The role of genes in tobacco smoking during adolescence and young adulthood: a multivariate behaviour genetic investigation

Victoria M. White¹, John L. Hopper², Alexander J. Wearing³ & David J. Hill¹

Centre for Behavioural Research in Cancer, The Cancer Council Victoria, Victoria, Australia

Department of Genetic Epidemiology, University of Melbourne, Parkville, Victoria, Australia

Department of Psychology, University of Melbourne, Parkville, Victoria, Australia

ABSTRACT

Aims To incorporate a psychosocial model of tobacco smoking into a behaviour genetic design to examine genetic and environmental influences on variation in smoking involvement.

Design Longitudinal twin study.

Setting and participants Twins initially aged between 13 and 18 years and registered with the Australian Twin Registry were surveyed three times between 1988 and 1996. A total of 414 pairs of identical and same-sex fraternal twin pairs participated in all three surveys, aged between 20 and 25 at wave 3. Biometric modelling estimated the influence of genetic and environmental factors in determining variation in smoking at each wave, both before and after adjusting for perceived smoking behaviours of peers and parents.

Measurements Twins answered a questionnaire on their own smoking status and reported on the use of tobacco by parents and friends as they perceived it, at each survey wave.

Findings At all three surveys, current smokers were more likely to have parents who smoked and to have smokers among their peers. Genes and environmental factors, both common and unique, contributed to variation in smoking behaviours. However, after controlling for the smoking behaviours of peers and parents, the role of genes in determining variation in smoking involvement was reduced by 100% at wave 1 and by 30% at wave 2. Friends' smoking reduced the magnitude of the common environment variance by 11%, 30% and 40% at waves 1, 2 and 3, respectively. Parents' smoking behaviours explained part of the common environment. Biometric modelling of the covariation between smoking involvement and peer smoking suggested that genes might influence smoking involvement at wave 1 by influencing choice of peers.

Conclusion Environmental factors play the greatest role in determining variation in tobacco smoking among adolescents and young adults. Among adolescents, genes may influence variation in smoking behaviours indirectly by influencing choice of peers. However, genes seem to have a direct influence on variations in the smoking behaviours of young adults.

KEYWORDS Genetics, parental smoking, peer smoking, tobacco smoking; twins.
INTRODUCTION

As in other western countries, for nearly 30 years Australians have been exposed to health warnings and education campaigns about the negative health effects of smoking tobacco, yet at the end of the 20th century, 30% of senior secondary students were current smokers (Hill, White & Effendi 2002). Understanding why adolescents start using tobacco, and why some progress to regular use, is critical for the development of effective prevention programmes. A great deal of research has now been conducted into the factors associated with experimental tobacco use and the progression to regular use (for reviews see Conrad, Flay & Hill 1992; US Department of Health and Human Services 1994; Tyas & Pedersen 1998). However, to date most school-based intervention programmes developed from this body of research have produced only short-term reductions in the proportion of adolescents smoking (Ellickson, Bell & McGuigan 1993; US Department of Health & Human Services 1994; Peterson et al. 2000). These programmes’ lack of success in reducing smoking in the long term (i.e. as adults) has led Swan (1999) to conclude that our understanding of the factors leading some adolescents to regular smoking is incomplete, and that past approaches to adolescent smoking need to be reconsidered.

Several studies of adult twins have suggested that genetic factors account for around 50% of the variance in adult smoking behaviours (Swan et al. 1990; Carmelli et al. 1992; Heath & Martin 1993; Heath et al. 1993; Heath et al. 1999; Madden et al. 1999). Consequently, Swan (1999) has argued that models of smoking initiation need to be broadened to include a role for genetic variability. In the study we present below, we have combined a social psychological approach to smoking initiation with the theory and analytical methods of behaviour genetics, to investigate how genetic factors influence smoking involvement among adolescents and young adults—the key periods for smoking initiation and consolidation.

Traditionally, smoking has been seen as a volitional, socially motivated behaviour studied most commonly within the field of social psychology (Chassin, Presson & Sherman 1990). The terms ‘smoking initiation’ and ‘smoking uptake’ are used to refer to the process of becoming a current smoker. It is postulated commonly that smoking initiation involves a progression through a series of stages that takes individuals from not smoking, to trying or experimenting with tobacco, to using tobacco regularly to finally becoming dependent on nicotine (Leventhal & Cleary 1980; Flay et al. 1983). Social learning theory (Bandura 1986) has been one of the main theoretical models used to study the process of smoking initiation. This model suggests that an adolescent’s involvement with role models who use tobacco leads to three sequential effects: (i) observation and imitation of tobacco use; (ii) reinforcement of behaviour; and (iii) formation of positive expectations about the social, psychological and physiological consequences of future tobacco use. According to this theoretical position, the anticipated consequences of smoking are largely social during the early stages of smoking initiation and become increasingly physiological during the latter stages (i.e. regular use). Thus social learning theories suggest that the smoking behaviours of important others are potent influences on smoking when it is first being learnt, but that this influence diminishes as use continues and as other factors such as the need to maintain nicotine levels become more important.

