RESEARCH REPORT

Genetic and social influences on starting to smoke: a study of Dutch adolescent twins and their parents

DORRET I. BOOMSMA, JUDITH R. KOOPMANS, LORENZ J. P. VAN DOORNEN & JACOB F. ORLEBEKE

Department of Psychonomics, Vrije Universiteit, Amsterdam, The Netherlands

Abstract

In a study of 1600 Dutch adolescent twin pairs we found that 59% of the inter-individual variation in smoking behaviour could be attributed to shared environmental influences and 31% to genetic factors. The magnitude of the genetic and environmental effects did not differ between boys and girls. However, environmental effects shared by male twins and environmental effects shared by female twins were imperfectly correlated in twins from opposite-sex pairs, indicating that different environmental factors influence smoking in adolescent boys and girls. In the parents of these twins, the correlation between husband and wife for 'currently smoking' (r = 0.43) was larger than for 'ever smoked' (r = 0.18). There was no evidence that smoking of parents (at present or in the past) encouraged smoking in their offspring. Resemblance between parents and offspring was significant but rather low and could be accounted for completely by their genetic relatedness. Moreover, the association between 'currently smoking' in the parents and smoking behaviour in their children was not larger than the association between 'ever smoking' in parents and smoking in their children.

Introduction

Recently Peto *et al.* (1992) reported that "Prolonged cigarette smoking causes even more deaths from other diseases than from lung cancer" and estimated that annual deaths from smoking in the developed countries will continue to increase. For preventive purposes it is important to gain insight into the factors determining smoking initiation during adolescence. Studies with genetically informative subjects such as twins and their parents provide information on genetic influences on smoking behaviour, as well

as on the influence of environmental factors shared by siblings or by parents and children. Smoking shows familial aggregation, but not much is known about the factors that determine the early interest in smoking behaviour. Gurling, Grant & Dangl (1985) and Hughes (1986) review adoption, twin and family studies and conclude that the influence of heredity on smoking behaviour is only small to moderate. More recently, studies employing very large samples of twins have suggested substantial genetic influences on several aspects of smoking behaviour. Carmelli *et al.* (1990) report a heritability (h^2) of 53% for quantity smoked in elderly male American twins. In a large twin

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Correspondence to: Dorret I. Boomsma, Department of Psychonomics, Vrije Universiteit, De Boelelaan 1111, 1081 HV Amsterdam, The Netherlands.

sample of World War II veterans Carmelli *et al.* (1992) show moderate genetic influences on several aspects of smoking behaviour such as never smoked, currently smoking and quitting. Heath *et al.* (1993) find in 2 cross-sectional samples of American and Australian twins that in American twins genetic factors are important in smoking initiation in both males ($h^2 = 60\%$) and females ($h^2 = 51\%$). In Australian twins the genetic contribution to risk of becoming a smoker was larger in females (67%) than in males (33%). Shared environmental effects explained between 15% and 39% of the variance. No birth cohort differences were seen, despite marked changes over time in the proportions of males who ever smoked. Smoking persistence in Australian twins also showed a genetic component which was independent of the genetic effects on smoking initiation (Heath & Martin, 1993). All these studies have focused on adult and elderly twins. In our study smoking in twins was assessed during adolescence, which is the period of life that most smokers take up the habit. In addition, we asked the parents of these twins whether they had ever smoked and whether they were currently smoking in order to explore if the contributions of shared environment to smoking behaviour in children can be explained by cultural transmission from parents to offspring.

Subjects and methods

Twin families were recruited by asking all city councils in The Netherlands (699) for addresses of twins aged 13–22 years. A positive response was received from 252 city councils that supplied 3859 addresses; 177 addresses were available from other sources. After contacting these families by letter, 2375 twin families indicated that they were willing to complete a questionnaire on health and lifestyle and 1610 families returned these questionnaires. Data from 17 families were not used because the twins were either too young or too old, or because one or both twins had not completed the questionnaire. Subjects answered the questions "Did you ever smoke?" and "Do you smoke now?" with "yes" or "no". In the twins only the response to the first question was analyzed, because few children—especially the younger twins—had ever smoked and quit. There were 1582 twin pairs with complete data for smoking, and 1324 families with complete data for both parents and children. Average age

of fathers and mothers was 48 (SD = 5.7) and 46 (SD = 5.2) years. Twin zygosity was determined by questionnaire (Nichols & Bilbro, 1966). In a group of 131 same-sex adolescent twin pairs who participated in a study of cardiovascular risk factors (Boomsma *et al.*, 1993) agreement between zygosity based on this questionnaire and zygosity based on blood group polymorphisms and DNA fingerprinting was 95%.

