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Original article

Relative Risks of Exposure to Different Smoking Models on the Development of Nicotine Dependence during Adolescence: A Five-Wave Longitudinal Study

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Abstract

Purpose: Many studies have focused upon predictors of smoking onset and continuation in adolescents. However, less is known about the development of nicotine dependence (ND) and how smoking in the interpersonal environment relates to this. To examine which smoking models have the largest impact on the development of ND, we examined the relative impact of current smoking from fathers, mothers, siblings, best friends, and friend groups on the development of ND in adolescents.

Methods: Data were used from five annual waves of the ''Family and Health'' survey project. At baseline 428 adolescents (mean age = 15.2 years; SD = .60), both their parents, and their siblings were participating. In this study we included only smoking adolescents (n = 175). To assess the individual development of ND of each adolescent, and whether current smoking exposure affected changes from baseline across time, we used latent growth curve modeling (LGC).

Results: Findings revealed that smoking of sibling and best friend were related to baseline levels of ND, but not to the rates of change over time. Of all models, only having smoking friends in the friend group was related to a faster development of ND.

Conclusions: The current findings highlight the key role of smoking friends in the development of ND and suggest that interventions targeting at peers are probably effective in reducing the prevalence of ND symptoms among adolescents.

Keywords: Adolescence; Nicotine dependence; Development; Smoking models

Smoking uptake in adolescence has been conceptualized as progressing through a sequence of developmental stages. All stages are characterized by differences in smoking frequency and intensity; these stages could be labeled as the nonsmoking stage, trying stage, experimental stage, regular smoking stage, and established/daily smoking stage [1]. Research so far has concentrated primarily upon the first stages of smoking, in which adolescents make a transition from never smoking to experimenting with cigarette use. However, transitions into the more advanced stages of smoking, along with the development nicotine dependence (ND), have been given relatively less attention [2], which is intriguing because it is exactly these stages that are closely linked to the detrimental health effects of smoking on the long term. Therefore, in the present study we focused upon predictors of the development of ND.

Although a ''gold standard'' for identifying and classifying ND is presently absent [3], according to most recent diagnostic guidelines it could be characterized by tolerance, craving, withdrawal symptoms, and loss of control over the amount or duration of use [4,5]. In contrast to the assumption that ND only develops after regular smoking, DiFranza *et al* [6] demonstrated that even irregular smokers can experience withdrawal, craving for cigarettes, and failed attempts at quitting. To advance understanding in the etiology of ND, it is important to elucidate how it develops or emerges [7]. Previous research showed that adolescents differ in their development of ND; individual variations were found in the occurrence and intensity of ND symptoms, with some

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adolescent smokers being more vulnerable for the development of dependence symptoms than others [6]. Insight into the mechanisms underlying these individual differences is needed to fully understand why some adolescents develop ND at a faster rate while others appear to be resistant to developing dependence.

A considerable body of empirical research has identified the social environment as an important factor affecting adolescent (non-)engagement in smoking. First, the family environment appears to play a role, as increased risks for experimentation and progression to regular smoking were found for adolescents with at least one smoking parent [1,2,8]. Adolescents who are exposed to sibling smoking are also more at risk for smoking, especially for initiation [1,2,8]. Second, of all persons in the social environment, friends are considered to be a key factor for smoking involvement. Both smoking by best friends and the prevalence of smoking in the peer group are found to be related to juvenile smoking [1,8,9]. Importantly, best friends and friend groups were found to contribute to the prediction of adolescents' smoking uniquely: Close friends appear to play a role in the initiation of smoking, whereas cigarette use in the friend group was related to the transition into regular smoking [10].