Friends and parents are important role models for adolescents, and thus their behaviours regarding tobacco use are thought to influence an adolescent’s use of tobacco (Jessor & Jessor 1977; Chassin et al. 1990; Petrakis, Flay & Miller 1995; Flay, Petrakis & Hu 1999). There is strong support for this proposition. Cross-sectional studies investigating smoking initiation have found consistently that adolescents who smoke have more friends who smoke and are more likely to have parents who smoke (for reviews see Conrad et al. 1992; Tyas & Pedersen 1998).

Longitudinal studies show that the smoking behaviours of friends predict the transition from never smoking to any smoking (e.g. Collins et al. 1987; Flay et al. 1994; Distefan et al. 1998; Flay, Hu & Richardson 1998), as well as influencing transitions in the later stages of the smoking initiation process (Collins et al. 1987; Distefan et al. 1998; West, Sweeting & Ecob 1999; Chassin et al. 2000). While results from longitudinal studies examining the influence of parental smoking on adolescent smoking behaviours are inconsistent, there is some evidence indicating that parental smoking also predicts smoking initiation (Flay et al. 1998; Engels et al. 1999).

Associations between the smoking behaviours of parents and their children may be due to an underlying genetic basis to smoking. Twin studies offer a method for quantifying, under specific assumptions (see below), the influences of genes and the environment on variation in behaviours, while controlling for the influence of age and, in same-sex twin pairs, gender. Behaviour genetics is based on the assumption that if a trait or behaviour is influenced by genes then the strength of genetic relationship between individuals (such as sibling, parent–child and grandparent–grandchild pairs) will determine similarity on the trait or behaviour. The classic twin study design explains individual variation in terms of three general variance components: genetic, shared family or ‘common’ environment and individual-specific environment. Studies generally focus on presenting estimates of the amount of variance attributable to each component.
and in particular define the ‘heritability’ of a trait as the proportion attributable to genetic factors. Classic twin studies rely heavily on the assumption that the effect of shared environment is independent of zygosity, so they must be seen as providing an upper estimate of heritability, given that genetically identical pairs may well share non-genetic factors more strongly than fraternal twins.

Four twin studies have examined the influence of genes on variation in adolescent smoking (Boomsma et al. 1994; Han, McGue & Iacono 1999; Koopmans et al. 1999; Maes et al. 1999). This work has generally investigated life-time use of tobacco (assessed by the question ‘have you ever smoked?’) and thus has examined the genetic and environmental influences on the transition from a never smoker to experimenter. Generally, these studies suggest that both genetic and environmental factors influence variation in smoking experimentation, with the common environment making the largest contribution (around 50% of the variance), while genetic factors account for around 35% of the variance. The exception was Maes et al. (1999), which concluded that genes but not the common environment explained the variation in the smoking behaviour of 16-year-old-twins from Virginia, USA. Several limitations reduce the strength of the conclusions that may be reached from the findings of these studies. First, as the pattern of regular smoking is usually acquired by the age of 18, only two twin studies have studied the smoking behaviours of twins in the process of smoking initiation (Han et al. 1999; Maes et al. 1999). As Boomsma et al. (1994) and Koopmans et al. (1999) include in their samples twins going through the tobacco initiation phase (age 13–18 years) and twins in the smoking consolidation period (18–25 years) their findings are uninformative regarding the importance of genes in the process of smoking initiation. While both Han et al.’s (1999) and Maes et al.’s (1999) studies are able to answer the question regarding the role of genes on smoking initiation, their divergent results means the role of genes in smoking initiation is unclear.

Secondly, these studies focus on analysing binary measures of smoking status most commonly ever smoked versus never smoked. The appropriateness of studying smoking initiation as a binary variable can be questioned on several grounds. The notion of an adolescent as a smoker or not is more ambiguous and fluid than it is among adults. Since the 1980s, prevalence studies of adolescent smoking behaviours have shown that while most adolescents try a cigarette, less than half of these become regular daily smokers (Hill et al. 1990; Hill et al. 1993; Hill, White & Segan 1995). Additionally, a longitudinal study of final-year secondary school students, found that around 20% of adolescents who described themselves as an ‘occasional smoker’ at one survey wave called themselves a ‘non-smoker’ at the next survey 6 months later (Schofield et al. 1998). As discussed above, psychosocial examinations of smoking uptake have postulated a stage model. By using a single question to assess smoking among adolescents, behaviour genetic studies of smoking are examining, in the main, genetic influences in the transition from never smoker to experimenter. Because most twin studies of smoking assess ‘ever smoking’ they categorize current regular smokers with past regular smokers and current and past experimental smokers in the same group. This strategy is likely to confound the smoking phenotype under investigation, as it classifies experimenters and regular smokers similarly.

Twin studies of smoking initiation might benefit by moving away from a binary assessment of smoking to an assessment that incorporates the concept of smoking uptake as a progression through a number of stages and begin to utilize measures of smoking involvement. Several investigators examining psychosocial influences on smoking initiation have used an indicator of overall smoking involvement to examine smoking uptake (Newcomb, McCarthy & Bentler 1989; Graham, Marks & Hansen 1991; Engels et al. 1999). These indices attempt to assess the individual’s past and current use of tobacco and thereby capture the process of smoking initiation in one measure. The present study adopts this approach and uses a variable assessing smoking involvement as an indicator of smoking initiation and consolidation.

