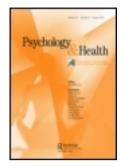
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# Psychology & Health

Publication details, including instructions for authors and subscription information:

http://www.tandfonline.com/loi/gpsh20

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Available online: 21 Aug 2009

To cite this article: Z. Harakeh, R. Engels, E. Den Exter Blokland, R. Scholte & A. Vermulst (2009): Parental communication appears not to be an effective strategy to reduce smoking in a sample of Dutch adolescents, Psychology & Health, 24:7, 823-841

To link to this article: http://dx.doi.org/10.1080/08870440802074649

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# Parental communication appears not to be an effective strategy to reduce smoking in a sample of Dutch adolescents

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(Received 1 May 2007; final version received 24 March 2008)

This longitudinal study examined the reciprocal effects of the frequency of parent–adolescent communication on tobacco-related issues (smoking-specific communication), and adolescents' smoking. Participants were 428 Dutch older and younger siblings between 13 and 16 years old. Smoking-specific communication did not affect youth smoking in general; however, among younger, but not older, siblings, smoking-specific communication was associated with a higher likelihood of smoking over time. In addition, when adolescents already smoked parents started to talk more frequently about smoking-related issues with their older and younger adolescents later on. Neither the quality of smoking-specific communication, the quality of parent–adolescent relationship, nor parental smoking moderated these reciprocal effects. In conclusion, prevention campaigns encouraging parents to undertake smoking-specific communication might not be desirable.

Keywords: adolescent; smoking; smoking-specific parenting; communication

#### Introduction

Adolescence is recognised as a period in which individuals initiate and experiment with substance use, such as smoking. Because of the addictive nature of nicotine, adolescents who experiment with smoking are at high risk to develop a regular smoking pattern and to continue smoking in young adulthood. In the Netherlands, the lifetime smoking prevalence rates (Stivoro, 2004) show a sharp increase in the adolescent years; i.e. among the 10-year-old youngsters 13% have smoked once or more, whereas among 13-year olds 35% have smoked and for 15- and 17-year olds, these rates increase to 59 and 69%, respectively. The prevalence of daily smoking increases from 0% for 10-year olds, 1% for 12-year olds, 3% for 13-year olds, 26% for 15-year olds, 28% for 17-year olds, to 30% for 19-year olds. Discouraging adolescents to start smoking remains a very important issue, and will protect them from smoking-related health problems in the future.

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To prevent their adolescents from smoking, parents have to resort to specific activities. Although the times spent together between adolescents and parents tend to decrease between early and late adolescence (Larson, Richards, Moneta, Holmbeck, & Duckett, 1996), the parents continue to influence their adolescents' norms, values and behaviours (e.g. Deković & Meeus, 1997; Parke & Ladd, 1992). In line with the evidence that parents remain influential in the adolescent period, a common assumption is that parents who continue to communicate with their adolescents are more successful in preventing risk behaviours of their offspring. Based on this assumption, prevention campaigns often recommend and encourage parents to communicate with their adolescents about tobacco-related issues (e.g. Miller-Day, 2002; Stivoro, 2005). The literature on the impact of smoking-specific communication is, however, not conclusive and has yielded conflicting results. Some of the findings in the literature challenge the assumption that parent-adolescent communication prevents adolescents from smoking. The majority of the studies examining the association between smoking-specific communication and adolescent smoking have been cross-sectional. These studies showed that parental smoking-specific communication was a protective factor (e.g. Chassin, Presson, Todd, Rose, & Sherman, 1998; Jackson, 1997; Jackson & Henriksen, 1997), while other cross-sectional studies indicated that parents who often communicate smoking-related topics may have children that are *more* likely to smoke (e.g. Engels & Willemsen, 2004; Harakeh, Scholte, De Vries, & Engels, 2005). In contrast, the few existing longitudinal studies showed that smoking-specific communication had no significant effect on adolescent smoking (e.g. Chassin, et al. 2005; Den Exter Blokland, Hale, Meeus, & Engels, 2006; Ennett, Bauman, Foshee, Pemberton, & Hicks, 2001; Otten, Harakeh, Vermulst, Van Den Eijnden, & Engels, 2006).

Miller and colleagues (Miller, Kotchick, Dorsey, Forehand, & Ham, 1998, p. 96; see also Miller-Day, 2002) define parent-adolescent communication as dialogues that are continuous, sequential and time sensitive. Therefore, they suggested that parents should react and build upon changes in the child's cognitive, emotional, physical and social development and should also respond to the child's questions and anticipated needs. This definition of communication between parents and adolescents indicates how complex the communication process is. In addition, there is evidence suggesting that communication between parent and adolescent may differ for mothers and fathers. It has been reported that, during childhood and adolescence, mothers (as compared to fathers) more frequently with their children and cover a broader range of topics (e.g. Miller-Day, 2002). Adolescents indicated that they felt more attached to their mothers and felt more at ease about talking with them about issues related to alcohol, tobacco and other drug use (Miller-Day, 2002). Therefore, it is important, when investigating communication between parent and adolescent, to look at mothers and fathers separately. In sum, communication should be continuous and interactive between parents (mother and father) and adolescent are important. Thus, it is not only a matter of investigating whether or not communication occurs between parents and adolescents (and its frequency) but also the quality and effectiveness of that communication. Previous studies on smoking, however, have frequently investigated the effects of parents on children but less is known on how children may affect the behaviour of their parents. This one-way approach frequently used in previous prospective studies on smoking limits our understanding of how parents and children interact in 'real life'. It is obvious that, when examining socialisation processes, both persons involved in the socialisation process affect each other. Parents react to the specific characteristics, needs and behaviours of their children and, in turn, their children react to their parents' characteristics and behaviours (Kerr & Stattin, 2003; Stice & Barrera, 1995). Therefore, it is plausible that adolescent smoking affects smoking-specific communication. Thus, the reciprocal effects of smoking-specific communication and adolescent smoking are important.