Although smoking by significant others turns out to be important predictors of the onset and continuation of smoking, the influence of these models upon the development of ND is not well illuminated. Having smoking individuals in the interpersonal environment may promote the growth of ND in various ways. According to the Social Learning Theory, the tendency to model or imitate behavior of others is an important determinant of human behavior [11], this applies also to smoking [2,9]. Exposure to smoking models might contribute to an acceleration of ND by increasing the intensity of smoking [12,13]. Besides evoking modeling behavior, smoking individuals may promote the development of ND also by providing adolescents with easy access to cigarettes, for instance by offering cigarettes [14], and by acting as cues that trigger craving [15].

Higher levels of ND in adolescents were indeed related to exposure to parental smoking [16–18], sibling smoking [19], and friends' smoking [16,19,20]. Nevertheless, in all these studies smoking is conceptualized in various ways, which makes it difficult to establish the actual impact of current exposure to each smoking model separately while taking the influences of all other models into account. In none of these studies was ND regarded as a developmental process to examine why some adolescents progressed in their levels of ND at a faster rate than others, except in the study from Audrain-McGovern *et al* [20]. The focus in that study was not, however, on exposure to smoking of several models, as only friends' smoking and household members' smoking were included. In the present study, we focused on the roles of smoking models in relation to development of ND [3,21,22].

The question arises which smoking models are most important in the development of ND. It is generally known that, during adolescence, the importance of friendships increases, whereas the intensity and exclusivity of the parent–child relationship decreases [23]. Therefore, friends are expected to play the most prominent role in evoking modeling, increasing the availability of smoking, and acting as smoking cues. This assumption is supported by a study on the relative contributions of exposure to smoking by parents and friends on smoking onset, revealing that among older adolescents, friends' smoking appeared to be more important in predicting smoking initiation than parental smoking, whereas for preadolescents the impact of parental smoking was found to be more essential [24]. Based on this, one can expect that of all smoking models in the interpersonal environment, smoking friends are more important in explaining the growth of levels of ND in youth.

To gain more insight into the etiology of ND, the present study extends previous work by investigating the relative impact of current exposure to smoking from fathers, mothers, siblings, best friends, and friend groups in relation to adolescents' development of ND. We hypothesized that the influence of smoking friends would be stronger than the influence of parental smoking. In our analyses we controlled for possible confounding effects of adolescents' gender, education level, age of first smoking, and baseline smoking [12,16,20].

Methods

Procedure

Data were used from the first five waves of the ''Family and Health'' project, in which 428 families with mother, father, and two adolescent children were participating [25,26]. The aim of this project is to gain insight into different socialization processes underlying various health-related behaviors in adolescence, such as smoking and alcohol use. Addresses of families eligible for participation were acquired from 22 municipalities in the Netherlands. Families were included if they fulfilled the following criteria: parents had to be married or living together, all family members had to be biologically related, and the siblings should be neither twins nor mentally or physically disabled. Initially, these families were invited to participate for three annual measurements. Later the families were approached again to ask whether they would participate for another three times. So far, data have been collected in five waves. Data collection for the baseline measurement (T1) took place between November 2002 and April 2003. The numbers of participating families were 416 (T2), 404 (T3), 356 (T4), and 326 (T5), resulting in a response rate of 76% across waves.

At baseline, interviewers visited the families at home, asking all family members to complete the questionnaires individually. During the subsequent measurements, interviewers visited the majority of these families again. Part of the families received the questionnaires by mail for practical and financial reasons. The numbers of families who participated by mail were 8% (T2), 24% (T3), 11%

(T4), and 25% (T5). To maintain confidentiality, the families who received mail-in surveys obtained a letter in which all family members were requested to complete the questionnaires individually and not to discuss the questions with each other. Each family received €30 per wave if all family members had completed the questionnaires. Between the families participating in the first three waves, five travel checks of €1000 were raffled. For participation in the other waves, five iPods and five additional travel checks will be raffled.

