RESEARCH PAPER

Challenges to the peer influence paradigm: results for 12–13 year olds from six European countries from the European Smoking Prevention Framework Approach study

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Objective: To examine whether smoking onset in young adolescents is predicted by peer or parental smoking.
Design: Longitudinal design with one pretest and one follow-up at 12 months.
Setting: Schools in Finland, Denmark, the Netherlands, the United Kingdom, Spain and Portugal.
Participants: 7102 randomly selected adolescents from six countries. Mean age was 12.78 years.
Main outcome measures: Smoking behaviour of adolescents, peers and parents.
Results: No support was found for peer smoking as an important predictor of smoking onset in most countries. Support was found for the selection paradigm, implying that adolescents choose friends with similar smoking behaviour. Support for the impact of parents on adolescent behaviour and the choice of friends was also found.
Conclusions: Smoking uptake in this age cohort may be more strongly influenced by personal and parental influences than initially believed. Hence, social inoculation programmes teaching youngsters to resist the pressures to smoke may be less appropriate if youngsters have a positive attitude towards smoking, associate smoking with various advantages and look for peers with similar values. For this group attitudes towards smoking may thus guide future friend selection.

Smoking prevention programmes can have short-term effects, but effects that remain for a period of five years or more are relatively rare.[1–4] Although one reason can be that most interventions are limited in duration, the lack of effect of a 15 year school-based study[4] suggests that extending the intervention period may not be effective by itself. Studies that do find longer term effects often integrate the intervention within a broader community-based approach also addressing other populations such as adults.[5 6] The results of the European Smoking Prevention Framework Approach (ESFA) study also suggest that an integral prevention strategy may be effective in some countries (Finland, Spain and Portugal) where the programme addressed successfully several target groups.[7–9] Another explanation concerns the paradigm that is used by most approaches, the peer behaviour or social pressure paradigm stating that smoking onset is caused by peer smoking and incapability of youngsters to resist peer pressures.[2 10]

Recent studies cast doubt on the validity of the social pressure paradigm as the sole explanation of smoking onset, and suggest alternative mechanisms. An alternative to peer pressure as an explanation for similarities between adolescent and peer smoking is the selection of similar smoking friends by a (smoking) adolescent. Moreover, many studies suggesting peer influences did not use longitudinal designs, hence limiting the possibility to detect causal patterns. Investigating influence and selection processes requires the utilisation of longitudinal designs.[11 12] Several studies included elements of the selection paradigm with regard to adolescent health behaviour.[12–24] Engels and colleagues found more evidence for the influence of parents than for that of (best) friends.[23] They did find an important effect of the smoking status of friends from cross-sectional data, but a much lower impact in adolescent smoking behaviour five years later. Some studies also found that the selection of friends was mediated and influenced by parents.[23 25 26] Flay and colleagues postulated both influences of parents and peers two decades ago. The impact of parents was assumed to be more important for young children, while the impact of peers was hypothesised as more important for adolescents.[27]

Finally, influence and selection need not be mutual exclusive patterns but can be complementary processes[28] implying that peer influences processes should be conceptualised broader, also incorporating sequential and reciprocal relationships.[16 29 30] As a consequence, Urberg and colleagues[29] propose a two-stage model of peer influence with the acquisition of a peer context to be the first stage and conforming to the behaviour of peers as the second. They also found not valuing school achievements and lower ratings of spending time with parents were predictors of choosing friends who used cigarettes at a higher level.

The goal and contribution of this paper to the existing literature is to use longitudinal data of three groups (adolescents, peers, and parents) to analyse influence and selection patterns in six different European samples using structural equation modelling (SEM) techniques. Several possible paths of influence and selection were included in the analysis (fig 1). Studies suggest that different predictive models can be hypothesised, but there is consensus about the fact that future behaviour is influenced by previous behaviour; the paths 1, 2 and 3 of fig 1 reflect this. Significant peer influences, indicating that friends' smoking behaviour at T1 predicts adolescent smoking behaviour at T2, will be reflected by path 4. The selection model, however, predicts that adolescents choose friends with similar smoking behaviour; this is reflected by path 5. Potential parental influences are reflected by a strong relationship between parental behaviour at T1 and smoking behaviour of their children at T2; this is suggested by path 6. Another potential relationship is that

Abbreviations: CFI, comparative fit index; ESFA, European Smoking Prevention Framework Approach; RMSEA, root mean square of approximation; SEM, structural equation modelling; TLI, Tucker-Lewis index

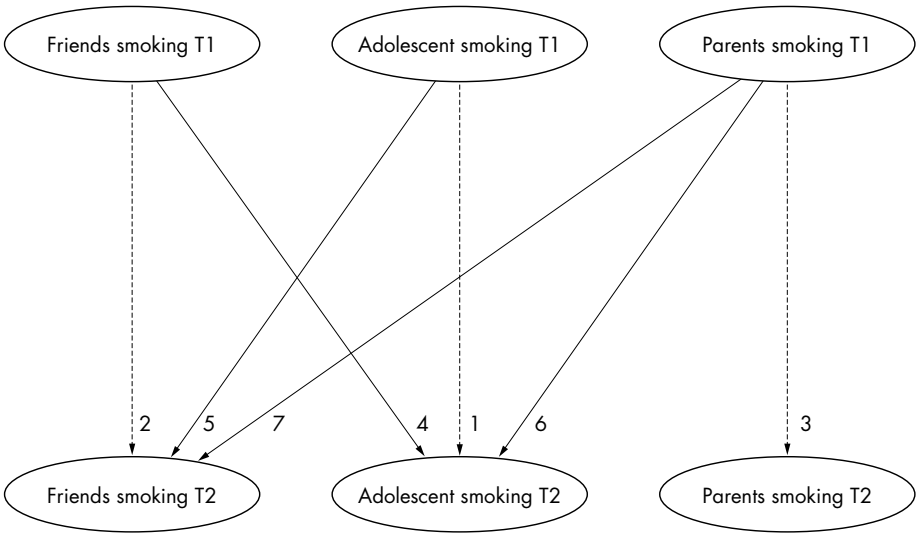

Figure 1 Overview of influence and selection paths tested.

parental smoking behaviour may have an impact on the choice of (smoking) friends,[23] which is reflected by path 7.