In aiming to understand the effectiveness of frequent communication on health issues like smoking, other important aspects in the process of communication (such as timing, style or manner, general family environments and parental smoking) are necessary to take into account. First, with respect to timing, Ennett, et al. (2001) suggested that parents who waited or delayed communication about smoking-specific issues until they assumed that their adolescent was smoking, and then tried to discourage him/her, are counterproductive. Therefore, communication might be most effective when children had not yet started to experiment with smoking. The timing of communication was also important, since older and younger siblings within a family might react differently to their communications, other parenting strategies and/or parental (e.g. Sulloway, 1995). A review by Darling and Cumsille (2003) suggested that older children might react differently to parental strategies than their younger siblings. In addition, Sulloway (1995) argued that older siblings felt more connected with their parents and acted perhaps more responsible, while younger siblings reacted more rebellious towards parental actions. Therefore, it could be possible that the effects of frequency of communication on adolescent smoking might have counterproductive effects on younger siblings' smoking but not on older siblings' smoking. The current study included two adolescent siblings of one family and will provide new insights into the effects of smoking-specific communication on older and younger siblings' smoking.

Second, it may be important to know whether the smoking-specific discussions between parents and adolescents take place in a respectful and constructive manner (i.e. quality of smoking-specific communication). Previous findings showed that the higher the quality of parent–adolescent smoking-specific communication, the less likely adolescents are to smoke (Harakeh et al., 2005). We, however, do not know yet whether the quality of smoking-specific communication moderates the effects of frequency of parent–adolescent communication and adolescent smoking.

Third, the quality of the parent-adolescent relationship might also moderate the effects of frequency of parent-adolescent communication and adolescent smoking. Previous studies showed that, if the overall quality was satisfactory, parents would be more accurate in identifying the smoking status of their adolescents, would monitor their adolescents in an appropriate way and would also communicate more with their adolescents on different topics, including smoking (Jaccard, Dittus, & Gordon, 1998).

Finally, non-smoking parents are more frequently and more constructively engaged in discussing smoking related-topics with their adolescents than smoking parents (Den Exter-Blokland et al., 2006; Harakeh et al., 2005; Henriksen & Jackson, 1998). A longitudinal study by Chassin et al. (2005), including mother and child reports, showed that there was no main effect of smoking-specific communication on adolescent smoking, but child reports indicated an interaction effect of smoking-specific communication and parental smoking. Adolescents with non-smoking parents were less likely to smoke if their parents communicated with them about smoking-specific topics while smoking-specific communications between adolescents and smoking parents did not affect adolescents' smoking (Chassin et al., 2005). It is important to take parental smoking into account because of the above-mentioned evidence that smoking and non-smoking parents differ in their communication strategies.

# Design of the present study

The aim of this longitudinal study was to gain information on the reciprocal effects of frequency of smoking-specific parent—adolescent communication on one hand and older and younger siblings' smoking on the other. One of the unique features of this present study was that the two adolescent siblings participated within one family. Therefore, the present study enabled us to provide new and unique insights into whether the patterns of smoking-specific parent—adolescent communication and adolescent smoking were similar for older and younger adolescent siblings within one family.

Longitudinal data from 428 families including two adolescent siblings from 13- to 16-years old were used. Further, this present study examined whether the reciprocal effects of smoking-specific communication and adolescent smoking might depend on timing, the quality of smoking-specific communication between parent and adolescent, the overall quality of the relationship between parent and adolescent and parental smoking behaviour.

In this present study, we formulated the following hypotheses. First, although past research showed inconsistent results we hypothesised that parental communication would be ineffective for both older and younger siblings. Second, we hypothesised that when the adolescent was smoking the smoking-specific communication between parents and adolescent would increase 1 year later. In other words, we assumed that smoking-specific communication between parents and adolescent was more a reaction on adolescent smoking than an action to prevent or discourage adolescents from smoking. Third, we hypothesised that the timing of communication was important, and that smoking-specific communication might only prevent adolescents who never had smoked from smoking, but would not affect the smoking behaviour of already smoking adolescents. Finally, we hypothesised that the quality of smoking-specific communication, the overall quality of the relationship between parents and adolescent, and parental smoking behaviour moderated the effects of smoking-specific communication on adolescent smoking.

#### Method

# **Participants**

The addresses of two-parent families with at least two adolescents (aged 13–16) were selected from the registers of 22 municipalities in the Netherlands. A letter was sent to all these families inviting them to participate in a longitudinal study 'Family and Health'; 885 families responded that they were willing to participate and gave their consent. These families were then telephoned to establish whether they fulfilled all the inclusion criteria, resulting in 765 families fulfilling the criteria. To ensure an equal distribution of the educational levels of the adolescents, and an equal number of all the possible sibling dyads (i.e. boy-boy, girl-boy, boy-girl and girl-girl), finally, a total of 428 families were selected to participate. These families were approached between November 2002 and April 2003.

A full-family design was used: each family consisted of a mother, father and two adolescents (for more details, see Harakeh et al., 2005). Families had to fulfil the following inclusion criteria to participate in the present study: the adolescents in the families were biologically related to each other and the mother and father were the biological parents of these adolescents; parents were married or living together during the project (two families had to be excluded from the third measurement because the parents divorced or were no longer living together) and the two adolescents participating in each family were neither

Table	1.	Demograp	hic	inf	ormation	of t	he	particip	ants	in	this	study.