Swan et al. (1990) noted that estimates of the variance in appetitive behaviours explained by genes may be biased if other sources of variance underlying twin similarities are not included in the modelling. None of the adolescent twin studies discussed above have included measures of peer smoking in their models. Hopper et al. (1992) have modelled the likelihood of being a smoker given the smoking status of the co-twin and friends in their study of the smoking behaviours of adolescent Australian twins. They found that the likelihood of a twin smoking was about six times greater if at least one friend smoked than if no friends smoked, similar to the odds of smoking among dizygotic (DZ) pairs. They suggested that the greater similarity in the smoking behaviours of monozygotic (MZ) twin pairs may be a function of these twins spending more time together than did DZ pairs. Their results also suggested that peer smoking is an important factor that should be included in biometric models of smoking initiation.

The present study extends the work of Hopper et al. (1992) and attempts to obtain estimates of the role of genes in explaining variation in smoking involvement after controlling for parental smoking and peer smoking, two known correlates and predictors of adolescent smoking.
The data for this study are taken from three waves of a longitudinal study of adolescent twins growing into young adulthood. Twins answered questionnaires about their smoking behaviours, first when they were aged between 13 and 18 years, the second 3 years later and the third a further 4 years after the second survey. When twins were aged 20–25 years. The development of regular smoking usually occurs during adolescence, with the smoking habit consolidating during young adulthood (US Department of Health & Human Services 1994). Because the 7-year period of this study includes adolescence and young adulthood the role of genetic and environmental factors on smoking involvement and consolidation could be examined. The focus of this current paper is the biometric modelling of smoking involvement at each survey wave. This allows us to examine the role of genes and the environment in determining smoking involvement at key points in the smoking uptake and consolidation process.

**METHOD**

**Procedure**

Data for this study are taken from the Australian Adolescent and Young Adult Twin study, a longitudinal study examining smoking and alcohol behaviour. Twins for this study were recruited via the Australian Twin Registry. Repeated data from the twins who were aged between 13 and 18 years of age at wave 1 were utilized in this study.

In 1987, the parents of 2967 twin pairs aged between 11 and 18 years then registered with the volunteer Australian Twin Registry were approached to enrol their twin children in a longitudinal study on the health and lifestyle of adolescent twins. Of these parents, 1450 (49%) consented. During 1988, questionnaires were mailed to twins and completed questionnaires were received from 2892 twins, of whom 1400 were twin pairs representing a 97% pairwise response. During 1991, parents and their twins were re-approached by letter and asked to complete the wave 2 survey. At wave 2, 2280 completed questionnaires were returned, which included 1121 twin pairs. Four years later, the wave 3 questionnaire was mailed to all twins participating in the wave 1 survey. A total of 1843 individuals returned the questionnaire and the sample consisted of 739 twin pairs.

**Measures**

Three self-report questionnaires were developed for this study. Only those questions assessing smoking behaviours of respondents and family and peers are detailed here.

**Smoking status**

At waves 1 and 2, four questions asked respondents about their use of tobacco: in their lifetime, in the past 12 months, in the last 4 weeks and in the last 7 days. Lifetime experience of tobacco was assessed by respondents indicating if they had smoked even part of a cigarette, choosing one of the following responses: 'No', 'Yes, just a few puffs', 'Yes, I have smoked fewer than 10 cigarettes in my life', or 'Yes, I have smoked more than 10 cigarettes in my life'. Respondents then indicated whether they had smoked a cigarette in the last 12 months and in the last 4 weeks. Use of tobacco in the last week was assessed by a day-specific record, where respondents indicated the number of cigarettes consumed, if any, on each of the 7 days prior to completing the survey.

At wave 3, respondents answered 'yes' or 'no' to the questions: 'Have you ever smoked more than 10 cigarettes in your life?' and 'Have you smoked in the last four weeks?'. Smoking in the last week was again assessed by use of a day-specific record, as previously.

Based on responses to these questions, subjects were classified into four groups reflecting an increasing involvement with tobacco. The groups were: never smokers—individuals who reported never having had even a puff of a cigarette; triers—individuals who had tried smoking but had not had as many as 10 cigarettes; experimenters—individuals who had had more than 10 cigarettes but had not smoked in the week prior to being surveyed; and regular users—individuals who had smoked more than 10 cigarettes and had smoked in the previous week. The classification system used here and the definition of regular or experimental smoker follows that outlined by Mayhew, Flay & Mott (2000). They (2000) showed that a regular smoker is commonly defined as smoking in the week prior to the survey in psychosocial investigations of smoking initiation.