METHODS

The sample consisted of adolescents from six European countries (Finland, Denmark, the Netherlands, the United Kingdom, Spain and Portugal) that participated as a control group in ESFA.[31 32] Since interventions were planned to take place at the community level, communities (or regions) were chosen as the unit of assignment in each country. Experimental regions would execute the ESFA programme, while control regions would provide usual care. Schools in these regions were asked to participate in the project, indicating that they would have a 50% chance of becoming an experimental school. Power calculations were run in order to estimate the number of pupils to be included in each national sample, thus resulting in different sample sizes per country.[7]

The number of schools and the number of classes differed from country to country due to school size differences in the various countries. Consequently 14 control schools with 80 classes participated in Finland, 30 schools with 54 classes in Denmark, 17 schools with 87 classes in the Netherlands, 21 schools with 166 classes in the United Kingdom, 31 schools with 37 classes in Spain, and 11 schools with 76 classes in Portugal).[9] Only adolescents of the second class of secondary schools were included in the study.

The total sample of this study consisted of 7102 adolescents from six European countries with 50% boys and 50% girls. Overall mean age was 12.78 years. In Finland 1243 students participated (mean age 13.26 years); in Denmark 572 students participated (mean age 13.28 years); in the Netherlands 1987 students participated (mean age 12.96 years); in the United Kingdom 1746 students participated (mean age 12.78 years); in Spain 647 students participated (mean age 12.36 years); and in Portugal 907 students participated (mean age 12.7 years).

Procedure

Questionnaires were distributed to schools during the autumns of 1998 (T1) and 1999 (T2). Students were asked to participate, and it was explained to them that responses would be treated confidentially and that they could refuse to participate. The questionnaire[32] measured adolescents' weekly smoking behaviour, parents' smoking behaviour, friends' smoking behaviour, and demographics.

Adolescent smoking behaviour was assessed by three questions. The first question asked the student to indicate which option best described them: ''I smoke at least once a day; I do not smoke daily, but at least once a week; I do not smoke weekly, but at least once a month; I smoke less than once a month; I have tried smoking up to five times; I have tried smoking, but it was only a few puffs; I have quit smoking; I never smoked a cigarette''. The responses were recoded in the following way: smoking status (0 = non smoker, 1 = less than monthly smoker, 2 = monthly smoker, 3 = weekly smoker, 4 = daily smoker). Second, smoking frequency during the past week was assessed (0 = 0; 1 = 1; 2 = 2–10; 3 = 11–30; 4 = >30). Third, the number of cigarettes smoked during lifetime was measured: less then 100 (0); more than 100 (1).[32 33]

Parental smoking behaviour was assessed by means of two questions measuring the smoking behaviour of the father and the mother respectively, using a three-point scale for each item (0 = no, 1 = don't know, 2 = yes).[34]

Friends' smoking behaviour was assessed using two questions: ''Does your best friend smoke?'' with answer categories ranging from no (0), maybe (1), to yes (2), and: ''How many of your friends smoke?'' with answer categories ranging from nobody (0), less than half of my friends (1), half of my friends (2), more than half of my friends (3), to all of my friends (4).[34]

Statistical analysis

The analysis included smoking behaviour of adolescents, friends and parents at T1 and T2, and the covariates of adolescents' smoking behaviour.

First, we analysed the associations between the actors' smoking behaviour at T1 and T2. The adolescent was classified as a smoker if at least smoking monthly. Parents were classified as non-smokers if both parents did not smoke, or else as smokers. Friends were classified as smokers if at least half of them smoked. For each actor at T1 we can compare the proportion of smoking actors at T2 between smoking and non-smoking actors at T1. This risk difference is an indication of the influence of the smoking behaviour of the actor at T1 on the actor at T2.

Second, we used structural equation modelling to be able to draw more solid conclusions on influences in the social network of the adolescent, including all variables that measured smoking behaviour of the actors in the network, not restricting us to the dichotomy of smoking and non-smoking behaviour, and to be able to correct for potential confounders of adolescent's smoking behaviour. Next to the

smoking behaviour of adolescents, friends, and parents at T1 and T2 the structural equation analysis involved covariates of adolescents' smoking behaviour, thus controlling for sex, age, religion (yes, no), race (non-native born, native born), job father (father has a paid job, father does not have a paid job), job mother (mother has a paid job, mother does not have a paid job), medium performance at school (adolescent belongs to the middle third of his/her class, adolescent does not belong to the middle third), best performance at school (adolescent belongs to the highest third of his/her class, adolescent does not belong to the highest third), and alcohol consumption (average number of glasses of alcohol consumed per week, including the weekend). The covariates were allowed to influence smoking behaviour of all actors in the network both at T1 and T2.

