaire (HLQ) study of Australian twins aged 18–26 yr did not support the quantitative nutrient density based Falconer *et al.* (1993) findings derived from a general adult population aged 18–70 yr. Nutrient densities are a measure of nutrient intake in terms of total food consumption. Fat, salt and fibre consumption estimates calculated as nutrient densities are more likely to reflect intake of such nutrients unconfounded by individual differences due to size, activity and metabolic efficiency (Willett, 1990). The more precise measures used by Falconer *et al.* to derive fat, salt and fibre consumption estimates probably resulted in greater power to detect personality associations than was possible for our qualitatively based HLQ food consumption measures.

For males, Falconer *et al.* found that neuroticism and psychoticism were negatively correlated with fat intake ($r = -0.25$ and -0.13, respectively). Findings for females were that sodium intake was negatively correlated with psychoticism and positively correlated with extraversion ($r = -0.28$ and 0.22, respectively). We found for females that both psychoticism ($r = -0.10$) and extraversion ($r = -0.12$) were negatively associated with consumption of high salt food and for males high fibre food consumption was positively associated with psychoticism ($r = 0.07$) and neuroticism ($r = 0.10$). However, all correlations were modest and most of the male correlations were not significant (half of the female correlations were not significant).

If the true level of familial aggregation in high fat, salt and fibre food consumption is greater than that identified here, then our HLQ qualitative measures have failed to adequately discriminate the true range of consumption frequencies for the 18–26 yr age group. Since the correlations found between nutrient density and personality by Falconer *et al.* (1993) were much stronger than our correlations between food consumption and personality, one possibility is that our food consumption measurement errors are too large.

Reliability and validity of our food consumption measures are uncertain since neither test–retest data nor external validating information are available. Also we cannot identify the proportion of measurement error in individual environmental variance. Alternative food consumption measures, such as those in the short fat questionnaire devised by Dobson, Blijlevens, Alexander, Croce, Heller, Higginbotham, Pike, Plotnikoff, Russell and Walker (1993) may yield a more diverse range of variance estimates, but would also considerably increase respondent burden in our study, which was part of a larger health and lifestyle survey. Estimates may be biased since they have been derived from a voluntary sample. We compared demographic characteristics of our sample with that in the 1986 Census and found that our voluntary sample is representative of the Australian population in terms of occupational status and marital status for females. The sample over-represents persons with university and senior secondary school education (Baker, Treloar, Reynolds, Heath & Martin, 1996). This may have reduced our (genetic) variance estimates for high fat and fibre food consumption since it has been found that lower educational status groups prefer high fat and low fibre foods (Smith & Baghurst, 1992).

Multivariate analysis of the contribution of the personality dimension of social conformity (EPQ lie scale) to genetic and environmental variance and covariance in food consumption indicated that additive genetic effects on social conformity had a minor influence on additive genetic variance in consumption of high fat and salt food. Additive genetic effects associated with variance in this personality trait differed from those associated with consumption of high fat and salt food.

In summary we find only modest evidence of genetic influences on self-reported fat, salt and fibre consumption of 18–26 yr olds and still more modest influences of personality variables—the most significant being that those with higher social conformity (lie) scores self-reported lower fat and salt consumption. However, even this association may be due to dissembling. A possible explanation for our negative findings is the qualitative nature of our food consumption data.

Acknowledgements—This work was supported by grants from the National Health and Medical Research Council of Australia and from the National Institute of Alcoholism and Alcohol Abuse (NIAAA), U.S.A. We thank Theresa Pangan and John Pearson for computing assistance.

REFERENCES

Baker, L., Treloar, S., Reynolds, C., Heath, A. & Martin, N. (1996). Genetics of educational attainment in Australian twins: Sex differences and secular changes. *Behavior Genetics*, *26*, 89–102.

Cloninger, C., Przybeck, T. & Svakic, D. (1991). The tridimensional personality questionnaire: U.S. normative data. *Psychological Reports, 69*, 1047–1057.

Dobson, A., Blijlevens, R., Alexander, H., Croce, N., Heller, R., Higginbotham, N., Pike, G., Plotnikoff, R., Russell, A. & Walker, R. (1993). Short fat questionnaire: a self-administered measure of fat-intake behaviour. *Australian Journal of Public Health, 17*, 144–149.

Eaves, L. & Eysenck, H. (1975). The nature of extraversion: A genetical analysis. *Journal of Personality and Social Psychology, 32*, 102–112.

Eysenck, S., Eysenck, H. & Barrett, P. (1985). A revised version of the psychoticism scale. *Journal of Personality and Individual Difference, 6*, 21–29.

Falconer, H., Baghurst, K. & Rump, E. (1993). Nutrient intakes in relation to health-related aspects of personality. *Journal of Nutrition Education, 25*, 307–318.

Heath, A., Cloninger, C. & Martin, N. (1994). Testing a model for the genetic structure of personality: A comparison of the personality systems of Cloninger and Eysenck. *Journal of Personality and Social Psychology, 66*, 762–775.

Hopkins, P., Williams, R., Kuida, H., Stults, B., Hunt, S., Barlow, G. & Ash, K. (1989). Predictive value of a short dietary questionnaire for changes in serum lipids in high-risk Utah families. *American Journal of Clinical Nutrition, 50*, 292–300.

Jardine, R., Martin, N. & Henderson, A. (1984). Genetic covariation between neuroticism and the symptoms of anxiety and depression. *Genetic Epidemiology, 1*, 89–107.

Jöreskog, K. & Sörbom, D. (1993). *Prelis* 2: *Lisrel* 8 *and Prelis* 2 *Documentation*. Chicago: Scientific Software International.

Kasriel, J. & Eaves, L. (1976). A comparison of the accuracy of written questionnaires with blood-typing for diagnosing zygosity in twins. *Journal of Biosocial Science, 8*, 263–266.

MacFie, H. & Thomson, D. (Eds.) (1994). *Measurement of food preferences*. London: Blackie Academic and Professional.

Martin, N. & Martin, P. (1975). The inheritance of scholastic abilities in a sample of twins. I. Ascertainment of the sample and diagnosis of zygosity. *Annals of Human Genetics, 39*, 213–218.

Neale, M. & Cardon, L. (1992). *Methodology for genetic studies of twins and families*. Dordrecht: Kluwer Academic.

Nutbeam, D., Wise, M., Bauman, A., Harris, E. & Leeder, S. (1993). *Goals and targets for Australia's health in the year* 2000 *and beyond*. Sydney: University of Sydney.

Statistical Analysis Systems Institute Inc. (1989). *SAS/STAT User's guide, Release* 6.03. Cary, NC: SAS Institute Inc.

Smith, A. & Baghurst, K. (1992). Public health implications of dietary differences between social status and occupational category groups. *Journal of Epidemiology and Community Health, 46*, 409–416.

Willett, W. (1990). *Nutritional epidemiology*: *Monographs in epidemiology and biostatistics*, Vol. 15. New York: Oxford University Press.