Sample characteristics

Research questions were tested for the older siblings only, as symptoms of nicotine dependence (ND) are more likely to occur in this age group [16]. (It should be noted that there were pragmatic reasons for not testing our research questions for the younger siblings as well. First, the group of currently smokers was quite small for investigating a rather complex model ($n = 138$). Second, the LGC model without the predictors showed no optimal fit (χ^2 [$df = 10$, $n = 138$] = 18.69, CFI = .93, and RMSEA = .08), which implies that the development of nicotine dependence is difficult to establish for adolescents in this age group.) At baseline, the mean age of these adolescents was 15.2 years (SD = .60). Boys and girls were approximately equally represented, with 47.2% of the adolescents being girls. Attrition analyses revealed differences between families that participated in five times and those that dropped out. Children of families that dropped out were less likely to follow higher education (OR = .52, 95% CI = .31–.89, $p < .05$) and to have higher levels of nicotine dependence at baseline (OR = 1.82, 95% CI = 1.09–3.04, $p < .05$).

Measures

Adolescents' levels of nicotine dependence. Levels of nicotine dependence (ND) were measured with the Fagerström Test for Nicotine Dependence (FTND), which showed to have acceptable levels of internal consistency and was found to be related closely to biochemical indices of heaviness of smoking [27]. The minimum score on this scale is zero, indicating no dependence, with scores to increase up to a maximum score of 10, indicating strong dependence. An advantage of having repeated measures of this variable is examination of the rate at which adolescents develop ND over time [20].

Adolescents' smoking at baseline. Adolescents were asked to report on a nine-point scale which smoking stage applied to them [25]. Responses ranged from 1 = ''I never smoked, not even one puff'' to 9 = ''I smoke at least once a day.'' Because of the skewness of the distribution, this variable was transformed into a new variable ranging from 1 to 5.

Adolescents' age first smoked. Adolescents were asked at each wave: ''At what age did you first smoke, even though it was only one puff?'' For this study we used the responses most closely to the actual age of onset, namely the first measurement moment at which adolescents reported to smoke. By doing this the period of time between the actual age of initiation and the report of this age was as short as possible, which contributed to the establishment of this age most adequately [28].

Parental smoking. Both fathers and mothers were asked the similar question as the adolescents to assess their smoking status. However, one of the nine responses (i.e., ''I try smoking once a while'') was not appropriate for parents, as trying out smoking seldom occurs in adulthood when most people already have established a certain smoking status. Therefore, parents were asked to report on an eight-point scale which stage of smoking applied to them [25]. As the present study aimed at providing insight into the impact of currently smoking parents, we classified each parent, based on their baseline responses, into ''current nonsmokers'' or ''current smokers.''

Sibling's smoking. Siblings were asked the same question as the target adolescents [29]. Because we aimed to study the impact of current exposure responses to this question at baseline were dichotomized into ''current nonsmokers'' versus ''current smokers.''

Best friend's smoking. Respondents were asked to report on a nine-point scale which stage of smoking applied to their best friend [29]. Responses ranged from 1 = ''My best friend never smoked, not even one puff'', to 9 = ''My best friend smokes at least once a day.'' Answers to this question at baseline were dichotomized into ''current nonsmokers'' versus ''current smokers.'' This dichotomization was required to provide insight into the effect of current exposure.

Having smoking friends. To determine whether the adolescents had smoking friends, they were asked: ''How many of your friends smoke?'' Responses were 1 = ''No one,'' 2 = ''Less than half,'' 3 = ''Half,'' 4 = ''More then half,'' and 5 = ''All of them.'' Answers to this question at baseline were dichotomized into ''having no smoking friends'' versus ''having smoking friends.''

Strategy of analyses. For the purpose of this article, we included exclusively adolescents who were currently smoking (i.e., occasionally or daily) at least at one point in time during the measurement period (n = 175). After calculating descriptive statistics, we used latent growth curve modeling (LGC), which is a multivariate method that models repeated measures of an observed variable on latent variables representing the initial status at baseline and the rate of change over time [30]. In this study, individual development is captured by the initial degree of

ND at baseline (intercept) and the rate of change from baseline across time (slope). An advantage of this approach is that it not assumed that all adolescents start at the same level of ND at baseline and progress in ND at the same rate: individual growth is established for each adolescent separately. Hence LGC is an excellent way to take individual variations in the development of ND into account and to determine which variables are associated with these different developments. For these analyses we used Mplus [31]. Parameters in the models were estimated by applying the maximum likelihood estimator with robust standard errors (MLR) [31]. This estimator was developed to obtain robust standard errors when dependent variables have a nonnormal distribution. To make optimal use of the data we used the full-information maximum likelihood (FIML) approach. In this case, all available information in the data will be used, using pairwise comparisons [31].