To determine which variables predicted smoking behaviour one year later, structural equation modelling was done using the LISREL8.7 programme.[35] Since the variables turned out to be not normally distributed, robust maximum likelihood analyses (method 1) were done.[36] Since the sample sizes of several countries were below 1000, this method was considered to be preferable to the asymptotic distribution free estimation.[35] For robust maximum likelihood analysis, no missing values are allowed. Missing values were therefore replaced by estimates obtained through expectation maximisation.[37] To study the robustness of the analysis results under imputation, also a maximum likelihood analysis was done without imputation, making use of all available data (full information maximum likelihood in LISREL8.7; method 2).

First, the measurement models for the latent variables that the smoking variables purported to measure was examined. The model fit as well as the discriminant validities of the scales involved were investigated. The latter was done by comparing a model in which two scales were assumed to measure the same latent variable to a model in which the two scales each measured a different latent variable. In the next step, complete models comprising both the measurement model and the structural model, specifying possible relations between the actors' smoking behaviour, were examined. Relevant background variables that might influence smoking behaviour of the actors in the network on T1 and T2 are also included, such as baseline adolescent smoking behaviour in order to control for similarity in friend smoking behaviour.[16 29]

As measures of overall model fit, for robust maximum likelihood a χ^2 statistic corrected for non-normality, known as the Satorra-Bentler statistic, will be reported.[36] Since sample sizes are considerable, minor deviations from the data may make the models perform badly as measured by the p value of the Satorra-Bentler statistic. Therefore, also the comparative fit index (CFI) and the Tucker-Lewis index (TLI) are given. A good model fit is indicated by CFI and TLI values higher than 0.90.[38] In addition, the root mean square of approximation (RMSEA) is given. RMSEA values lower than 0.05 also indicate a good model fit.[38] The R^2 of the latent variable representing adolescent's smoking behaviour will be presented. This expresses how well adolescent's smoking behaviour can be explained by the determinants included in the model. Finally, for robust maximum likelihood analysis the statistical significance of the structural relations between the latent variables is based on standard errors that also are adjusted for non-normality.[36]

Effect sizes were calculated for the four most important paths of this study: (path 4) T1 friends smoking behaviour on T2 adolescent's smoking behaviour; (path 5) T1 adolescent's smoking behaviour on T2 friends' smoking behaviour; (path 6) T1 parents' smoking behaviour on T2 adolescent's smoking behaviour; and (path 7) T1 parents' smoking behaviour on T2 friends' smoking behaviour. A measure of effect size is obtained by comparing the χ^2 of model fit before and after including one of these paths in the model, and relating this to the χ^2 of model fit of a null model. The latter is a model where there are no relations between the latent variable to be predicted in the path and all predictor variables in the model. This effect size measure thus calculated can be interpreted as a measure of proportional model fit improvement due to including a particular path in the structural equation model.

RESULTS

Smoking behaviour

The percentage of weekly smokers increased from 4.3% at time T1 to 12% at time T2. In Finland the percentage of weekly smokers increased from 8.4% at T1 to 20.4% at T2, from 4.5% to 13.4% in Denmark, from 3.3% to 9.8% in the Netherlands, from 4.4% to 12.3% in the United Kingdom, from 1.6% to 9.2% in Spain, and from 2.5% to 6.0% in Portugal. These figures are to a large extent comparable to those found in an earlier study,[39] although figures differ slightly because of sampling and questionnaire differences.

Missing values

An overview of the percentages of missing values for each of the variables involved is given in table 1. Note that the number of missing values for the predictor variables (variables relating to the smoking behaviour of all actors involved at T1) is not that large, seldom exceeding 15%. For the covariates and the outcome variables (variables relating to the smoking behaviour of all actors involved at T2), the percentages of missing values are larger, sometimes exceeding 25%. The highest numbers of missing values occur for the variables measuring adolescents' smoking behaviour at T2. This underlines the importance of treating missing values in a careful way. As delineated in the method section, two different methods of handling missing data will be used. Similarity of the results obtained with both methods will support the way the missing value issue is resolved.

Risk differences

Table 2 shows the proportion of adolescents who had started to smoke at T2, both for those who had smoking friends and those who had non-smoking friends at T1 and the relative risks. With regard to Finland, table 2 shows that 54% of the adolescents whose friends smoked at T1 had taken up smoking at T2 versus 17% of adolescents whose friends did not smoke at T1, resulting in a relative risk—defined as the absolute difference in the event rate between the two comparison groups—of friend smoking of 0.37 (0.54–0.17). Table 2 furthermore shows proportions of smokers at T2 for both adolescents with smoking parents at T1 and adolescents with non-smoking parents at T1. In addition, table 2 shows the proportions of adolescents with smoking friends at T2 for adolescents who smoked at T1 and for adolescents who did not smoke at T1. In the case of Finland, table 2 shows that if the adolescent smoked at T1 the chance of having smoking

Table 1 Percentages of missing values (minimum, maximum) for each of the three groups of variables (predictor variables, covariates and outcome variables)

	Predictor variables	Covariates	Outcome variables
Finland	0%; 16.6%	0%; 8.1%	0%; 31.7%
Denmark	0%; 14.0%	0%; 12.8%	0%; 29.9%
Netherlands	0%; 11.3%	0%; 16.8%	0%; 19.6%
United Kingdom	0%; 10.3%	0%; 18.7%	0%; 20.3%
Spain	0%; 11.4%	0%; 25.7%	0%; 20.1%
Portugal	0%; 7.6%	0%; 21.5%	0%; 14.6%