Statistical analysis

Tetrachoric correlations among family members for smoking status were computed by maximum likelihood with PRELIS (Jöreskog & Sörbom, 1986). In computing tetrachoric correlations it is assumed that the distribution underlying the dichotomous trait is continuous and normal, with a threshold that distinguishes smokers from non-smokers (Falconer, 1989). Correlations were estimated separately for monozygotic (MZ) and dizygotic (DZ) male and female, and DZ opposite-sex twin pairs; for fathers and mothers with their male and female offspring and for spouses (husband-wife pairs). These correlations were used to test different models of familial resemblance (Neale & Cardon, 1992). We first fitted different models of family resemblance to the twin data and next included the parents of twins in a quantitative genetic analysis.

Path analysis was used to quantify the contributions of genetic and environmental factors to variation in smoking behaviour. The methodology was based upon the standard biometrical model (Eaves *et al.*, 1978). Figure 1 shows a path model for twins, in which variation in smoking behaviour is influenced by additive genetic factors, common environmental influences shared by siblings growing up in the same family and individual specific environmental influences.

Under a model where familial resemblance is explained by additive genetic factors MZ twins, who are 100% genetically identical, are expected to be twice as similar as DZ twins, who share 50% of their genetic material. Under a shared environmental model MZ and DZ same-sex correlations are predicted to be the same. Two different models were examined to assess sex differences in genetic architecture: a model in which estimates for h, c, and e are allowed to differ in magnitude between males and females, and a model in which environmental factors that
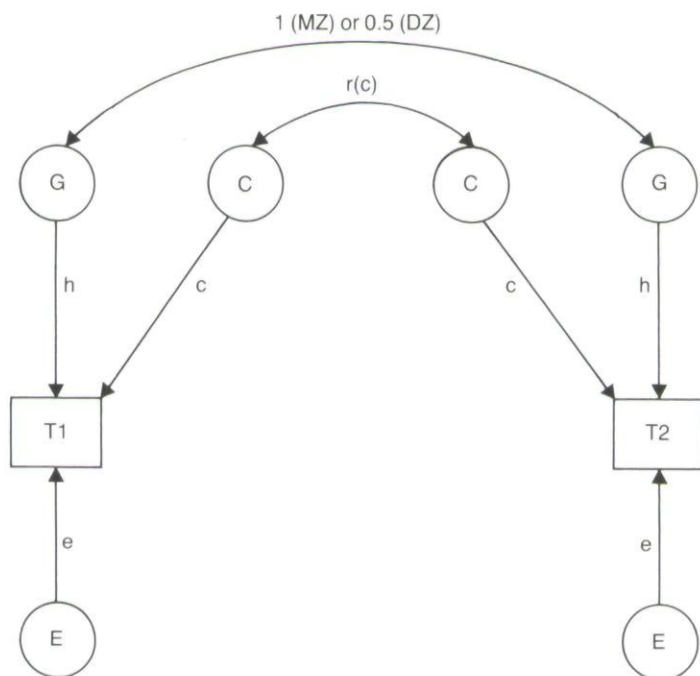
Figure 1. *Path diagram for DZ and MZ twins. Squares represent observed variables in Twin 1 and Twin 2 and circles represent latent variables. G stands for additive genetic factors, C for common environmental influences shared by siblings and E for individual environmental factors that are not shared between family members. The influence of G, C and E on the phenotype is given by path coefficients h, c and e, respectively. These path coefficients are equivalent to standardized partial regression coefficients and the proportion of variance due to genetic and environmental influences is equal to the squared path coefficients. The correlation between G1 and G2 is 1 for MZ and 0.5 for DZ twins. The correlation between C1 and C2 is 1 for same-sex twins and is allowed to vary for DZ opposite-sex twins. The expectation for the MZ correlation equals $h^2 + c^2$ and the DZ correlation $0.5h^2 + c^2$*

influence smoking behaviour in males and females are imperfectly correlated.