In the first step, we tested the LGC model without the predictors. In the second step, we tested the relation between each predictor and the development of ND separately. Finally, we included all predictors to investigate the unique variance of each predictor in relation to the development of ND while controlling for the others (Figure 1). It is important to note that we investigated the relative impact of smoking by father, mother, sibling, best friend, and friend group on the development of ND, while controlling for possible confounding effects of adolescents' gender, education level, age first smoked, and baseline smoking. To avoid statistical power problems we decided not to include additional control variables. Model fit was assessed by the following global fit indices: χ^2, CFI (with a cut-off value of .95), and RMSEA (with a cut-off value of .06) [32].

Results

Descriptive statistics

To ensure that none of the adolescents were already strongly dependent at baseline, we checked the levels of nicotine dependence (ND). Findings showed that none of the adolescents were strongly dependent at the first wave (scores on the Fagerström Test for Nicotine Dependence [FTND] were $\leq$7, indicating that the target adolescents were not strongly dependent). Further descriptive findings revealed that the average level of ND at baseline was low (mean = .70; SD = 1.49) and that during the measurement period the average level of ND increased significantly (Wilks' Λ = .85, F [1, 65] = 11.56, $p < 001$) to a level of .90 (SD = 1.64) at the last wave. The mean levels of the five repeated measures of ND are illustrated in Figure 2. Descriptive statistics for the independent variables are presented in Table 1. Chi-square tests and t tests showed no gender differences for any independent variable and the repeated measures of ND. Correlations between the model variables are presented in Table 2. These findings showed significant associations between ND and the independent variables, except for gender and paternal smoking.

Model findings

First, we tested the latent growth curve (LGC) model without predictors. This model showed a good fit to the data (χ^2 [df = 10, n = 175] = 11.77, CFI = .99, and RMSEA = .03). The intercept and slope were significant (respectively β_0 = .43, $p < .001$ and β_1 = .36, $p < .001$), indicating that the participants on average scored greater than zero on level of ND at baseline and that levels of