Table 2 Proportions of smoking actors at T2

	Friends smoked at T1			Parents smoked at T1				Adolescent smoked at T1		
	Yes	No	RR	Yes	No	RR		Yes	No	RR
Adolescent smoking at T2							Friends' smoking at T2			
Finland	0.54	0.17	0.37	0.31	0.17	0.14	Finland	0.79	0.26	0.53
Denmark	0.44	0.13	0.31	0.19	0.13	0.06	Denmark	0.67	0.23	0.44
Netherlands	0.44	0.10	0.34	0.16	0.09	0.07	Netherlands	0.60	0.14	0.46
United Kingdom	0.42	0.12	0.30	0.21	0.10	0.11	United Kingdom	0.56	0.17	0.39
Spain	0.22	0.09	0.13	0.13	0.07	0.06	Spain	0.63	0.25	0.38
Portugal	0.28	0.05	0.23	0.10	0.05	0.05	Portugal	0.54	0.13	0.41

RR, relative risk (proportion ''yes'' – proportion ''no'').

friends at T2 is 79%, whereas this chance of having smoking friends is 26% if the adolescent did not smoke at T1. These differences in relative risks suggest a stronger effect for selection than for peer influence processes. When looking at all other countries we see larger relative risks for the selection patterns than for the peer influence patterns. Additionally it can be seen that the proportions of T1 non-smoking adolescents that have smoking friends at T2 in all countries, in particular Finland (26%), Denmark (23%) and Spain (25%), are quite high, demonstrating that still many non-smoking adolescents have smoking friends. The relative risks of parental smoking were found to be quite modest.

Predictors of adolescent smoking behaviour

More in-depth analyses were run using structural equation techniques in order to take into account the interrelationships of the actors (that is, adolescent, friends, parents) as well corrected for potential confounding effects of other factors. Although the measurement models have significant Satorra-Bentler statistics ($p < 0.05$), the fit indices that are less dependent on sample size, point to a good model fit for each country (CFI $\geqslant 0.987$, TLI $\geqslant 0.978$, and RMSEA $\leqslant 0.055$). Testing the discriminant validity of the scales employed for measuring smoking behaviour of the different actors in the social network, showed for each country significant decreases ($p < 0.001$) in model fit when different scales were restricted to measure the same latent variable.

The results concerning the models comprising both a measurement and structural model are summarised in table 3. For each country we see that the Satorra-Bentler statistic is significant (p value < 0.001). The other fit indices on the other hand point to very good model fits (CFI $\geqslant 0.987$, TLI $\geqslant 0.976$, RMSEA $\leqslant 0.0417$). Furthermore, adolescent's smoking behaviour could be explained reasonably well by the determinants included in the model ($R^2 \geqslant 0.406$).

The relation of the covariates to smoking behaviour differed strongly from one country to the other. The significant findings are discussed below ($p < 0.05$). In Finland, the Netherlands, the United Kingdom and Spain female adolescents smoked significantly more than male adolescents. Furthermore, in the United Kingdom adolescents' smoking behaviour was positively associated with the adolescents' alcohol consumption. In Denmark and the United Kingdom, being the best performing pupil of the class was negatively associated with adolescents' smoking behaviour. When mothers had a paid job, this was positively associated with adolescents' smoking behaviour in Finland. In Spain, adolescents' smoking behaviour was positively associated with fathers having a paid job. Finally, in the Netherlands religious adolescents were smoking less than non-religious adolescents.

Inspection of table 3 reveals five important findings. Firstly, the relationship between friends' smoking at T1 and adolescent smoking at T2 was found to be significant in only one country. In Portugal there was a positive relation ($p = 0.02$). Hence, we did not find strong support for the peer influence hypothesis for this age cohort. Secondly, in all countries except for Spain and Portugal we found very significant positive relations between adolescent smoking at T1 and friends' smoking behaviour at T2 ($p < 0.01$). These results support the selection hypothesis. Thirdly, in all countries except for Spain and Portugal we found a significant relation between parents' smoking at T1 and adolescent's smoking behaviour on T2 ($p < 0.02$). Fourthly, in all countries except for Spain and Portugal parents' smoking behaviour at T1 was related to friends' smoking behaviour at T2 ($p < 0.02$). Fifthly, examining the relations between the smoking behaviour of the same actors across time reveals significant relations between T1 and T2 ($p < 0.02$) for almost all countries. This points to a certain stability of these behaviours across time.

The effect size measures for the four most important paths of the model were also calculated (table 3). The effect sizes for the path from T1 friends' smoking behaviour to T2 adolescent's smoking behaviour are small (0.002–0.55%), except for Portugal (2.04%). The reverse holds for the effect sizes for the path from T1 adolescent's smoking behaviour to T2 friends' smoking behaviour, since these are very small for Portugal (0.06%), but moderate to small for the other countries (1.2–5.2%). The effect sizes for both parental paths ranged from 0.08–1.38%. The same pattern is found in case the effect sizes calculated under full information maximum likelihood are considered (table 4).

To examine to what extent these results are dependent on the chosen imputation method, we replicated the analyses using full information maximum likelihood estimation on the available data. Table 4 shows that these results are also very similar to those reported in table 3. An exception is the negative relation for the Dutch sample between friends and adolescents suggesting less smoking onset when peers smoked. This possibly is due to this method being too sensitive to violations of normality.[40] Additionally, to study how dependent the results are upon the selection of covariates, robust maximum likelihood analysis was also done without any of the covariates. These results were very similar to the results obtained when the covariates were included. This shows the robustness of the results under inclusion of covariates and thus also indicates that the covariates are not on the causal pathways between predictor variables and the outcome variables.