In order to examine if one sibling's smoking behaviour directly influences smoking behaviour in his or her twin, MZ and DZ prevalences were compared. A difference in base rates between MZ and DZ twins (MZ prevalence > DZ prevalence) for a heritable dichotomous trait, may constitute evidence for sibling interaction (Carey, 1992).

In analyzing the parent-offspring data, the association between spouses was modeled as phenotypic assortment and cultural transmission from parents to children as transmission from parental phenotype to the shared environment of the children. In this model the shared environment of siblings is partitioned into cultural transmission from parents to children and environmental effects that are shared by offspring

only. The effects of cultural transmission and assortative mating induce a correlation between genes and environment (Fulker, 1982, 1988; Boomsma & Molenaar, 1987). Model fitting was carried out with LISREL 7 (Jöreskog & Sörbom, 1988) and Mx (Neale, 1991) using weighted least squares, which is an asymptotically distribution-free procedure. Goodness-of-fit was assessed by likelihood ratio χ^2 tests. A large χ^2 indicates a poor fit, while a small χ^2 indicates that the data are consistent with the model. Submodels excluding parameters are compared to the full model by hierarchic χ^2 tests.

Results

Table 1 gives the percentage of offspring who ever smoked for the total sample and for different age groups. The first group consists of twins

aged 13 and 14 years, the second group of twins aged 15 and 16, the third group of 17- and 18-year-olds and the last group of all twins aged 19 or older. With increasing age there was an increase in the number of children who reported that they ever smoked. In the youngest cohort only 6.3% of the twins stated that they had ever smoked. This percentage was 14% in the 15- and 16-year-olds, and increased to 26% in 17- and 18-year-olds. After the age of 18, smoking prevalence stabilized around 36%. Within each group, there were no significant age differences in smoking status, while the difference between the 4 groups was highly significant ($\chi^2 = 196$, df $= 3$, $p > 0.00$). There also were no significant sex differences in smoking status within groups, except in the oldest cohort. In this group males indicated more often than females that they had ever smoked ($\chi^2 = 7.1$, df $= 1$, $p = 0.01$). In the parental generation, there were significant sex differences for 'ever smoked' (84% of fathers and 66% mothers had ever smoked, $\chi^2 = 106.7$, df $= 1$, $p = 0.000$) and for 'currently smoking' (fathers 38%, mothers 29%, $\chi^2 = 20.8$, df $= 1$, $p = 0.000$).

Genetic analyses were first carried out separately for the different age groups. For these groups as well as for the complete sample Table 1 lists for each zygosity the twin concordances and tetrachoric correlations with their standard errors for smoking behaviour. For the total sample tests of differences in base rates between MZ and DZ twins neither yielded significant results for males ($\chi^2 = 3.37$, df $= 2$, $p = 0.19$) nor for females ($\chi^2 = 1.68$, df $= 2$, $p = 0.43$). In the different age groups, there also was no evidence that the base rates for smoking were higher for MZ than for DZ twins (results not shown).

The pattern of twin correlations suggests a different mode of inheritance across age. For males heritability seems to decrease with increasing age, because the differences between MZ and DZ correlations get smaller as the twins get older. For females heritability seems to increase with increasing age, because the differences between MZ and DZ correlations slightly increase. The almost equal MZ and DZ correlations for females indicate a substantial contribution of shared environmental factors. The pattern of correlations also suggests that—relative to DZ same-sex correlations—DZ opposite-sex correlations decrease with increasing age. However, the standard errors around some of these correlations are rather large.