	Older sibling (in %)	Younger sibling (in %)
Dutch origin	98.1	98.8
Male	52.8	47.7
Education level:		
Lower-level education	30.9	36.7
Middle-level education	29.3	35.5
Higher-level education	39.6	26.3
	Mother (in %)	Father (in %)
Dutch origin	97.4	96.1
Education level:		
Primary school only	2.1	1.4
Secondary school	31.4	17.9
Technical and vocational training	30.0	30.5
College	30.3	32.2
University	5.4	17.4
Job:		
No work	18.6	3
Work $> 33 \text{ h/w}$	5.7	91.4
Current Smoker	20.6	23.7

Note: Lower-level education (i.e. preparatory secondary school for technical and vocational training); middle-level education (i.e. preparatory secondary school for colleges below university level); and high-level education (i.e. preparatory secondary school for university).

twins, nor mentally or physically disabled. At baseline, the older siblings were aged  $14-17^1$  (M=15.22, SD=0.60), the younger siblings 13-15 (M=13.36, SD=0.50), the mothers 35-56 (M=43.82, SD=3.57) and the fathers 37-62 (M=46.18, SD=4.00). More demographic details of the participants in this study are depicted in Table 1.

Attrition of the participants was low, only 26 families left the study. A logistic regression analysis was used to test whether these families significantly differed from the families that had participated at all three waves on demographic variables (gender, age, education level) and adolescent smoking behaviour at Time 1. The adolescents of the 26 families that dropped out, compared to the 402 remaining families, did not differ on demographic variables but did so, on smoking behaviour. The older siblings that left the study were more likely to smoke compared to those participating in all three waves (p = 0.042). The younger siblings that left the study were more likely to smoke compared to those participating in all three waves (p = 0.018).

## **Procedure**

Interviewers visited all the families at home at baseline (Time 1) between November 2002 and April 2003 (T1; N=428), the first follow-up was 1 year later at Time 2 (T2; N=416), followed 1 year later by a second follow-up at Time 3 (T3; N=404). However, because at T3 two of the 404 families were divorced or no longer living together, these two families had to be excluded from the third measurement. Therefore, for the analysis at T3 the sample included 402 families. Attrition between the three waves was low. Most important reasons why families left the study were that they did not want to participate in it anymore,

or they divorced or moved. During the home visits, each family member filled in their questionnaire individually and separately in the presence of the interviewers. The questionnaire took about 90 minutes to complete. At each wave, the family (as a whole) received 30 euros and at the end of the study five cheques of 1000 euros were raffled between the 404 families who took part in all three waves.

#### Measures

We used the adolescent reports with regard to the frequency of smoking-specific communication, the quality of communication measures, and the scale assessing the overall quality of the relationship.

# Frequency of smoking-specific communication

This variable referred to how often in the past 12 months the mother and father talked with their adolescents about issues concerning smoking (Ennett et al., 2001). This scale was similar to the one used by Ennett and colleagues to assess smoking-specific communication and consisted of eight items. For example, the questionnaire version for adolescents was 'During the past 12 months, how many times did your mother talk to you about how to resist peer pressure to use tobacco?' and adolescents had to answer a similar question about their father. Response categories ranged from 1 'never' to 5 'very often'. Cronbach's alphas at T1, T2 and T3 for older and younger siblings reporting on their mother's and father's behaviour ranged from 0.87 to 0.91.

# Quality of smoking-specific communication

This concept represented the quality of communication about smoking between parent and adolescent, and indicated whether this took place in a constructive and respectful manner (Harakeh et al., 2005). We used the quality of smoking-specific communication assessed at T1. The scale consisted of six items on a five-point scale. For example, the questionnaire version for adolescents was 'My mother and I can talk easily with each other about our opinions of smoking' and 'If my mother and I talk with each other about smoking then I am being taken seriously by my mother'. Response categories ranged from 1 'completely untrue' to 5 'completely true'. Cronbach's alphas at T1 for older and younger siblings reporting on their mother and father ranged from 0.74 to 0.84.

#### Quality of parent-adolescent relationship

The quality of this relationship represented the affect dimension of parenting and was assessed by the Inventory of Parent and Peer Attachment (IPPA; Armsden & Greenberg, 1987). We used the information on the quality of the parent–adolescent relationship assessed at T1. The IPPA distinguishes three subscales: communication, trust and alienation. The response scales of these three subscales range from 1 ('never') to 6 ('always'). In the present study, we used the total scale of the IPPA assessing the general quality of the parent–adolescent relationship (see also Heiss, Berman, & Sperling, 1996). Cronbach's alphas for older and younger siblings reporting on their mother and father ranged from 0.83 to 0.87.

# Parent smoking

To assess parent smoking, parents were asked at T1 to report which stages of smoking applied to them (De Vries, Engels, Kremers, Wetzels, & Mudde, 2003). On an eight-point scale, responses ranged from 1 = 'I have never smoked, not even one puff' to 8 = 'I smoke at least once a day'. This same item was used to measure adolescent smoking, although adolescent smoking was measured with a nine-point scale. Because one of the nine responses was not appropriate for parents ('I try smoking once in a while'), parents could respond on an eight-point scale. To use parental smoking as a moderator, we recoded the responses into a binary variable to compare smoking and non-smoking parents in the multi-group analyses: 1 = 'not a current smoker' (this category included the response category 'I have never smoked not even one puff', 'I tried smoking, but I do not smoke anymore', 'I quit, I used to smoke less than once a week' and 'I quit after I had smoked for a period at least once a week') and 2 = 'current smoker' ('I smoke less than once a month', 'I do not smoke weekly but at least once a month', 'I do not smoke daily but at least once a week' and 'I smoke at least once a day').