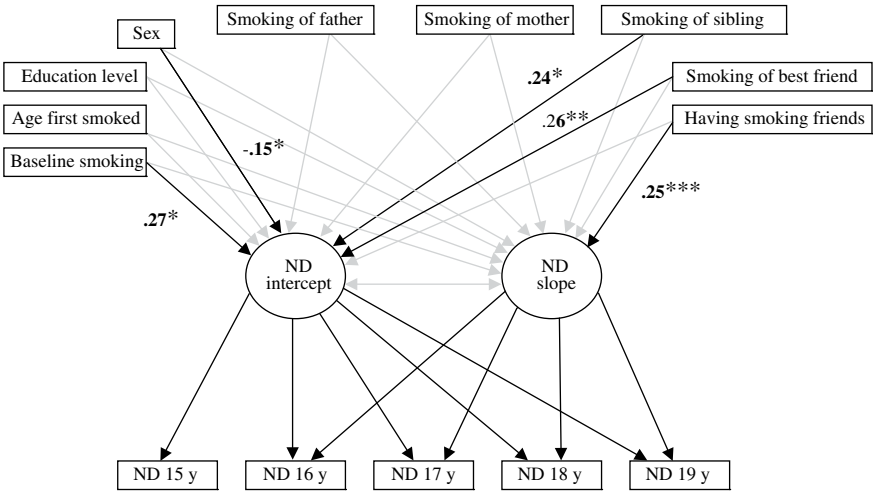

Figure 1. Latent growth curve (LGC) model concerning the role of smoking models on the development of nicotine dependence (ND) in adolescence. The five lower boxes in the figure represent the repeated measures of nicotine dependence. ND intercept represents the initial level of nicotine dependence at baseline and ND slope represents the rate of change over time. Only standardized estimates for the significant paths are presented. Nonsignificant pathways are portrayed in gray. Gender is represented as follows: 0 = boy and 1 = girl. * $p < .05$ ** $p < .01$, *** $p < .001$.

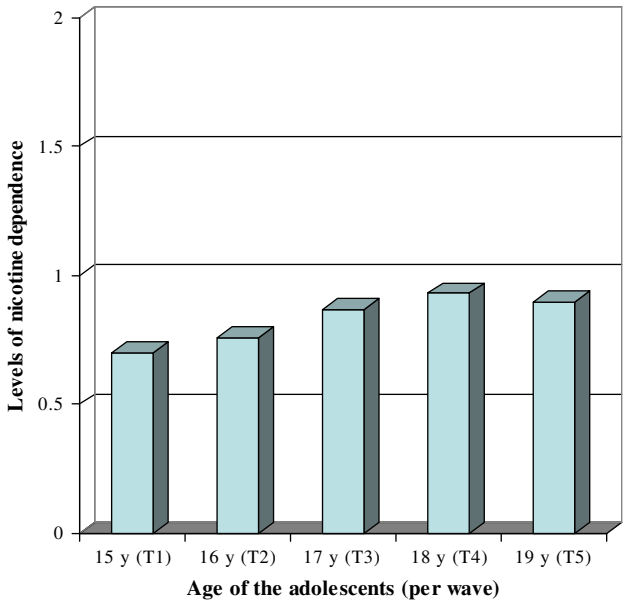

Figure 2. Bar chart representing the mean levels of the repeated measures of nicotine dependence.

dependence became higher over time. We also tested the model with a quadratic trend, but this trend was not significant and was therefore omitted.

Second, we examined the predictive values of smoking of significant others in relation to ND. All standardized estimates and standard errors of these analyses are presented in Table 3. Initially, each predictor was included separately to examine its predictive value while not taking other influences into account. Findings from these analyses showed that all independent variables were related significantly to ND if included as single predictors, except for gender and smoking of the father. Finally, the full model with all predictors was tested. This model showed a good fit (χ^2 [$df = 37$, $n = 175$] = 40.33, CFI = .99, and RMSEA = .02). Findings of the full model revealed that gender was significantly and negatively related to the intercept ($\beta = -.15$, $p < .05$), indicating that girls were less likely to have higher levels of ND at baseline than boys. Baseline smoking was positively associated with the intercept ($\beta = .27$, $p < .05$), which means that the more adolescents smoked, the higher their initial levels of ND. Smoking of sibling ($\beta = .24$, $p < .05$) and best friend ($\beta = .26$, $p < .01$) were also positively associated with the intercept, implying that having smoking siblings or best friends is related to higher levels of ND at baseline. Finally, having smoking friends in the friend group was positively related to the slope ($\beta = .25$, $p < .001$), indicating that having smoking friends resulted in an increase of ND across the five waves. Findings for the full model are displayed in Figure 1.