At wave 3, respondents who had not smoked more than 10 cigarettes in their lifetime and had indicated that they had never smoked even part of a cigarette in the wave 1 and wave 2 questionnaires were classified as never smokers. Respondents who had smoked less than 10 cigarettes at either wave 1 or wave 2, and had not smoked more than 10 cigarettes at wave 3, were classified as triers. Respondents who had smoked more than 10 cigarettes in their lifetime, but had not smoked in the last week, were classified as experimenters. Those who had smoked in the last week, and had smoked more than 10 cigarettes in their lifetime, were classified as current smokers. Because a current smoker at wave 3 needed to have smoked in the previous week, respondents who had smoked regularly at wave 1 or wave 2, but had not smoked in the last week at wave 3, were classified as experimenters. Because never smoking was not assessed...
at wave 3, some twins who smoked less than 10 cigarettes for the first time between waves 2 and 3 might have been classified as never smokers at wave 3 rather than triers. However, as data from the current study showed that the proportion of 18-year-olds smoking between one and 10 cigarettes was only about 2% more than the proportion among 16-year-olds, we believe any misclassification would be small and have minimal impact on results.

An index of overall tobacco involvement, based on smoking stage, was then created following procedures outlined by Hill et al. (1987). Weights were assigned to reflect an individual’s stage of smoking as follows: a never smoker was assigned a score of 1; anyone who had tried smoking was assigned a score of 5; if someone had experimented with smoking they were given an additional score of 5; and someone who was a current smoker was given an additional score of 10. The number of days the individual had smoked in the past week was then added to the index score, as it captures the regularity of smoking among adolescents. At each survey this index ranged from 1 to 27.

Smoking behaviours of parents and peers

In each survey, respondents were asked to indicate the perceived smoking status of their mother, father, twin and for each of up to four friends. Respondents classified each relative and friend as either a ‘non-smoker’, an ‘ex-smoker’, or a ‘smoker’. Respondents also classified their parents as ever smokers or non-smokers, and for the purposes of this study parental smoking was classified as: (a) one or both parents had been a smoker, or (b) both parents were non-smokers.

An index referred to as the peers’ smoking index was calculated from the proportion of respondents’ friends who were smokers. At each wave the number of friends listed by a subject was calculated and the smoking status of these friends assessed. The proportion of friends who were smokers at each data wave was determined by dividing the number of smoking friends by the total number of friends listed. This was then multiplied by 100 to give an index ranging from 0, indicating no friends smoked, to 100, indicating that all friends smoked.

Statistical analyses

Phenotypic analyses

The associations between age, gender, parental and peer smoking and respondent’s smoking status was examined at each of the three survey waves. The statistical package STATA (StataCorp 1999) was used for these analyses, as it permits the calculation of standard errors (SE) robust to the non-independence of data from both members of a twin pair. In these analyses, the twin pair identifier was the clustering unit and the Huber/White/sandwich estimator of variance was used to calculate standard errors.

Biometric modelling

As the basis of biometric analyses was to determine the causes of variation between individuals, and that variation depends on how the trait mean is modelled, the mean of the trait was first defined (Hopper 2000). Genetic and environmental models of variation about this mean were then fitted following the model \( Y_i = \mu + G + C + E \), where \( Y_i \) is the trait score for twin \( i \) of a pair \((i = 1, 2)\), \( \mu \) is the mean, and \( G \) refers to additive genetic effects, \( C \) refers to non-genetic factors common to the twin pair (common environment), and \( E \) refers to effects specific to the twin and includes measurement error. These variables are assumed to be independent and to each follow a normal distribution with mean 0 and variances \( \sigma^2_g \), \( \sigma^2_c \) and \( \sigma^2_e \), respectively. The total variance is \( \sigma^2 = \sigma^2_g + \sigma^2_c + \sigma^2_e \). According to the classic twin model, the pair covariance associated with C is \( \sigma^2_c \) irrespective of the zygosity of the pair (that is, common environment effects are assumed to be the same within MZ and DZ pairs). The unique environmental component, \( E \), creates differences rather than similarities between twins within a pair. As indicated above, this component also includes random measurement error.

Models were fitted by maximum likelihood under the assumptions of a multivariate normal distribution (Hopper et al. 1998). Four basic models were fitted: model I: \( \sigma^2_g = 0 \) and \( \sigma^2_c = 0 \) [the unique environment only model (E)]; model II: \( \sigma^2_c = 0 \) [the genetic (A) and unique environment model (AE)]; model III: \( \sigma^2_e = 0 \) [the common environment (C) and unique environment model (CE)]; and model IV: no variance component constrained (the full ACE model). The first model fitted to the data was the full model (IV), then models II and III, and finally model I. For each model, maximum log likelihood estimates were calculated. For nested models, the change in the log likelihood can be used to indicate the significance of the parameter that has been removed or added. Twice the difference in the log likelihood has an approximately \( \chi^2 \) distribution, with degrees of freedom equal to the difference in the number of parameters between the nested models, under the null hypothesis that the new parameter(s) are zero. There are no formal tests of significance for making comparisons between non-nested models (e.g. when comparing the AE model with the CE model). The Akaike Information Criterion (AIC), defined as \(-2 \maximized \text{log likelihood} + 2 \text{(number of parameters)}\) was used to compare the AE and CE biometric models. The model with the smallest AIC is considered to be the most parsimonious (Macaskill et al. 1994). As these two models have the...
same number of parameters, the AIC is defined by the maximised log likelihood. These procedures were used to determine the best-fitting genetic–environment model.

Standard errors were estimated under asymptotic likelihood theory, and were used for statistical testing of differences between estimated correlations.