#### Adolescent smoking

To assess adolescent smoking, adolescents were asked to report which stages of smoking applied to them at T1, T2 and T3 (De Vries et al., 2003). On a nine-point scale, responses ranged from 1= 'I have never smoked, not even one puff' to 9= 'I smoke at least once a day'. To get a more well-balanced distribution among these nine categories, we combined some successive categories and created four new categories: 1= 'never smoked' (this category included the response category 'I have never smoked not even one puff'), 2= 'stopped smoking' ('I tried smoking, but I do not smoke anymore', 'I quit, I used to smoke less than once a week' and 'I quit after I had smoked for a period at least once a week'), 3= 'smoked occasionally, less than weekly' ('I try smoking once in a while', 'I smoke less than once a month' and 'I do not smoke weekly but at least once a month'), 4= 'smoked at least once a week' ('I do not smoke daily but at least once a week' and 'I smoke at least once a day').

# Plan of analysis

Models were tested using Structural Equation Modelling (SEM). In our models, the frequency of the smoking-specific communication (T1, T2 and T3) is a latent variable, and adolescent smoking (at T1, T2 and T3) (measured by one item) a manifest variable. For the latter variable, it is not possible to model error variance. This is a slight disadvantage, because models including error variances lead to more accurate estimates of structural relations between latent variables. Because the smoking variables are categorically ordered (ordinal) variables, the software package MPLUS (Muthén & Muthén, 1998–2004) was used. The WLSMV-estimator (Weighed Least Square in combination with adjusted Mean and Variance chi-square statistics) is suited for dependent-ordinal variables. The categories of the dependent-ordinal variables are assumed to reflect an underlying normal distribution. Relations between variables are expressed in terms of polychoric correlations, and estimates of regression weights are obtained by using linear probit equations between the variables of the model. Standard chi-square tests are replaced by more robust chi-square variates (adjusted mean and variance chi-square statistics with adjusted df) to test model fit. This explains why the

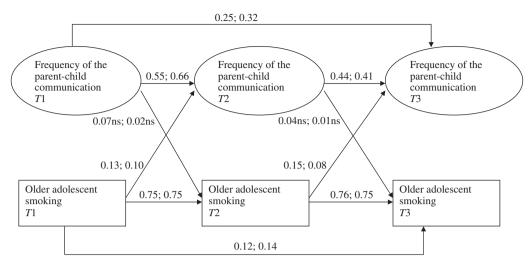


Figure 1. Model on frequency of smoking-specific communication and older adolescent smoking. Note: On each path two results are shown, the first number is of the model on the mother, and the second number is of the model on the father. The letters ns after the number and before the semicolon, indicate that this number is not significant.

number of degrees of freedom for identical models can vary across several groups. Our data showed structural missing values because of the families who dropped out of the study at Time 2 and Time 3, but also occasional missing values (varying from  $\approx 0-6$  missing values on an item). To make optimal use of the information in our data, we decided to use the missing option in MPLUS. In this case, the WLSMV-estimator utilised all available pair-wise information between variables.

To overcome the problem of insufficient statistical power (too many parameters in relation to the number of respondents), we decided to use parcels for the latent variable 'frequency of communication' by replacing the original eight items of a latent variable with two parcels of four items each (e.g. Bandalos & Finney, 2001; Harakeh, Scholte, Vermulst, De Vries, & Engels, 2006). The factor loadings in the four models for smoking (i.e. model reported by the younger sibling about the mother, model reported by the younger sibling about the father; two similar models for the older siblings) ranged from 0.79 to 1.02, and for the four models on smoking onset, the factor loadings ranged from 0.79 to 0.97, indicating adequate representation of latent variables by their respective indicators.

To examine the reciprocal relations between the frequency of smoking-specific parent–adolescent communication and adolescent smoking, cross-lagged panel analyses (Finkel, 1995) were carried out. In such models, error terms of corresponding indicators (parcels) between T1, T2 and T3 are allowed to correlate (Byrne, 1998, pp. 359–360). Significant cross-relations over time (T1-T2 and T2-T3) were indications for causal predominance: Did the frequency of communication have an impact on adolescent smoking behaviour, or did the adolescent smoking behaviour have an impact on the frequency of communication? The existence of two significant cross paths was an indication for a reciprocal relationship. We started with an initial model with specified paths as shown in Figures 1 and 2, including correlations between the two variables at T1 and correlations between the disturbance terms at T2 and T3.

Moderation effects of the quality of smoking-specific parent-adolescent communication, quality of parent-adolescent relationship, and parents' smoking on cross-lagged

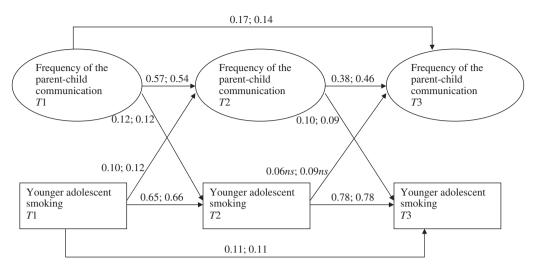


Figure 2. Model on frequency of smoking-specific communication and younger adolescent smoking. Note: On each path two results are shown, the first number is of the model on the mother, and the second number is of the model on the father. The letters ns after the number and before the semicolon, indicate that this number is not significant.

effects of the frequency of smoking-specific communication and adolescent smoking were tested with multi-group analyses (see for similar method, Poelen, Engels, Van Der Vorst, Scholte, & Vermulst, 2006). For the qualities of smoking-specific parent–adolescent communication and of parent–adolescent relationship, we dichotomised each of these variables into high and low scores using median split (Poelen et al., 2006). With regard to parents' smoking, two groups were formed: one group with the non-smokers and a second group with the smokers. We tested the model separately for fathers and mothers. Differences in structural paths between the two groups were tested with chi-square difference tests. Because differences between robust chi-square variates do not have a standard chi-square distribution, the robust chi-square values are first rescaled to standard chi-square values. This procedure is standard in MPLUS. Since testing the moderating influences for several parameters and many models will increase the risk of Type 1 errors, we decided to use p < 0.01 as significant criterion for moderating effects.