Table 2 shows the χ^2 goodness-of-fit statistics for different models of twin resemblance in each age group. These indicate that in the group aged 15–16 years resemblance for smoking status in males was best explained by genetic factors and in females by shared environment. In the 17–18-year-olds, both genetic and shared environmental influences contributed to variation in males, while in females shared environment remained the only significant source of variation. In the oldest group (19–22 years) genetic and shared environmental factors were of equal importance in males and females. The best fitting model for this group included both these sources of variation and let the correlation between the shared environments of boys and girls take its own value. Under this model, the estimate of the correlation between boys and girls for shared environmental influences was 0.27, which did not differ significantly from zero. At the bottom of Table 2 are the estimates for the contributions of genetic and shared environmental factors from the best fitting model. Heritability estimates for males were high in the youngest group (94%) and then decreased to 41 and 55% in the older groups. For females, genetic factors only came into play at age 19–22. At earlier ages a large contribution of shared environmental factors (84%) was seen.

Next, the differences in parameter estimates across age groups and sexes were formally tested for significance in a 15-group analysis (5 zygosities $\times$ 3 age groups). Specifying for each age group the best fitting model from Table 2 gave a χ^2 of 7.13 (df $= 8$, $p = 0.52$). However, constraining all parameters to be equal across age groups and sexes did not lead to a significant decrease in fit ($\chi^2 = 11.54$, df $= 12$, $p = 0.48$). This constrained model gave a heritability of 39% for smoking behaviour and a contribution of shared environmental influences of 53%. The correlation between environmental effects shared by male and female twins was 0.53 (SE $= 0.14$). These estimates from the 15-group analysis were almost identical to the estimates that obtained when the same model was fitted to the tetrachoric correlations computed for the whole twin sample aged 15 and older ($h^2 = 34\%$, $c^2 = 56\%$ and Rc $= 0.63$), or to the whole sample including 13- and 14-year-olds ($h^2 = 31\%$, $c^2 = 59\%$ and Rc $= 0.65$). In these last two models, part of the

Table 1. *Smoking prevalence and twin concordance for total sample and for different age groups*

	N pairs	Smoking (%)	Concordance smoking (%) Both	One	Neither	Tetrachoric correlation and SE	
All twins							
MZM	250	23	17.1	12.6	70.2	0.87	(0.042)
MZF	329	22	17.6	9.5	72.9	0.92	(0.026)
DZM	241	26	16.1	19.5	64.4	0.73	(0.067)
DZF	304	23	14.0	16.2	69.8	0.77	(0.056)
DOS	458	29(M)/26(F)	14.1	26.0	59.9	0.54	(0.064)
Total	1582	26(M)/23(F)					
13–14-year-olds[*]							
MZM	39	6	5.1	2.6	92.3		
MZF	40	4	2.5	2.5	95.0		
DZM	30	8	6.7	3.3	90.0		
DZF	40	8	5.0	5.0	90.0		
DOS	40	10(M)/3(F)	2.5	7.5	90.0		
Total	189	8(M)/5(F)					
15–16-year-olds							
MZM	58	14	10.3	6.9	82.8	0.94	(0.065)
MZF	72	11	6.9	8.4	84.7	0.86	(0.108)
DZM	55	12	3.6	16.4	80.0	0.44	(0.276)
DZF	73	20	13.7	13.6	72.6	0.82	(0.099)
DOS	120	13(M)/12(F)	3.3	18.3	78.3	0.34	(0.199)
Total	378	13(M)/15(F)					
17–18-year-olds							
MZM	65	28	20.0	15.4	64.6	0.84	(0.089)
MZF	100	26	19.0	14.0	67.0	0.86	(0.066)
DZM	66	23	12.1	22.7	65.2	0.59	(0.167)
DZF	67	23	14.9	16.4	68.7	0.79	(0.113)
DOS	141	28(M)/27(F)	15.6	24.1	60.3	0.61	(0.105)
Total	439	27(M)/26(F)					
19–22-year-olds							
MZM	88	34	25.0	18.2	56.8	0.81	(0.081)
MZF	117	32	28.2	8.5	63.2	0.96	(0.026)
DZM	90	41	28.6	26.4	45.1	0.70	(0.105)
DZF	124	27	17.6	20.8	61.6	0.72	(0.095)
DOS	157	45(M)/41(F)	24.2	37.6	38.2	0.37	(0.115)
Total	576	40(M)/33(F)					