Including psychosocial variables in genetic and environmental models

As Hopper and colleagues have demonstrated (Hopper et al. 1998; Harrap et al. 2000), the mean can be adjusted for covariates and biometric modelling conducted on the residual variance. Following methods described by Hopper et al. (1998), this study included psychosocial variables in the genetic and environmental models by adjusting the means of smoking involvement for parents’ and peers’ smoking.

Mean smoking involvement was modelled as a function of age and the covariates as:

\[ \mu = a_0 + a_1 x_{1i} + a_2 x_{2i} + \ldots + a_q x_{qi}, \]

where \( a_0 \) is the intercept, \( a_1 x_{1i} \) is the first covariate, \( a_2 x_{2i} \) is the second covariate and so on. The four biometric models described above (ACE, AE, CE and E) were then fitted to the residual values—which were approximately normally distributed—following the procedure described above. Again, the relative fit of each of the four models was assessed. The parameter estimates obtained for the residual variance could then be compared with those obtained for models of the variance around the mean adjusted only for age. This comparison allowed for the effects of the covariates on each of the total, genetic, common environment and unique environment, variance components to be estimated. Change in the different variance components as a result of adjusting for the covariate indicated that the covariate was contributing to the genetic, common environment and/or unique environment variance of smoking involvement.

Modelling covariation between traits

If the covariate analyses described above indicated that there was a ‘genetic’ association between smoking involvement and peer or parental smoking, biometric modelling of the covariation between the two variables was undertaken. In these analyses, the two traits (e.g. smoking involvement and peer smoking) were modelled and the four biometric models described above (ACE, AE, CE and E) were then fitted to the covariation between these two variables following the procedure described above. The relative fits of the nested and non-nested models were assessed using the procedures described above.

Cross-twin, cross-variables correlations were determined by correlating, for example, twin 1’s score on smoking involvement with twin 2’s score on peer smoking and vice versa.

Biometric models were fitted using the statistical package FISHER (Lange, Boehnke & Weeks 1987).

Sample description

This investigation utilized data for twins aged between 13 and 18 years of age at wave 1. At wave 1 there were 1163 twin pairs in this age range. Of these, 574 twin pairs responded at wave 3. Of these twin pairs, 42% were MZ pairs, 36% were same-sex DZ pairs and 22% were opposite-sex DZ pairs. Only data from 414 same-sex twin pairs were used in phenotypic and biometric modelling of smoking involvement. Both members of these pairs participated in all three surveys and had no missing data at each data wave. As we expected male and female twins in these pairs to have different sets of friends, their inclusion in the dataset may confound associations. For this reason we excluded data from opposite sex twins from our analyses.

RESULTS

Cross-sectional associations between respondent’s smoking status and the smoking behaviours of friends and parents

Table 1 shows that the proportion of respondents involved with smoking increased as the cohort aged. At each wave there was no association between smoking status and gender, while age was associated with smoking status at wave 1 (\( F_{1,441} = 30.23, df = 1, p < 0.01 \)) and wave 2 (\( F_{1,441} = 4.05, df = 1, p < 0.05 \)), showing that older respondents were more likely than younger respondents to be current smokers across the ages 13–21. There was no linear association between age and smoking status at wave 3, by which time the youngest respondent was 20 and the oldest was 26.

Table 1 Proportion of never smokers, triers and experimenters among individual twins at each survey wave.

<table>
<thead>
<tr>
<th>Wave</th>
<th>Age (years)</th>
<th>Wave 1</th>
<th>Wave 2</th>
<th>Wave 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wave 1</td>
<td>(13–18)</td>
<td>35%</td>
<td>23%</td>
<td>21%</td>
</tr>
<tr>
<td>Wave 2</td>
<td>(16–21)</td>
<td>39%</td>
<td>39%</td>
<td>25%</td>
</tr>
<tr>
<td>Wave 3</td>
<td>(20–25)</td>
<td>18%</td>
<td>20%</td>
<td>26%</td>
</tr>
</tbody>
</table>

*Smoked up to 10 cigarettes in lifetime.

**Smoked more than 10 cigarettes but did not smoke in week prior to survey.

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On average, about 40% of respondents reported that neither parent smoked. The proportion of respondents for whom one or both parents smoked decreased across the survey period from 37% at wave 1 to 26% at wave 3. This decrease was accompanied by a corresponding increase in the number of ex-smokers among parents from 26% at wave 1 to 40% at wave 3. There was an increase in the mean friends smoking index between waves 1 and 3 from 23 to 32.

Cross-sectional associations examined whether the level of smoking among parents and friends varied as a function of the individual’s smoking status. The proportion of respondents in each of the smoking status groups reporting that at least one of their parents smoked at each wave is shown in Table 2. At all waves, respondents who had never smoked had fewer smokers among their friends than did regular users, experimenters or triers. After controlling for gender and age, the association between smoking status and peer smoking was significant at all time periods (wave 1: $F_{1,4,10} = 87.46$, $P < 0.01$; wave 2: $F_{1,4,10} = 95.79$, $P < 0.01$; wave 3: $F_{1,4,10} = 57.97$, $P < 0.01$). Post-hoc analyses indicated that differences between all pairs of groups were significant. Current users of tobacco had more friends who smoked than experimenters, who had more friends who smoked than triers and never smokers.