Discussion

In this study we established the individual development of nicotine dependence (ND) among a group of adolescent smokers and we examined the relative impact of exposure to current smoking of father, mother, sibling, best friend, and the friend group on this development. Findings revealed that smoking of sibling and best friend were related to the initial level of ND but not to changes over time. Only having smoking friends was related to increased levels of ND, implying that, of all smoking persons, friends are the most influential. The important role of friends in the final stages of smoking is also acknowledged by Urberg *et al* [10], who demonstrated that friend-group smoking was related to adolescents' transition into regular smoking. Mechanisms underlying this relationship may be that friends increase adolescents' frequency of smoking along with higher levels of ND, by evoking modeling [2,9,11], providing easy access to cigarettes [14] and acting as craving cues [15]. Nevertheless it is plausible that befriended adolescents affect each other also directly in their ideas about smoking. Perhaps friends talk with each other about their feelings of craving, for instance, by expressing their needs for a cigarette, and through that they might stimulate each other in their experiences of ND. It is important that future research unravels the mechanisms underlying the association between friends' smoking and adolescents' acceleration of ND more closely through, for example, observation and quantitative diary studies. It is also relevant to disentangle not only the effects that friends have on the adolescents but also the effects that the adolescents have on their friends. After all, the association between friends' smoking and increased levels of ND reflects most probably a reciprocal relationship.

Table 1
Percentages for independent variables (n = 175)

	%
Age first smoked	
≤12 years	32
13–15 years	51
≥16 years	16
Adolescent smoking at baseline	
Never smoked	26
Tried smoking	24
Stopped smoking	7
Smoked occasionally	22
Smoked daily	19
Smoking status of father	
Nonsmoking	73
Smoking	26
Smoking status of mother	
Nonsmoking	74
Smoking	25
Smoking status of sibling	
Nonsmoking	85
Smoking	14
Smoking status of best friend	
Nonsmoking	63
Smoking	35
Having smoking friends	
No	15
Yes	84

Table 2
Correlations between the model variables

	1	2	3	4	5	6	7	8	9	10	11	12	13
Gender[a]													
Education level	.08												
Age first smoked	.06	.21**											
Baseline smoking	.06	−.34**	−.58**										
Smoking of father	−.07	−.01	−.05	.05									
Smoking of mother	−.02	−.20*	−.04	.10	.43**								
Smoking of sibling	.05	−.14	−.30**	.26**	.09	.12							
Smoking of best friend	.05	−.21**	−.29**	.57**	−.01	.02	.18*						
Having smoking friends	−.09	−.19*	−.07	.32**	.03	.03	.03	.28**					
Nicotine dependence T1	−.14	−.34**	−.42**	.50**	.14	.26**	.37**	.44**	.16				
Nicotine dependence T2	−.15	−.25**	−.46**	.43**	.09	.10	.29**	.40**	.19*	.73**			
Nicotine dependence T3	−.14	−.32**	−.32**	.45**	.10	.11	.25**	.28**	.21*	.63**	.71**		
Nicotine dependence T4	−.10	−.22*	−.09	.16	.01	.13	.01	.26**	.21**	.47**	.70**	.56**	
Nicotine dependence T5	−.12	−.12	.11	.05	−.03	.06	.01	.14	.22**	.15	.37**	.44**	.77**

[a] 0 = boy; 1 = girl.
* $p < .05$.
** $p < .01$.

Remarkably, while controlling for the impact of all other models, parental smoking was not related to the initial level of ND or to changes in ND over time. This is in contrast with previous findings [16–19]. In the present study we were interested in current exposure to parental smoking, rather than parental history of smoking, which might explain why these differences were found. In addition, instead of focusing on the emergence of ND as a state outcome, we investigated how levels of ND progressed over time, which makes comparison between our findings and previous research difficult. Nevertheless, on a theoretical basis these findings can be explained by the fact that during adolescence friendships become closer and more important, while the time spent with parents is reduced [23]. This shift in close relationships might clarify why no effects were found for parental smoking.