#### Results

## Descriptive statistics

Table 1 shows the smoking prevalence of mothers and fathers at T1. The majority of the current smokers were daily smokers; 17.6% of the mothers and 16.4% of the fathers. Table 2 gives the smoking prevalence of the adolescents at waves T1, T2 and T3; the majority of the adolescents had never smoked or had stopped smoking.

#### **Correlations**

Pearson correlations were reported between the frequency of communication and adolescent smoking at the three waves. Table 3 shows that the frequency of

Table 2. Smoking prevalence (in %) over the three waves for older and younger siblings.

	Time 1 ( <i>T</i> 1)		Time	2 (T2)	Time 3 ( <i>T</i> 3)	
	Older sibling	Younger sibling		Younger sibling	Older sibling	Younger sibling
Smoking status						
Never smoked	51.6	64.0	48.3	57.6	44.8	51.9
Quit smoking	31.2	28.2	31.3	27.2	31.2	28.2
Smoked occasionally but less than weekly	8.2	2.8	8.7	5.1	9.3	5.5
Smoked at least once a week	8.9	4.9	11.8	10.1	14.6	14.5

Note: Percentages for the older and younger siblings are shown separately.

Table 3. Correlations between the frequency of smoking-specific communication and adolescent smoking.

	Smoking T1	Smoking T2	Smoking T3
Older sibling			
Freq. com. mother $T1$	0.14**	0.15**	0.15**
Freq. com.mother T2	0.18***	0.22***	0.22***
Freq. com. mother T3	0.22***	0.27***	0.3***
Freq. com. father T1	0.12*	0.11*	0.11*
Freq. com father T2	0.16**	0.19***	0.19***
Freq. com. father T3	0.15**	0.18***	0.22***
Younger sibling			
Freq. com. mother $T1$	0.11*	0.16**	0.18***
Freq. com. mother T2	0.16**	0.15**	0.23***
Freq. com. mother T3	0.09	0.15**	0.24***
Freq. com. father T1	0.10*	0.18***	0.18***
Freq. com. father T2	0.16**	0.16**	0.23***
Freq. com. father T3	0.15**	0.16**	0.20***

Note: p < 0.05, p < 0.01, p < 0.001, p < 0.001

communication and adolescents smoking was cross-sectionally positively related at all three waves.

# Bi-directional associations between frequency of communication and adolescent smoking

To answer the first and second hypotheses of this study, we investigated the reciprocal associations between the frequency of smoking-specific parent—adolescent communication and adolescent's smoking. The following four models were tested for the total sample including both non-smokers and smokers: two for the older siblings and two for the younger siblings. The fit of all four models was satisfactory (Table 4). The cross-sectional correlations between the frequency of communication and adolescent smoking are given in Table 5. In the model, the stability paths of the frequency of communication over time and the stability paths of smoking over time were significant (Figures 1 and 2).

In the first hypothesis we predicted that parental communication would not be related to adolescent smoking, for both older and younger siblings. The frequency of

Table 4.	Fit measures	for eacl	ı of	the	eight	models;	four	models	for	smoking	and	four	models
for smoki	ng onset.												

		Sm	oking		Smoking onset					
	Older sibling report		Younge rep	r sibling ort	Older s	υ	Younger sibling report			
	Mother	Father	Mother	Father	Mother	Father	Mother	Father		
N df $\chi^2$ p CFI RMSEA	393 6 2.802 0.8332 1.000 0	428 6 7.626 0.2666 0.997 0.025	428 6 7.638 0.3655 0.999 0.015	428 6 3.404 0.7566 1.000 0	220 6 3.820 0.7009 1.000 0	220 6 3.340 0.7651 1.000 0	272 6 3.214 0.7814 1.000 0	272 6 2.957 0.8141 1.000 0		

Note: We left the missing option out in some models, which is the reason why the sample sizes in this table for one of the four models of smoking does not equal 428.

Table 5. Correlations in the models between the frequency of communication and smoking at T1 and between their disturbance terms at T2 and T3.

	Smo	king	Smoking onset		
	Mother	Father	Mother	Father	
Older sibling					
Freq. com. $T1 \times Smoking T1$	0.13	0.12	_	_	
Freq. com. $T2 \times \text{Smoking } T2$	0.05	0.08	0.13	0.10	
Freq. com. $T3 \times \text{Smoking } T3$	0.06	0.06	0.05	0.05	
Younger sibling					
Freq. com. $T1 \times Smoking T1$	0.12	0.11	_	_	
Freq. com. $T2 \times \text{Smoking } T2$	-0.00	0.00	-0.01	-0.02	
Freq. com. $T3 \times \text{Smoking } T3$	0.08	0.01	0.06	0.01	

Notes: The underlined correlations are not significant (p > 0.05). The model on smoking onset is smoking onset at T2; never smokers at T1 were selected and examined whether they smoked 1 year later (T2).

communication with the mother or father generally did not affect older adolescent sibling's smoking (Figure 1). The betas were non-significant and varied from 0.01 to 0.07 (Figure 1). In contrast, this pattern differed for the younger adolescent siblings (Figure 2). The frequency of communication with the mother or father influenced the younger adolescent siblings' smoking 1 year later. Younger siblings who frequently talked with their parents about smoking-related issues at T1 were more likely to smoke 1 year later at T2 (betas with respect to mother and father were both 0.12). Also, younger siblings who frequently talked with their mother or father at T2 were more likely to smoke at T3 (betas were 0.09 and 0.10). In sum, only the findings with respect to the older siblings, but not with respect to the younger sibling, were in line with our hypothesis.