MZM, Monozygotic Males; DZM, Dizygotic Males; MZF, Monozygotic Females; DZF, Dizygotic Females; DOS, Dizygotic Opposite–Sex Twins

*Because of the small number of subjects and the low rate of 'ever smoking' tetrachoric correlations were not computed for these twin pairs.

shared environmental variance can be explained by age differences between twin pairs. Including age as a covariate in the analysis of the whole sample, showed that 9% of the total variance in smoking behaviour could be explained by age, 50% by shared environment, 31% by genetic factors and the remaining 10% by random environmental factors.

Because parameter estimates for genetic and environmental variances were virtually identical when using data from all twins compared to using the data from twins aged 15 years and older, all data were used in a 5-group parent-offspring analysis. Table 3 lists the number of families in each of the 5 groups, the twin correlations for 'ever smoked', the correlations between spouses for 'currently smoking' and 'ever smoking' and the correlations between these variables in fathers and mothers with smoking in their sons and daughters. The association between

Table 2. *Model fitting to twin data*

Model			Chi-squared statistic and probability					
			15–16 year		17–18 year		19–22 year	
Males/Females		df	χ^2	p	χ^2	p	χ^2	p
GCE GCE,	Rc(M,F) = 1	1	0.04	0.83	0.00	0.96	0.00	0.98
GCE CE,	Rc(M,F) = 1	2	0.18	0.91	0.43	0.81	9.19	0.01
GE CE,	Rc(M,F) = 1	3	2.97	0.40	34.59	0.00	22.70	0.00
GCE = GCE,	Rc(M,F) = 1	3					9.28	0.03
GCE = GCE,	Rc(M,F) = 0	3					3.74	0.29

	Variance estimates (%) based on best fitting model						
	15–16 year		17–18 year		19–22 year		
	Males	Females	Males	Females	Males	Females	
h^2	94	—	41	—	55	55	
c^2	—	84	42	84	40	40	

E represents individual specific environment (including measurement error), G genetic factors, C common or shared environmental influences and Rc(M,F) the correlation between shared environmental influences of males and females. h^2 and c^2 give the percentages of the total phenotypic variance that can be attributed to genetic and shared environmental factors.

Because correlations instead of covariances were analyzed degrees of freedom (df) were adjusted. There are 5 statistics per age group, and as e is not a free parameter the df equal: 5–(number of parameters to be estimated) + p, where p = 1 if there are no sex differences in parameter estimates and where p = 2 if there are sex differences in estimates.

spouses for 'ever smoked' was significant, but rather low (0.18), while it was quite high (0.43) for 'currently smoking'. Correlations between parents and offspring were also low (between 0.05 and 0.24) and did not depend on either the sex of the parent or the offspring. Correlations between 'currently smoking' in the parents and smoking in children were not systematically higher than correlations between 'ever smoking' in parents and smoking in children. Genetic model fitting to smoking data of twins and parents gave estimates for cultural transmission parameters from parents to offspring that did not differ significantly from zero. Resemblance between parents and offspring could be accounted for completely by their genetic relatedness, both when considering smoking behaviour in children with 'currently smoking' in their parents and smoking in children with 'ever smoked' in their parents. However, the first model showed a better fit to the data than the second. This is probably due to the low correlation between mothers and sons for 'ever smoking'. From both analyses we obtained heritability estimates that showed a close resemblance to each other and to the estimate from the analysis of the twin data. From the analysis with 'currently smoking' in the parents the estimates for h^2 and c^2 were 30% and 62%, and from the analysis with 'ever smoking' in parents these estimates were 32% and 61%.