Cross-sectional biometric modelling

The index of overall smoking involvement was used to examine the role of genes and the environment as causes of variation in smoking. Mean scores on this index for non-smokers, triers, experimenters and current users at each wave are shown in Table 3. At all waves, respondents who had never smoked had fewer smokers among their friends than did regular users, experimenters or triers. After controlling for gender and age, the association between smoking status and peer smoking was significant at all time periods (wave 1: $F_{1,4,10} = 87.46$, $P < 0.01$; wave 2: $F_{1,4,10} = 95.79$, $P < 0.01$; wave 3: $F_{1,4,10} = 57.97$, $P < 0.01$). Post-hoc analyses indicated that differences between all pairs of groups were significant. Current users of tobacco had more friends who smoked than experimenters, who had more friends who smoked than triers and never smokers.
increased significantly between waves 1 and 2 (Z-scores > 2.60, P < 0.01) but not between waves 2 and 3 (Z-scores < 1.96, P > 0.05).

Before biometric modelling took place, the distributions of the sex and age adjusted measures were checked for deviations from normality. The distributions of scores on all three variables were positively skewed, so they were log transformed. The residuals of the age-adjusted transformed scores were then found to be approximately normally distributed.

Table 4 shows the intraclass correlations for smoking involvement between MZ and DZ pairs at waves 1, 2 and 3, for male and female identical (MZ) pairs and same-sex non-identical (DZ) pairs and the maximum log-likelihood estimates for the four genetic and environment variance component models (same-sex twins only).

When data for male and female twins were modelled together, the ACE model provided the best fit to the data at all three waves. At wave 1, genetic factors accounted for 15% of the variance in smoking involvement while the common environment accounted for around 55%. At wave 2, genes accounted for 20% of the variance while the common environment accounted for around 50%. At wave 3, the common environment and genes both accounted for around 35% of the variance in smoking involvement.

Integrating social influence variables into the genetic and environmental models of smoking behaviour among all same-sex twin pairs

This section presents the results from the biometric modelling of variation in smoking involvement after adjusting
How do genes and friends influence smoking initiation?

for smoking behaviours of parents and peers, both individually and together. Analyses combine data from both male and female twin pairs.

Adjusting for peer smoking

Table 5 shows the maximum log-likelihood parameter estimates for the ACE model of the unadjusted and adjusted mean for all same-sex twin pairs.

After adjusting smoking involvement at wave 1 for peer smoking, the estimate of the genetic variance component reduced by 100% while the common environment estimate changed minimally (by 11%). For wave 2 smoking involvement, after adjusting the mean for peer smoking, the genetic variance component reduced by about 37%, while the common environment component of variance reduced by about 30%. At wave 3 adjusting for peer smoking reduced the common environment component of variance by around 20%. At wave 3, adjusting smoking involvement for parental smoking changed the genetic variance component by 14% and reduced the common environment component of variance by about 20%.

Adjusting for both peer and parental smoking

Adjusting for both peer and parental smoking reduced the variance estimates for both the genetic and common environment components at all three waves. The genetic component of variance was reduced at wave 1 by 100% and at wave 2 by 31%. The reduction in genetic variance at wave 1 was significant (Z = 3.3). At both waves this reduction was due to controlling for peer smoking. At wave 3, adjusting for both peer and parental smoking had no effect on the estimate of the genetic component of variance. Adjusting for peer and parental smoking reduced the common environment component by 19% at wave 1, 43% at wave 2 and 60% at wave 3. This reduction was significant at wave 2 (Z-score = 1.98) and wave 3 (Z-score = 1.98).

Adjusting for parental smoking

At wave 1, adjusting smoking involvement for parental smoking had little effect on either the genetic or common environment variance component. Adjusting smoking involvement for parental smoking at wave 2 had minimal impact on the estimate of genetic variance but reduced the common environment component by around 20%.

Modelling covariance between smoking involvement and peer smoking

The results presented in the previous section suggest that if genes influence smoking behaviours of adolescents, this influence is indirect and works largely through genetic influences on an individual’s choice of friends. To exam-
In this possibility in more detail we modelled the covariation between peer smoking and smoking involvement as a function of genes and environmental factors. Before these analyses could be undertaken, we needed to determine that genes influenced variation in peer smoking behaviour. The intraclass correlations for peer smoking among MZ pairs and DZ pairs were: wave 1, MZ: 0.46, DZ: 0.48; wave 2, MZ: 0.51, DZ: 0.43; wave 3: MZ: 0.41, DZ: 0.38—suggesting genes might influence variation in peer smoking. Biometric modelling suggested that the ACE model (ML = 623.4) was the best fit to the data (ACE: ML = 619.5; CE: ML = 619.7; E: ML = 544.3). At wave 2, the CE model (ML = 497.6) provided the best fit to the data (ACE: ML = 498.1; AE: ML = 495.2; E: ML = 458.0), while the AE model (ML = 595.1) was the best model at wave 3 (ACE: ML = 595.2; CE: ML = 592.7; E: ML = 569.4). As the results suggest that genes play a role in determining variation in peer smoking behaviour at waves 1 and 3 and might play a role at wave 2, we progressed to modelling the covariation between smoking involvement and peer smoking in terms of genes and the environment. The results of these analyses along with the cross-twin correlations are shown in Table 6. The cross-twin correlations for MZ pairs were higher than those for DZ pairs at all three time periods; however, these differences were significant only at wave 1 (change in $\chi^2 = 15.21$, df = 1, $P < 0.001$). These results suggest that genes might play some part in determining the covariation between smoking involvement and peer smoking at wave 1. Biometric modelling confirmed this with the AE model being the preferred model at wave 1 while the CE models were preferred at waves 2 and 3.