In the second hypothesis we predicted that in the case where the adolescent is smoking the smoking-specific communication between parents and adolescent will increase 1 year later. The results depicted in Figures 1 and 2 showed that older and younger siblings' smoking at T1 did affect the frequency of communication with both parents 1 year later. The betas with respect to the frequency of communication with their mother and father were 0.13 and 0.10 for the older siblings (Figure 1), and 0.10 and 0.12 for the younger siblings (Figure 2), respectively. Older and younger siblings, who smoked at T1, were more likely to talk frequently with their mother or father about smoking-related issues 1 year later. Older siblings at T2 were also more likely to talk frequently with mothers (beta was 0.15) as well as with fathers (beta was 0.08) 1 year later. In contrast, younger sibling smoking at T2 did not influence the frequency of communication with their mothers (beta was 0.06) or their fathers (beta was 0.09) 1 year later at T3. In general, the findings showed to be consistent with our second hypothesis.

We also conducted additional analyses to examine whether the bidirectional associations between frequency of communication and adolescent smoking would differ for families with smoking and non-smoking siblings. However, a small number of younger and older siblings were current smokers, resulting in insufficient power to perform multi-group analyses. Instead, we reran the analyses in all four models, controlling for sibling smoking at T1, T2 and T3. Sibling smoking did not change these models. However, there was one exception. The model of older sibling reporting on father, controlling for younger sibling smoking, indicated that the effect of adolescent smoking at T2 on frequency of communication at T3 was not significant (p = 0.087). This is in contrast with the model excluding sibling smoking (p = 0.047), although the betas in both models were similar (0.08), only the p-value differed. In conclusion, these analyses implied that it did not matter whether the older or younger sibling was smoking within families; the bidirectional effects of frequency of communication and adolescent smoking remained similar.

# Timing of communication

In the third hypothesis, we predicted that smoking-specific communication only prevent adolescents from smoking who initially never had smoked. To test this hypothesis, we tested four models on smoking onset: two for older siblings and two for younger siblings. To construct models for smoking initiation, we selected adolescents who had never smoked at T1. To test the smoking onset models, we used the model as depicted in Figures 1 and 2 but excluded the smoking variable of adolescents at T1. The fit of the four models was satisfactory (Table 4). The cross-sectional correlations between the frequency of communication and adolescent smoking at T1 and their disturbance terms at T2 and T3 are given in Table 5. The stability paths of the frequency of communication over time and the stability paths of smoking over time were relatively strong.

The frequency of communication with mothers or fathers at T1 had no relations with smoking for the older (betas were 0.11 and 0.07, respectively) and younger (betas were 0.06 and 0.12, respectively) sibling's smoking onset 1 year later (Table 6). The frequency of communication with mothers or fathers at T2 affected younger sibling smoking at T3 (betas were 0.22 and 0.19, respectively), but not older sibling smoking at T3 (betas were -0.01 and -0.03). Thus, younger adolescent siblings who communicated frequently with their parents about smoking-related issues at T2 were more likely to smoke 1 year later at T3.

In general, older and younger sibling smoking at T2 had no relations with the frequency of communication with the mother or with the father (betas ranging from 0.07 to 0.09) at T3. However, there was one exception with regard to younger sibling's

Table 6. Standardized beta weights for the tested models on smoking onset.

	Smokin	g onset
	Mother	Father
Older sibling		
Freq. com. $T1$ – Freq. com. $T2$	0.53	0.66
Freq. com. $T2$ – Freq. com. $T3$	0.45	0.36
Freq. com. $T1 - Freq.$ com. $T3$	0.29	0.38
Smoking $T1$ – Smoking $T2$	_	_
Smoking T2 – Smoking T3	0.53	0.53
Smoking T1 – Smoking T3	_	_
Freq. com. T1 – Smoking T2	0.11	0.07
Freq. com. T2 – Smoking T3	$-\frac{0.11}{-}$	-0.03
Smoking $T1$ — Freq. com. $T2$		
Smoking $T2$ – Freq. com. $T3$	0.07	0.08
Younger sibling		
Freq. com. $T1$ – Freq. com. $T2$	0.58	0.52
Freq. com. $T2$ – Freq. com. $T3$	0.39	0.45
Freq. com. $T1$ – Freq. com. $T3$	0.22	0.13
Smoking T1 – Smoking T2	_	_
Smoking T2 – Smoking T3	0.55	0.55
Smoking T1 – Smoking T3	_	_
Freq. com. T1 – Smoking T2	$\frac{0.06}{0.22}$	0.12
Freq. com. T2 – Smoking T3	$\overline{0.22}$	0.19
Smoking $T1$ — Freq. com. $T2$	_	_
Smoking T2 – Freq. com. T3	0.13	0.07

Note: The underlined estimates are not significant (p > 0.05).

perspective on the mother. Younger siblings, who smoked at T2, were more likely to be involved more frequently in communication with their mothers on smoking-related issues at T3 (beta was 0.13).