Discussion

We observed high resemblances between adolescent twin siblings aged 13–22 for smoking behaviour. The data seemed to imply that these resemblances differed as a function of age. Model fitting to twin correlations from different age groups suggested that genetic factors in boys became less and in girls became more important with increasing age. However, a formal test of this hypothesis showed that homogeneity in genetic and non-genetic parameters across age groups could not be rejected. A model in which genetic influences accounted for 31% and shared environment for 59% (including 9% that can be attributed to age differences between twin pairs) of the variation in smoking behaviour gave the most parsimonious account of the data. Two important conclusions may be drawn from this study. Firstly, there appears to be no direct influence from parental smoking behaviour (at present or in the past) on the smoking behaviour of their children. Secondly, the environmental factors that influence smoking in boys differ from

Table 3. *Twin correlations for ever smoked and spouse and parent-offspring correlations for ever smoked in children with ever smoked and smoking now in parents for 1324 families with complete data; model fitting results*

	Pairs	Correlation Ever smoked			Pairs	Correlation Smoke now		Correlation Ever smoked	
MZM	211	0.87	(0.047)	Spouses	1324	0.43	(0.040)	0.18	(0.052)
MZF	277	0.94	(0.023)	Father-son	1222	0.19	(0.048)	0.24	(0.060)
DZM	208	0.67	(0.079)	Mother-son	1222	0.14	(0.050)	0.05	(0.051)
DZF	244	0.75	(0.066)	Father-daughter	1426	0.17	(0.046)	0.21	(0.055)
DOS	384	0.52	(0.073)	Mother-daughter	1426	0.23	(0.046)	0.20	(0.047)

	Parameter estimates				
Ever smoked in twins with		Parents smoke now		Parents ever smoked	
h		0.70	0.54	0.64	0.57
c		0.74	0.79	0.75	0.78
e		0.26	0.28	0.26	0.27
Rc(M,F)		0.56	0.55	0.66	0.64
Spouse correlation		0.45	0.45	0.21	0.20
Cultural transmission		-0.12	—	-0.06	—
G × C correlation		-0.11	—	-0.04	—
χ^2		23.50	25.87	45.02	45.67
df		25	26	25	26
p		0.55	0.53	0.01	0.02

h represents the influence of the genotype on the phenotype, c the influence of common or shared environment and e the influence of individual specific environment. The spouse correlation is the correlation between husband and wife. Cultural transmission parameters represent the influence of parental phenotype on the shared environment of children. This transmission induces a correlation (G × C) between genotype and environment.

the environmental factors that influence smoking in girls.

Resemblances in smoking behaviour among parents and offspring were weak. Biometrical analyses of the parent-twin data showed that parental smoking behaviour does not directly influence smoking behaviour of their children. Although adolescents who smoke more often had parents who also smoked, this association was completely accounted for by their genetic relatedness. It is remarkable that the correlation between smoking behaviour in children and 'currently smoking' in their parents is not higher than the correlation between smoking in children and 'ever smoking' in their parents and that for 'currently smoking' parent-child correlations were lower than the spousal correlations. Pérusse, Leblanc & Bouchard (1988) observed the same patterns of higher family resemblance within generations than across generations for smoking behaviour and suggest that familial resemblance results from environmental influences common to members of the same generation.

The most important sources for sibling resemblance in smoking behaviour were environmental factors shared between siblings but not between

parents and offspring. This finding is in agreement with other studies of adolescent smoking behaviour (e.g. McNeill *et al.*, 1988; Swan, Creeser & Murray, 1990) in which parental smoking was only a weak predictor of taking up of smoking in their children. There were no differences between boys and girls in the amount of variance explained by genetic or shared environmental influences. However, the resemblance for smoking status in opposite-sex twins was lower than in same-sex DZ twins. This lower correlation was explained by the fact that environmental effects shared by male twins and environmental effects shared by female twins were imperfectly correlated in twins from opposite-sex pairs. This sex difference is in agreement with results from other studies. Swan *et al.* (1990) found, for example, that sports participation decreased the risk of taking up smoking in girls, but not in boys, whereas organized social activities increased risk in girls but not in boys.

This is the first study of smoking behaviour in adolescent twins and their parents. The influence of shared environment is larger and the influence of genetic factors smaller than commonly has been found in studies of adult twins.

This modification of the inheritance of smoking behaviour may have important implications for smoking prevention. Successful prevention needs at least partly be targeted at boys and girls separately and has to focus on those shared environmental influences that are largely independent of parents.

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