DISCUSSION

The most remarkable finding of this study is that for most countries peer smoking among adolescents aged 12–13 years is no significant predictor of adolescent smoking behaviour one year later. The findings with regard to the relative risks suggest stronger selection then influence processes, and modest parental influences. However, these analyses do not correct for confounders and interrelationships between the actors (parents, friends and parents). The findings based on

Table 3 Results of the structural equation modelling using robust maximum likelihood for six countries: path-coefficients (with standard errors), significance levels and effect sizes

	Finland n = 1243	Denmark n = 572	Netherlands n = 1987	United Kingdom n = 1746	Spain n = 647	Portugal n = 907
Adolescent–adolescent (path 1)	0.818 (0.070) p<0.001	1.152 (0.103) p<0.001	1.180 (0.071) p<0.001	0.934 (0.085) p<0.001	0.973 (0.110) p<0.001	0.726 (0.143) p<0.001
Friend–friend (path 2)	0.693 (0.108) p<0.001	0.443 (0.176) p = 0.012	0.498 (0.089) p<0.001	0.215 (0.124) NS	0.606 (0.139) p<0.001	0.994 (0.313) p = 0.001
Parent–parent (path 3)	0.938 (0.043) p<0.001	0.862 (0.088) p<0.001	0.962 (0.034) p<0.001	0.887 (0.037) p<0.001	0.824 (0.085) p<0.001	0.822 (0.071) p <0.001
Friend–adolescent (path 4)	−0.021 (0.172) NS ES = 0.002%	−0.083 (0.199) NS ES = 0.03%	−0.224 (0.125) NS ES = 0.39%	−0.203 (0.208) NS ES = 0.13%	0.214 (0.156) NS ES = 0.55%	1.103 (0.480) p = 0.02 ES = 2.04%
Adolescent–friend (path 5)	0.115 (0.040) p = 0.004 ES = 1.25%	0.310 (0.074) p <0.001 ES = 5.16%	0.234 (0.046) p<0.001 ES = 4.2%	0.210 (0.047) p<0.001 ES = 3.31%	0.144 (0.074) NS ES = 1.16%	0.043 (0.083) NS ES = 0.06%
Parent–adolescent (path 6)	0.337 (0.099) p<0.001 ES = 1.38%	0.356 (0.160) p = 0.013 ES = 0.87%	0.160 (0.058) p = 0.006 ES = 0.55%	0.142 (0.054) p = 0.009 ES = 0.74%	0.025 (0.128) NS ES = 0.008%	0.158 (0.096) NS ES = 0.09%
Parent–friend (path 7)	0.153 (0.055) p = 0.003 ES = 1.08%	0.256 (0.109) p = 0.019 ES = 1.07%	0.132 (0.040) p = 0.001 ES = 1.24%	0.068 (0.029) p = 0.019 ES = 0.83%	0.116 (0.101) NS ES = 0.35%	0.044 (0.062) NS ES = 0.31%
Satorra Bentler	306.367 df = 97 p<0.001	236.394 df = 121 p<0.001	387.348 df = 114 p<0.001	266.803 df = 129 p<0.001	200.947 df = 129 p<0.001	259.920 df = 122 p<0.001
CFI	0.991	0.987	0.990	0.995	0.989	0.988
TLI	0.984	0.975	0.982	0.990	0.978	0.976
RMSEA	0.0417	0.0409	0.0347	0.0247	0.0294	0.0353
R^2 for adolescent smoking	0.442	0.627	0.527	0.455	0.406	0.552

CFI, comparative fit index; ES, effect size; RMSEA, root mean square of approximation; TLI, Tucker-Lewis index.

structural equation modelling, that do take into account the potential confounding effects, suggest more support for selection processes particularly in the Finish, Danish, Dutch and United Kingdom samples. We did not find this relation for the Spanish and Portuguese samples. Previous studies also found evidence for the importance of selection processes.[11 23 30 31 41] An interpretation of the path from T1 adolescent's smoking behaviour to T2 friends' smoking behaviour as social influence is highly unlikely, given that we did not find evidence for the social influence path from friends (a group of adolescents) to the adolescent.

Why was there not more evidence for peer influence, since this pattern was only found in Portugal? It may be that peer influences are most likely to be felt if youngsters are not smoking much themselves, a phenomenon found in Portugal. Another explanation may be that peer influences may be more prominent during different stages in adolescence. Hence, more studies among older adolescent groups are needed to test this hypothesis. Both explanations deal with peer influence salience, the first suggesting that peer influences towards smoking become salient when smoking is not yet a normal behaviour, the latter suggesting that salience increases as smoking prevalence among youngsters increases as well. The fact that we found a negative effect for peer influence in the Netherlands is hard to explain, although this finding only became significant when using the full

Table 4 Results of the structural equation modelling using full information maximum likelihood for six countries: path-coefficients (with standard errors), significance levels and effect sizes (ES) for path 4, 5, 6 and 7