### Quality of smoking-specific communication

In the fourth and final hypothesis of this study, we predicted that quality of communication, quality of parent–child relationship and parental smoking moderate the effects of smoking-specific communication on adolescent smoking. Multi-group analyses were conducted for the four models on the total sample to examine whether the quality of smoking-specific communication moderated the effects of the frequency of smoking-specific communication or adolescent smoking. Due to multiple testing, we employed p < 0.01 as significant criterion for moderating influences. The findings showed that the quality of communication generally did not moderate the relations between the frequency of communication and older and younger sibling's smoking. However, there were two exceptions with respect to the older and younger sibling's perspective on the mother. First, the quality of communication between mother and older sibling moderated the effects of older sibling smoking at T2 on the frequency of communication at T3 ( $\Delta \chi^2(1) = 8.46$ , p = 0.004). The path for the low-level group was positive (unstandardised b = 0.238, SE = 0.06) and for the high-level group the path was not significant (unstandardised b = 0.037, SE = 0.06). This finding showed that the older siblings, who

smoked at T2 and perceived the frequent discussions with mothers on smoking-related issues taking place in a constructive and respectful manner, will not communicate more frequently 1 year later, in contrast to smoking adolescents who perceived the discussions taking place in a less constructive and respectful manner. Second, the quality of smoking-specific communication between the mother and the younger sibling moderated the effects of the younger siblings smoking at T1 on the frequency of communication between the younger sibling and the mother at T2 ( $\Delta \chi^2(1) = 6.95$ , p = 0.008). The path for the low-level group was still positive and significant (unstandardised b = 0.302, SE = 0.10) and for the high-level group, this was not the case (unstandardised b = -0.166, SE = 0.12).

# Quality of parent—adolescent relationship

Multi-group analyses were also conducted for the four models on the total sample to see whether the quality of parent–adolescent relationship moderated the relations between the frequency of smoking-specific communication or older and younger siblings' smoking (p < 0.01). We found no indications for such moderating effects. In all four models, it was found that the quality of the relationship between mother or father and adolescent did not moderate the effects of the frequency of communication or of adolescent's smoking.

#### Parental smoking

Multi-group analyses were also conducted for the four models on the total sample to see whether the parental smoking moderated the relations between the frequency of smoking-specific communication and adolescent's smoking (p < 0.01). In general, the findings showed that maternal and paternal smoking does not moderate the effects of the frequency of communication or of older and younger sibling's smoking.

However, with respect to the older sibling there were two exceptions to the model concerning the older sibling's perspective on the mother. The first finding showed that mother's smoking moderated the effect of older sibling smoking at T1 on the frequency of communication at T2 ( $\Delta\chi^2(1)=11.48$ , p=0.0007). For the group with non-smoking mothers, this path was positive (unstandardised b=0.220, SE=0.06), while for the group with smoking mothers, this path was not significant (unstandardised b=-0.154, SE=0.13). This finding indicated that the older siblings who smoked at T1 and have a smoking mother will not communicate more frequently with their mother 1 year later, in contrast to smoking adolescents with non-smoking mothers. Further, mother's smoking moderated the effect of older sibling smoking at T2 on the frequency of communication at T3 ( $\Delta\chi^2(1)=7.65$ , p=0.0057). For the group with non-smoking mothers, this path was positive (unstandardised b=0.103, SE=0.05), while for the group with smoking mothers this path was positive and higher than the group with the non-smoking mothers (unstandardised b=0.165, SE=0.078).

#### Discussion

The aim of this present study was to obtain more insight into the reciprocal associations between smoking-specific communication in families on one hand and older and younger siblings smoking on the other. We first hypothesised that parental communication would

be ineffective for both older and younger siblings. The results were not completely in line with this hypothesis. Smoking-specific communication indeed did not affect older siblings smoking, while, in contrast, smoking-specific communication had counterproductive effects on younger siblings smoking at T2 as well as T3. The findings regarding the older siblings are in line with the few longitudinal studies on smoking-specific communications (Chassin et al., 2005: Ennett et al., 2001). These previous longitudinal studies did also not find any main effects of smoking-specific communication on adolescent smoking. These studies (including our study), however, investigated whether smoking-specific communication predicted adolescents smoking 1–2 years later, although it may be that this period was too long to find any effects, therefore, shorter time intervals are needed. The counterproductive effect of smoking-specific communication on adolescent smoking was, in our study, only found among the younger siblings within the family. The more frequently the parents talked with their younger sibling about smoking-related issues, the more likely the younger sibling smoked 1 year later. A possible explanation could be that, on average, parents talk more frequently with their younger adolescent about smoking-related topics than with their older adolescent. For example, a cross-sectional study of Harakeh and colleagues (2005) showed that fathers reported that they communicated more with the younger than with the older sibling. This difference in parental treatment between older and younger siblings could exist because perhaps parents are more protective towards their younger adolescent, especially, when the sibling(s) and friend(s) of the child is (are) already smoking. With regard to older siblings, perhaps parents think that their older adolescent is more responsible and does not smoke or maybe they respect the older adolescent's wish to be more independent and to be treated on an equal basis when it comes to more adult behaviour, such as smoking. Younger adolescents within the family may experience that they are treated differently than their older sibling and will react the opposite of their parents' expectations, which could be interpreted as an act of rebellion (Spijkerman, Van Den Eijnden, & Engels, 2005; Sulloway, 1995).

Our second hypothesis was that if the adolescent is smoking, the smoking-specific communication between parent and adolescent will increase 1 year later. The results of the present study are in line with this hypothesis. The findings showed that adolescent smoking affects the frequency of smoking-specific communication between parent and adolescent. If adolescents smoked, the likelihood increased that 1 year later they would have more smoking-related conversations with their parents. Previous cross-sectional and longitudinal studies did not examine the effect of adolescent smoking on smoking-specific communication. A possible explanation that adolescent smoking influences smoking-specific communication is that parents who know or suspect that their child is smoking may try to discourage him/her from smoking by talking about smoking-related topics more frequently. Future research is needed to gain more insight into who initiates the communication on smoking-related topics, how the other person reacts to it, and the barriers parents and adolescents have to overcome in communicating with each other on smoking-related topics. Observational studies could provide more information on the communication processes in 'real life'.