### DISCUSSION

In the analyses presented here, we have demonstrated a new approach to the investigation of genetic contributions to smoking involvement during the period when people are most likely to become smokers—adolescence and young adulthood. Our results take into account the contributions of other known correlates of smoking initiation and smoking involvement. Because this approach treats the biometric analyses of smoking involvement as a multivariate problem it provides more information on the influence of genes in determining variation in smoking involvement. This study’s conclusion was that the common environment was the most important source of variation in smoking involvement during adolescence. Any influence of genes on smoking involvement during adolescence was minimal and likely to be due to genetic influences on choice of friends. However, among older adolescents and young adults, both the common environment and genes contributed almost equally to explaining familial aggregation in smoking behaviours. These results suggest that genes might have a stronger and more direct role in determining variation in continued use of tobacco and perhaps dependence on nicotine.

Most previous behaviour genetic studies of adolescent smoking have used a binary indicator of smoking and have used the polychoric correlations to estimate the heritability of the ‘liability’ to smoke. In contrast, the present study used a series of questions to assess particular aspects of smoking status and developed an index of actual smoking involvement. Thus this study examined genetic and environmental influences on actual smoking behaviours rather than the liability to smoke. Although it could be argued that because this indicator of smoking involvement includes ever smoking as well as the number of days smoked in the past week, it confounds genetic influences on smoking initiation with those on smoking persistence, we do not believe this to be the case. The argument arises from discussions about whether smoking initiation and smoking persistence are part of the same continuum of liability to smoke and whether the same genetic and environmental factors determine smoking initiation and smoking persistence (Hannah, Hopper & Mathews 1985; Heath & Martin 1993; Madden et al. 1999). The evidence suggesting that genetic
effects on smoking persistence are different from those for smoking initiation derives mainly from studies of adult smoking and is, at best, inconclusive. In addition, because the measure of tobacco involvement included the number of days smoked rather than the quantity smoked, it may be considered an indicator of smoking regularity or initiation rather than smoking addiction or persistence. This interpretation is supported by Mayhew et al.’s (2000) discussion about the stages in the development of smoking. According to these authors, the last stage of the smoking initiation process is ‘established’ or ‘daily smoking’, with ‘regular’ smoking (the penultimate stage) involving smoking on a non-daily basis (e.g. on weekends or on schooldays). Our measure of smoking involvement could further be criticized for classifying regular smokers at wave 2, who were not regular smokers at wave 3, as experimenters at wave 3. It could be argued that because these individuals were regular smokers at wave 2 they have shown a predisposition to smoke and should henceforth be labelled regular smokers. We disagree with this argument. A stage model approach to smoking uptake suggests that individuals can exit the path to smoking at any stage of the process. Following Mayhew et al.’s (2000) model of smoking stages, we believe that individuals are not destined to become adult smokers because they smoke at least weekly for some period of their adolescence. Our data support this proposition. Twenty per cent of our wave 2 regular smokers did not smoke regularly at wave 3 and of these, 60% smoked on 4 or less days per week at wave 2. Accordingly, we classified these individuals as experimenters at wave 3 to indicate that they had smoked in the past but that they were no longer regular smokers. We believe this is a more accurate depiction of their smoking behaviour at wave 3 and allows a more accurate examination of the genetic contribution to variation in smoking initiation.

Behaviour genetics studies have been criticized for failing to include direct measures of the environment in their modelling (Rose 1995; Maccoby 2000). This study attempted to address this issue by including peer and parental smoking in genetic and environmental models. By adjusting mean smoking involvement for these variables and modelling the residual variance, this study determined that peers, and not parents, have the major role in influencing the smoking behaviours of adolescents. However, as the inclusion of both parents and peer smoking improved the model significantly, the results suggest that the smoking behaviours of peers and parents contribute independently to adolescents’ smoking behaviours. The finding that genetic influences on variation in smoking behaviour during adolescence reduced when the behaviours of peers were included in the model suggests the importance of including other known influences in behaviour genetic models. The implications of this finding are discussed in detail below.