	Finland n = 1243	Denmark n = 572	Netherlands n = 1987	United Kingdom n = 1746	Spain n = 647	Portugal n = 907
Friend–adolescent (path 4)	0.002 (0.148) NS ES = 0.00%	0.032 (0.179) NS ES = 0.005%	−0.184 (0.087) p = 0.0344 ES = 0.28%	−0.205 (0.166) NS ES = 0.13%	0.241 (0.126) NS ES = 0.77%	0.859 (0.308) p = 0.005 ES = 1.16%
Adolescent–friend (path 5)	0.133 (0.035) p<0.001 ES = 1.63%	0.333 (0.060) p<0.001 ES = 5.91%	0.243 (0.033) p<0.001 ES = 4.57%	0.224 (0.040) p<0.001 ES = 3.72 %	0.163 (0.066) p = 0.013 ES = 1.60%	0.040 (0.075) NS ES = 0.05%
Parent–adolescent (path 6)	0.328 (0.092) p<0.001 ES = 1.55%	0.351 (0.150) p = 0.0195 ES = 0.96%	0.156 (0.054) p = 0.004 ES = 0.57%	0.141 (0.048) p = 0.004 ES = 0.79%	0.024 (0.113) NS ES = 0.01%	0.122 (0.086) NS ES = 0.23%
Parent–friend (path 7)	0.148 (0.05) p = 0.003 ES = 1.18%	0.248 (0.107) p<0.001 ES = 1.21%	0.130 (0.037) p<0.001 ES = 1.27%	0.068 (0.027) p = 0.012 ES = 0.89%	0.108 (0.093) NS ES = 0.41%	0.045 (0.062) NS ES = 0.11%

information maximum likelihood method; a method not robust to violations of normality.[40] This finding may suggest that, sometimes, overt peer influences at a young age when smoking initiation has already started may have counter productive effects at this age as well.

Although modest relative risks were found in favour of support of parental influences, the more refined results of the structural equation techniques from the samples of Finland, Denmark, the Netherlands and the United Kingdom furthermore showed the importance of parental influences. Moreover, the data also revealed that parents' smoking behaviour at T1 was also related to friends' smoking behaviour at T2 (path 7). A previous longitudinal study also found an important contribution of parental smoking that was comparable in magnitude with peer influences for this age group.[31] One interpretation of this interesting finding is that parents may act as gatekeepers in the sense that parents' smoking behaviour can influence children to choose friends partly based on their friends' smoking status.

Our results suggest some different patterns for Portugal and Spain for two processes. Selection processes were found in the four northern countries, but were absent in Portugal, and only significant for Spain when using the full information maximum likelihood method. Similar differences were found for parental influences, which were absent in the Spanish and Portuguese samples. Although an explanation could be that differences in cultural climate may contribute to this finding[42] and result in more influences of other factors—such as positive attitudes towards smoking, lower self-efficacy expectations towards non-smoking, and more positive norms about smoking—more research is needed to be able to replicate and explain our finding for these two countries.

The study is subject to limitations. First, self-reported smoking behaviour was not validated by biochemical measures. Conversely, self-reports have been shown to be reliable and to correspond well with biological indicators when measurements are done under optimised measurement conditions, ensuring anonymity.[43] We optimised measurement conditions by reassuring the respondents of strict confidentiality of their responses.[3 43 44] Second, reports on parental and friends' smoking were based on the adolescents' reports. However, studies also utilising independent reports showed adolescents to be well aware of their parents' and friends' risky behaviours.[45] Third, friendships may change rapidly in adolescence and we may not have been able to assess these changes. Future research should use a strategy with shorter time intervals. Fourth, this study studied adolescents of 12–13 years of age. More research is therefore needed to be able to generalise the findings to older age adolescents. Finally, interpretation of parameter estimates of SEM techniques can be subject to discussion. Moreover, SEM techniques do not allow incorporating changes in friendship compositions during the waves. Future research using social network analysis may allow this possibility.

A major strength of this study is that the study has a 12 month longitudinal design. Two other strengths are that the study was carried out in six countries employing the same questions, and that the results showed fairly consistent results with regard to the absence of a peer influence path, and rather consistent patterns of selection processes. Additionally, SEM techniques were used to analyse the data. Relations between latent constructs based on observed variables were examined, thereby taking care of unreliability in the measures used. This can be considered an advantage over previous work.[23]

What are potential implications of our study? First, in line with several other studies we conclude that a more complicated smoking uptake pattern exists than suggested by the social influence approach. We need to realise that not all youngsters will take up smoking because of peer pressure but that a substantial portion of adolescents choose to do so themselves and consequently select their (smoking) friends. This implies that smoking prevention programmes for these youngsters should focus more thoroughly on developing an attitude in favour of non-smoking by outlining more clearly the advantages of non-smoking and the disadvantages of smoking. It is also conceivable that these youngsters have developed a positive attitude as the result of the influences of smoking adults, smoking peers, and the advertisements of the tobacco industry.[46] More research is needed to identify this particular group of youngsters that develop a pro-smoking attitude and how we can approach them by means other than programmes aimed at coping with peer pressures alone. Hence, social inoculation programmes alone may not suffice and smoking prevention methods may need a stronger emphasis on reinforcing non-smoking attitudes than resistance to peer pressure for this particular group.[41] Second, our results and other studies suggest that parental behaviours are still important for this age group,[21 47–49] although our study also suggests that the parental effect was not significant in all countries. Hence, studies analysing peer influences also need to include assessments of parental influences, since they have been found to be as least as important as peer influences in some countries for this age group.[31] More research is also needed to identify how parental behaviours exert their influence and whether they remain important during adolescence for older age groups. Moreover, studies suggest different mechanisms, such as parenting styles and parenting practices.[21 47–49] The lack of evidence based effective parenting practices stimulating smoking prevention indicates a clear need for more research in this area. Since our data support earlier evidence suggesting the impact of parental smoking, future smoking prevention research needs to include effective modules that stimulate smoking parents to quit smoking. Moreover, studies analysing reciprocal relationships are needed as well.[16 29 30] Third, the strongest relationship between T1 factors and adolescent smoking at T2 was found for previous adolescent behaviour. Hence, prevention interventions that focus on keeping the adolescent from starting smoking are vital. These programmes need to be tailored to the individual needs of the adolescent, since some youngsters may be in need of inoculation training, others may need feedback on attitude beliefs, and others may need self-efficacy enhancing information. The results of two Dutch