Our third hypothesis was that the timing of communication would be important, and that smoking-specific communication might only prevent adolescents who never had smoked from smoking, but did not affect the smoking behaviour of already smoking adolescents. Our results were not in line with this hypothesis. It was shown that parents who frequently talk with their adolescents about smoking-related issues were not effective in preventing their offspring from initiating smoking; in a positive sense one might argue

that at least this did not show a counterproductive effect. With regard to the effects of the frequency of smoking-specific communication on adolescents' smoking onset, the results showed a similar pattern for the older siblings and the younger siblings. The basic assumption that communication itself would prevent adolescents from starting to smoke was not supported by our study. Our findings were in line with other prospective studies (e.g. Chassin et al., 2005; Den Exter-Blokland et al., 2006; Ennett et al., 2001). However, some previous studies on smoking-specific communication did support the assumption that smoking-specific communication prevented adolescent smoking. The inconsistent findings in the literature may be due to studies having assessed smoking-specific communication in different ways; some only asked the adolescents how often parents talked with them about not smoking cigarettes, others assessed how often parents talked with adolescents more extensively by including several specific smoking topics (including our study), and yet, others assessed smoking-specific communication as parents' intentions to discuss reasons for not smoking with their children.

Finally, we hypothesised that certain factors (i.e. quality of the smoking-specific communication, overall quality of the relationship, parents' smoking) might moderate the effects of smoking-specific communication on adolescent smoking. The results were not in line with our hypothesis. Neither the quality of the smoking-specific communication between parent and adolescent, nor the overall quality of the relationship between parent and adolescent or parents' smoking moderated the associations between the frequency of smoking-specific communication and adolescent smoking. This underlines the robustness of our findings.

#### Limitations

Some limitations of this study should be mentioned. First, because we included only intact families in which the children were biologically related and the mother and father were married or living together, we cannot generalise these findings to single parent or remarried families, or to families with step-siblings or adoptive siblings. However, the families participating in our study were representative for the Dutch families with respect to socio-economic status, but did not represent the daily smokers among Dutch adolescents (i.e. fewer daily smokers in our study). Second, adolescents might have under-reported their actual smoking because they completed their questionnaire in the presence of their parents. To diminish this problem, interviewers were also present when the four family members completed the questionnaire, and family members were asked to complete the forms separately without discussing it with each other. In addition, self-administered questionnaires have been found as reliable and valid as other more objective methods, such as biochemical verification (e.g. Patrick, et al. 1994). Third, family members were asked by means of questionnaires how often they talked about smoking-related issues in the last 12 months; this might not be precise enough. Besides this, we lack information about the communication processes in childhood, which might be important as well, since some parents may have communicated about smoking with their children long before they reached adolescence. Fourth, in this present study it is more likely we are measuring birth-order effects rather than age-effects. This is because in our study design, the ages of the older and younger siblings overlap; older siblings were aged between 14 and 17 and the younger siblings were aged 13-15, respectively. However, age and birth-order are strongly related and to be able to disentangle the age and birth-order effects completely, other designs are necessary. Finally, the results of this study showed that parental smoking was a weak, often non-significant moderator. In our study, the non-smokers outnumbered the smokers among parents:  $\approx 80 \text{ versus } 20\%$ . This small group of smokers in our present study leads to low statistical power to test moderator effects.

# **Implications**

Prevention campaigns often recommend and encourage parents through television and newspapers to communicate with their adolescents about alcohol, tobacco and other drugs (Miller-Day, 2002). Our findings showed that such discussions did not prevent adolescents from smoking, and might even have counterproductive effects for younger siblings. This means that parents who undertake smoking-specific communication with their adolescents to prevent their children from smoking do not have the desired outcomes that parents intend and it applies, in particular, to the younger child of the family. However, this does not mean that parents are not able to undertake effective actions to prevent and discourage adolescents' from smoking. Empirical evidence indicated that parents who show affection, are warm and supportive towards their children, and/or communication with their adolescents in a respectful and constructive manner about smoking-related issues decrease adolescents' likelihood to smoke (e.g. Chassin et al., 1998; Foshee & Bauman, 1992; Harakeh et al., 2005). Also, adolescents who smoked were more likely to communicate with their parents about smoking-related issues, presumably because parents think that they might persuade their adolescent to quit smoking. So, adolescents' behaviour influences the actions parents undertake. Thus, before prevention programmes are developed to encourage parents to communicate about smoking-related issues as an effective strategy to prevent adolescents' from smoking, (observational) research is needed to elucidate the circumstances under which communication will be successful. This study showed that these circumstances did not contain: parents and adolescents talking in a constructive and respectful manner on smoking-related issues, a good relationship between adolescent and parent and adolescents having non-smoking parents. Also, this present study showed that the circumstances in which communication would be effective might differ for older and younger siblings in the family. To obtain a better understanding of the communication processes, we need to know how parents could effectively transmit their norms on risky behaviour, and empower their older and younger children to individually make responsible decisions regarding risky behaviour during childhood and adolescence (Miller-Day, 2002). We also need to gain more knowledge on the role of the (other) sibling in this process. Of course, siblings might communicate with each other or overhear conversations with parents and participate in conversations between parents and siblings. To understand the roles of siblings in the communication processes between parent and adolescent, future research is needed to collect information on these aspects of communication. In conclusion, encouraging parents to talk frequently with their adolescents about smoking-related issues through the media, prevention programmes or other sources may not be an appropriate message to broadcast.

#### Acknowledgement

This study was funded by a grant from the Netherlands Organization for Scientific Research (NWO).

#### Note

[1] Families with children between 13 and 16 years old were selected, but when collecting the data one adolescent had just reached the age of 17.

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