Some limitations of this study should be acknowledged. First, it is possible that our findings are affected by selective drop out, as attrition analyses showed that adolescents with higher levels of ND were slightly more likely to drop out. However, because of our analytic approach, in which we were able to make optimal use of the available data, the chance of distortion is minimal. Second, we used mainly self-reports. Previous research has shown that self-reported data of adolescents about their smoking are reliable [33]; however, self-reports are also found to be affected by the context in which these were assessed [34]. Thus, despite the fact that confidentiality was warranted by asking all family members to complete the questionnaires individually, adolescents might have underreported their smoking because they were in the presence of their parents. In the Netherlands, the prevalence of smoking is higher among the total population than among the adolescents in the current study [35]. Nevertheless, it is important to note that this lower prevalence could also be explained by the fact that the adolescents in the present study are from intact families; previous findings

Table 3
Standardized estimates and standard errors of the smoking status of significant others in relation to nicotine dependence

	Single predictor				Full model			
	Intercept		Slope		Intercept		Slope	
	β	SE	β	SE	β	SE	β	SE
Gender[a]	−.12	.08	−.05	.11	−.15*	.06	.01	.11
Education level	−.35***	.06	−.01	.11	−.11	.06	−.09	.12
Age first smoked	−.48***	.07	.25*	.12	−.08	.12	.08	.15
Baseline smoking	.56***	.06	−.19	.10	.27*	.14	−.15	.13
Smoking of father	.15	.12	−.09	.10	.02	.10	−.04	.09
Smoking of mother	.25*	.10	−.12	.10	.16	.10	−.10	.10
Smoking of sibling	.43**	.12	−.21*	.09	.24*	.12	−.10	.11
Smoking of best friend	.50***	.06	−.15	.10	.26***	.07	−.09	.12
Having smoking friends	.18***	.03	.14***	.04	−.07	.04	.25***	.06

[a] 0 = boy; 1 = girl.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

indicate a higher smoking prevalence among adolescents from single-parent families than among adolescents from two-parent homes [36]. The assessment of ND by self-reports has also the shortcoming of determining merely behavioral dependence rather than physiological dependence. However, at this time, research has not identified an epidemiologic instrument that fully captures the development of ND [3,21,22]. Third, to assess friends' smoking we used adolescents' reports. Adolescents are inclined to project their own behavior onto their friends [37]. Because of this rater effect, it might be that the smoking status of the best friend and the smoking prevalence within the friend group are somewhat overestimated. Finally, because of the inclusion criteria for participation in the ''Family and Health'' project, one should be cautious when generalizing the present findings to, for example, adolescents of nonintact families or adolescents with no siblings.

In this study we focused upon environmental factors to predict the development of ND. We took only the smoking status of significant persons at baseline into account, and did not examine whether these persons changed their behavior over time. It would be interesting to investigate whether changes in smoking by important persons are related to changes in adolescents' behavior. Moreover, we examined exclusively the impact of exposure to current smoking of significant others and not what these individuals might do to encourage or discourage adolescents to smoke. For instance, a recent study of Luther *et al* [38] showed that adolescents who were allowed to smoke at home smoked more cigarettes per day and had higher levels of ND than those who were not allowed to smoke. Future research should investigate whether such activities function as a protective buffer against the impact of smoking friends. Finally, it is essential to take adolescents' characteristics into account as well. Recently, Laucht *et al* [39] provided preliminary evidence of genetic influences on different stages of smoking and highlighted the importance of specific dopamine genes in smoking progression. It would be challenging to replicate the present findings while taking these genetic predispositions into account.

To summarize, the present findings revealed that, in the development of ND, friends are of major importance. Adolescents who smoke are more vulnerable to growth in their levels of ND if they have smoking friends. This study highlights the importance of focusing on different stages of smoking to map the different risk factors related to smoking and ND. We hope that our findings encourage future studies to concentrate on dissimilarities in (social) influences on different stage transitions to provide more insight into the development of smoking and ND. Moreover, this study has implications for policy strategies. If friends play a crucial role, it is effective to develop and carry out programs in which this role is acknowledged. A recent cluster randomized controlled trial from Campbell *et al* [40] revealed that targeting peers appears to be fruitful to reduce smoking prevalence in adolescents. Their intervention consisted of training influential students to act as peer supporters during informal interactions to encourage their peers not to smoke. Perhaps such interventions are a key to lower levels of ND in adolescents and, subsequently, to lower rates of smoking-related mortality.

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