What this paper adds

Peer smoking behaviour is regarded as one of the most crucial factors for smoking onset in adolescence. One alternative explanation postulates the importance of selection processes implying that an (smoking) adolescent selects (smoking) friends. Furthermore, the influence of parents may be stronger in adolescence than initially considered. Our longitudinal study showed for adolescents of 12 or 13 years of age in six European studies that in only one country a significant path was found between friends' smoking status and adolescent smoking status one year later. Strong support on the other hand was found for the selection paradigm, suggesting that adolescents choose friends with similar smoking behaviour. Support for the impact of parents on adolescent behaviour and the choice of friends was also found. This study thus adds to the growing evidence suggesting alternative explanations for smoking onset and, consequently, points to the need for improved smoking prevention approaches.

tailored programmes for youngsters suggest the possibility of computer tailored interventions for primary and secondary school children.[50 51] Lastly, the fact that the effect sizes of our social indicators are modest suggests that anti-tobacco policies concerning price, availability and advertisements remain an essential tool within a comprehensive tobacco control policy.

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REFERENCES

1 **US Department of Health and Human Services**. *Preventing tobacco use among young people. A report of the Surgeon General, 1994*. Atlanta, Georgia: Public Health Service, Centers for Disease Control and Prevention, Office on Smoking and Health, 1994, (US Government Printing Office Publication No S/N 017-001-00491-0.).
2 **Flay BR**. Adolescent smoking: onset and prevention. *Ann Behav Med* 1985;**7**:9–13.
3 **Hansen WB**. School-based substance abuse prevention: a review of the state of the art in curriculum 1980–1990. *Health Educ Res* 1992;**7**:403–30.
4 **Peterson AV**, Kealey KA, Mann SL, *et al*. Hutchinson smoking prevention project: long-term randomized trial in school-based tobacco use prevention. Results on smoking. *J Natl Cancer Inst* 2000;**92**:1979–91.
5 **Vartiainen E**, Puska P, Koskela K, *et al*. Ten-year results of a community-based anti-smoking program (as part of the North Karelia Project in Finland). *Health Educ Res* 1986;**1**:175–84.
6 **Perry CL**, Kelder SH, Murray DM, *et al*. Communitywide smoking prevention: long-term outcomes of the Minnesota Heart Health Program and the Class of 1989 study. *Am J Public Health* 1992;**82**:1210–6.
7 **De Vries H**, Mudde A, Leijs I, *et al*. The European Smoking Prevention Framework Approach (EFSA): An example of integral prevention. *Health Educ Res* 2003;**18**:611–26.
8 **De Vries H**, Dijk F, Wetzels J, *et al*. The European Smoking Prevention Framework Approach: effects after 24 and 30 months. *Health Educ Res* (in press.)
9 **De Vries H**, Mudde A, Kremers S, *et al*. The European Smoking Prevention Framework Approach (ESFA): short-term effects. *Health Educ Res* 2003;**18**:649–63.
10 **Evans RI**, Dratt LM, Raines BE, *et al*. Social influences on smoking initiation: importance of distinguishing descriptive versus mediating process variables. *J Appl Soc Psychol* 1988;**18**:925–43.
11 **Bauman KE**, Fisher LA. On the measurement of friend behavior in research on friend influence and selection: findings from longitudinal studies of adolescent smoking and drinking. *J Youth Adolesc* 1986;**15**:345–53.
12 **Bauman KE**, Ennett ST. Peer influence on adolescent drug use. *Am Psychol* 1994;**49**:820–2.
13 **Cohen DA**, Richardson J, LaBree L. Parenting behaviors and the onset of smoking and alcohol use: a longitudinal study. *Pediatrics* 1994;**94**:368–75.
14 **Billy JOG**, Udry JR. Patterns of adolescent friendship and effects on sexual behavior. *Soc Psychol Q* 1985;**48**:27–41.
15 **Fisher LA**, Bauman KE. Influence and selection in the friend-adolescent relationship: findings from studies of adolescent smoking and drinking. *J Appl Soc Psychol* 1988;**18**:289–314.
16 **Urberg KA**, Degirmencioglu SM, Pilgrim C. Close friend and group influence on adolescent cigarette smoking and alcohol use. *Dev Psychol* 1997;**33**:834–44.
17 **Ennett ST**, Bauman KE. Peer group structure and adolescent cigarette smoking: a social network analysis. *J Health Soc Behav* 1993;**34**:226–36.
18 **Brown AF**, Rix EA, Cholvat J. Changing promotion criteria: cognitive effects on administrators' decisions. *J Exp Educ* 1983;**52**:4–10.
19 **Norton EC**, Lindrooth RC, Ennett ST. Controlling for the endogeneity of peer substance use on adolescent alcohol and tobacco use. *Health Econ* 1998;**7**:439–53.
20 **Aseltine RH**. A reconsideration of parental and peer influences on adolescent deviance. *J Health Soc Behav* 1995;**36**:103–21.
21 **Baumrind D**. The influence of parenting style on adolescent competence and substance use. *J Early Adolesc* 1991;**11**:56–95.
22 **Berndt TJ**, Keefe K. Friends' influence of adolescents' adjustment to school. *Child Dev* 1995;**66**:1312–29.