Our analyses provide an interesting insight into how the smoking behaviours of parents influence their child’s smoking. First, adjusting for parental smoking reduced only the common environment variance component, not the genetic, contrary to the usual behaviour genetics interpretation of the influence of parental smoking. Thus, among this sample of adolescents and young adults, the association between the smoking behaviours of parents and their children was due to environmental factors (i.e. modelling, access, etc.) rather than genetic factors. It appears that adolescents with parents who smoke may be at an increased risk of smoking because of their social environment. This finding supports social modelling theories of smoking initiation. It is, however, possible that this result was due to our use of respondents’ assessments of parents’ smoking status rather than parents’ reports of their smoking histories. As adolescents reported on what they saw their parents do or what they knew of their parents past smoking, the finding may reflect twin pairs reporting on an event in their common environment. It is acknowledged that many children may not know their parents’ smoking status if their parents had quit smoking before they were born or when the child was very young. If parents’ reports of their actual smoking behaviours had been used, the variance component associated with genes, rather than the common environment, may have reduced. This possibility is supported by the analysis of Boomsma et al.’s (1994) study of Dutch adolescent twins that included parents’ self-reports of their smoking behaviours. Biometric modelling of this data suggested that any association between parents’ and children’s smoking was due to genetic factors (Boomsma et al. 1994). While differences in the analytical strategy and outcome variables between the two studies may contribute to some of the differences in the findings, the conflicting results suggest that further work in this area is needed.

Biometric analyses showed that adjusting mean smoking involvement for peer smoking reduced the genetic variance component. This effect was greatest during adolescence and weakened by young adulthood. This finding may seem counter-intuitive, as twins do not share genetic material with their friends. One explanation for this finding draws on Plomin’s (1994) interpretation of the ‘extended genotype’ concept (Dawkins, 1983, cited in Plomin 1994), which suggests that there is a link between an individual’s genotype and the environment they create and select for themselves. Because MZ pairs look and act alike and have similar interests, they may be attracted to the same types of people, and the same types of people may be attracted to each of them similarly. Genetic factors may influence the peers with whom an
individual wants to associate, as well as influencing the peers who want to associate with that individual. Evidence for this with regard to smoking among friendship groups was found in this study. Thus genes might influence smoking during adolescence indirectly by influencing the characteristics that determine peer groups. The veracity of this explanation was tested by treating smoking involvement and peer smoking as a comorbidity and determining the contribution of genes and the environment to the covariation between these two variables. The findings for wave 1 support the explanation given above.

The CE model provided the best explanation of the covariation between smoking involvement and peer smoking at waves 2 and 3. This pattern of results suggest that the genes that influence smoking among older adolescents and young adults are not those that influence peer choice—or at least the choice of peers that smoke. This finding supports the suggestion that genes have a direct influence on the smoking behaviours of young adults and might be associated with nicotine dependence.

Of course, an alternative explanation could be advanced for our wave 1 findings. The classic twin model assumes that the effects of the common environment are independent of zygosity and therefore attributes any greater similarity of MZ pairs to DZ pairs to genes. If, however, identical pairs have more friends in common than non-identical pairs, and these friends influence the smoking behaviours of MZ pairs more similarly than they influence the smoking behaviour of DZ pairs, the assumption of equal effects of a common twin environment might be invalid with respect to smoking. This interpretation is supported by finding that the effect of adjusting smoking involvement for peer smoking on genetic variance reduced as twins grew older and were spending less time together. These results provide some support for the suggestion by Stallings et al. (1999) that a special MZ environment might contribute to the greater similarities in the smoking behaviours of MZ twin pairs. Future studies examining genetic contributions to variation in adolescent substance use need to examine the assumption of equal effects of a common twin environment in more detail.

As indicated above, several limitations restrict the findings of this study. One is that our measures of peer and parental smoking were based on the reports of twins and not peers and parents. As twins were reporting on something in their common environment, this measurement strategy might favour the CE model over the ACE or AE model. Also, the number of MZ and same-sex DZ pairs participating in all three surveys was relatively small, leaving limited statistical power for analyses of males and females separately. Another was possible selection bias that may have resulted from the lower response rates at waves 2 and 3. Smokers were less likely to participate in the study at later waves than were non-smokers. In addition, the prevalence of smoking among female pairs concordant and discordant for study participation differed significantly at wave 3. Girls from pairs discordant for study participation (i.e. where only one member of the twin pair participated in all three surveys) were more likely to smoke in the week before the survey than girls from pairs concordant for study participation (i.e. where both twins participated in all waves). Only subjects with complete data for all waves were used in data analyses. As other studies (Jessar, Donovan & Costa 1991; Chassin et al. 2000) have found, this study found that participants who drop out are more likely to smoke. These results indicate that smokers were under-represented in this sample of twins—especially among female twins —and suggest that the current findings may not generalize fully to the broader population of adolescents or twins.

Smoking initiation is a multi-determined process. This research has suggested that, for a cohort of Australian adolescent twins growing up in the late 1980s and early 1990s, environmental rather than genetic factors were the main influence on determining variation in adolescent smoking. This conclusion is similar to those of Han et al. (1999) and Boomsma et al. (1994). If genes do influence smoking behaviours directly this study suggests that this influence is strongest in the later phases of the smoking uptake process and might result from genes influencing variation in physiological responses to nicotine and/or nicotine dependence. Swan (1999) has suggested that more refined measures of smoking behaviours and nicotine-related phenomena need to be included in behaviour genetic studies if questions regarding how genes influence smoking behaviours are to be answered. We concur with this, but also suggest that our understanding of the role of genes in determining variation in smoking may be increased by including social–psychological models of smoking behaviours within behaviour genetics designs to determine how and when genes influence behaviours.

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