23 **Engels RCME**, Knibbe RA, De Vries H, *et al*. Influences of parental and best friends' smoking and drinking on adolescent use: a longitudinal study. *J Appl Soc Psychol* 1999;**29**:337–61.
24 **Kandel DB**, Kessler RC, Margulies RZ. Antecedents of adolescent initiation into stages of drug use: a developmental analysis. *J Youth Adolesc* 1978;**7**:13–40.
25 **Hansen WB**, Graham JW, Sobel JL, *et al*. The consistency of peer and parent influences on tobacco, alcohol, and marijuana use among young adolescents. *J Behav Med* 1987;**10**:559–79.
26 **Allegrante JP**, O'Rourke TW, Tuncalp S. A multivariate analysis of selected psychosocial variables on the development of subsequent youth smoking behavior. *J Drug Educ* 1977;**7**:237–48.
27 **Flay BR**, D'Avernas JR, Best JA, *et al*. Cigarette smoking: why young people do it and ways of preventing it. In: Mc Grath PJ, Firestone P, eds. *Pediatric and adolescent behavioral medicine*. New York: Springer, 1983:132–183.
28 **Caspi A**. Why maladaptive behaviors persist: sources of continuity and change across the life course. In: Funder DC, Park RD, Tomlinson-Keesey C, *et al*. *Studying lives through time: personality and development*. Washington DC: American Psychological Association, 1993:343–76.
29 **Urberg KA**, Luo Q, Pilgrim C, *et al*. A two-stage model of peer influence in adolescent substance use: individual and relationship-specific differences in susceptibility to influence. *Addict Behav* 2003;**28**:1243–56.
30 **Ennett ST**, Bauman KE. The contribution of influence and selection to adolescent peer group homogeneity: the case of adolescent cigarette smoking. *J Pers Soc Psychol* 1994;**67**:653–63.
31 **De Vries H**, Engels R, Kremers S, *et al*. Parents' and friends' smoking status as predictors of smoking onset: findings from six European countries. *Health Educ Res* 2003;**18**:627–36.
32 **Kremers SP**, Mudde AN, De Vries H. ''Kicking the initiation'': do adolescent ex-smokers differ from other groups within the initiation continuum? *Prev Med* 2001;**33**:392–401.
33 **World Health Organization**. *Guidelines for controlling and monitoring the tobacco epidemic*. Geneva: WHO, 1988.
34 **De Vries H**. Socio-economic differences in smoking: Dutch adolescents' beliefs and behaviour. *Soc Sci Med* 1995;**41**:419–24.
35 **Jöreskog K**, Sörbom D. *LISREL 8: structural equation modeling with the SIMPLIS command language*. Hillsdale, New Jersey: Lawrence Erlbaum, 1993.
36 **Chou CP**, Bentler PM, Satorra A. Scaled test statistics and robust standard errors for non-normal data in covariance structure analysis: a Monte Carlo study. *Br J Math Stat Psychol* 1991;**44**:347–57.
37 **Schafer JL**, Graham JW. Missing data: our view of the state of the art. 2002;**7**:147–77.
38 **Hair JF**, Anderson RE, Tatham RL, *et al*. *Multivariate data analysis*. Upper Saddle River, New Jersey: Prentice-Hall, 1998.
39 **HBSC**. *Young people's health in context*. Copenhagen: Health Behaviour in School-aged Children (HBSC), 2004.
40 **Enders CK**. The impact of nonnormality on full information maximum-likelihood estimation for structural equation models with missing data. *Psychol Methods* 2001;**6**:352–70.
41 **Wang MQ**, Fitzhugh EC, Green BL, *et al*. Prospective social-psychological factors of adolescent smoking progression. *J Adolesc Health* 1999;**24**:2–9.
42 **Nichter M**. Smoking: what does culture have to do with it? *Addiction* 2003;**98**(suppl I):139–45.
43 **Dolcini MM**, Adler NE, Ginsberg D. Factors influencing agreement between self-reports and biological measures of smoking among adolescents. *J Res Adolesc* 1996;**6**:515–42.
44 **Murray DM**, O'Connell CM, Schmid LA, *et al*. The validity of smoking self-reports by adolescents: a reexamination of the bogus pipeline procedure. *Addict Behav* 1987;**12**:7–15.
45 **Wilks J**, Callan VJ, Austin DA. Parent, peer and personal determinants of adolescent drinking. *Br J Addiction* 1989;**84**:619–30.
46 **Cummings KM**, Morley CP, Horan JK, *et al*. Marketing to America's youth: evidence from corporate documents. *Tob Control* 2002;**11**(suppl I):i5–17.
47 **Kerr M**, Stattin H, Trost K. To know you is to trust you: parents' trust is rooted in child disclosure of information. *J Adolesc* 1999;**22**:737–52.
48 **Jackson C**, Henriksen L. Do as I say: parent smoking, antismoking socialization, and smoking onset among children. *Addict Behav* 1997;**22**:107–14.
49 **Henriksen L**, Jackson C. Anti-smoking socialization: relationship to parent and child smoking status. *Health Commun* 1998;**10**:87–101.
50 **Ausems M**, Mesters I, Van Breukelen G, *et al*. Effects in-school and tailored out-of-school smoking prevention among Dutch vocational school students. *Health Educ Res* 2004;**19**:51–63.
51 **Ausems M**, Mesters I, Van Breukelen G, *et al*. Short-term effects of a randomized computer-based out-of-school smoking prevention trial aimed at elementary schoolchildren. The impact of a computer-tailored nutrition intervention. *Prev Med* 2002;**34**:581–9.