
The contribution of parent–child interactions to smoking experimentation in adolescence: implications for prevention

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Abstract

Because few prospective studies have examined the independent influence of mothers and fathers on smoking experimentation, we tested the association between a set of parent-specific, familial and peer interactions with smoking experimentation in early adolescence. Data come from two cohorts in the British Youth Panel Survey ($N = 1736$; mean age at baseline, 11.26; $SD = 0.65$), a study of children resident with members of the British Household Panel Survey. Baseline data showed 8.2% of participants had smoked which increased to 40.3% after a 3-year follow-up. Multivariate logistic regression models showed risk factors for the onset of experimentation included frequent time spent with peers ($P < 0.001$), maternal smoking ($P = 0.001$), female gender and older participant age ($P < 0.001$). Parent–child quarrels, mother–child conversations, family meal frequency and household income were not significantly associated with experimentation. Frequent father–child conversations, about things which mattered to children, were the only type of parent–child contact associated with a reduced risk of experimentation ($P < 0.001$), and a significant interaction suggested that maternal smoking increased the likelihood of girls but not boys experimentation ($P = 0.01$). This study suggests that familial risk and protective factors operate independently and that more attention should be paid to the role of fathers in smoking prevention.

Introduction

A socio-ecological perspective suggests that health behaviours emerge in a dynamic system of influences operating at multiple levels [1]. Application of this perspective has led to a shift in health promotion away from intensive face-to-face programs towards targeting causal mechanisms at the interpersonal, organizational and community level [2]. As the family environment has a potent influence on shaping health behaviours in children, it has been a common setting for prevention programmes [3, 4]. Numerous studies have shown that various aspects of the family environment, such as family connectedness, communication, cohesion, frequent family meals and parenting style are important predictors of adolescent health behaviours [5–8]. Of particular interest in these studies and family-based interventions is the frequency and quality of contact between parents and their children. These interactions allow parents to monitor their child's health, well-being, and prompt the adoption of a healthy lifestyle. Equally, parent–child interactions can also have a negative impact on child health; frequent contact with parents who drink alcohol to excess [9] or smoke [10, 11] is likely to increase the likelihood children adopt these behaviours.

As most individuals from the United States and United Kingdom first experiment with smoking during early-middle adolescence, [12, 13] and as early experimentation (i.e. <13 years of age) has been linked to a reduced likelihood of cessation

as an adult [14–16], familial influences on adolescent smoking have been well investigated. For example, prospective cohorts have demonstrated that parental smoking status [17], parenting style [18], parent–child conflict [19, 20], general rather than smoking-specific parent–child conversations [5, 21] and the degree of parental monitoring [22, 23] are all associated with the age at which individuals first experiment with smoking [24, 25]. However, these studies have combined parental interactions, such that the independent influence of mothers and fathers is not investigated. As mothers tend to spend more time with their children than fathers [26], it could be argued that they may have more of an influence in the shaping of health behaviours.

The importance of estimating the risk associated with each parent is most evident in the literature on the risks associated with parental smoking. Parental smoking has been linked to a higher incidence of smoking in young people in some studies [10, 11] but not others [27]. Some of these inconsistencies can be attributed methodological differences in way parent (e.g. current or life-time use [28]) and child smoking (e.g. experimentation or frequency of use [29]) is defined. Another explanation that emerges from studies which examine the influence of parents separately, is that more consistent associations are seen between child smoking with maternal than paternal smoking [30–32]. There is also some suggestion that child gender may moderate the effect of maternal and paternal smoking status [33]. Although these studies provide good evidence that mothers and fathers do not influence child smoking to the same degree, this approach of estimating the risk associated with each parent and child independently has not been applied to other parent–child interactions (e.g. the frequency of supportive conversations, or arguments).

Most studies investigating the influence of parent–child interactions on smoking have combined the influence of parents [10, 11, 27], not consistently considered the role of child gender [24, 33], and tended to focus on the frequency, rather than the child’s perception of conversations [26]. These studies have also neglected to examine the possibility that risk and protective factors operating in the family context could interact [21, 34]. For example,

regular conversations with a parent who smokes could conceivably increase or decrease adolescents risk of smoking experimentation. These gaps in the research literature represent substantive barriers to extending the evidence from prospective cohorts on protective factors for adolescent smoking experimentation (e.g. frequent family meals [6, 7]; regular parent–child conversations [5]) to interventions that recommend improving the quality and frequency of these interactions.

In this paper, we sought to address these gaps in the research literature using data from two cohorts participating in a nationally representative panel survey over a 3-year period in early adolescence. Our first aim is to investigate the independent influence of two types of parent–child interactions on smoking experimentation: the frequency of supportive parent–child conversations (defined by the child) and quarrels after accounting for variations in more established risk factors for smoking during adolescence (i.e. the frequency of family meals [6, 7], time spent with peers [22, 35], parental monitoring [22, 23]). Second, we investigate child gender as a moderator of (i) maternal and paternal smoking status, (ii) frequency of conversations with each parent, (iii) frequency of quarrels with each parent and (iv) meal frequency, on smoking experimentation. Finally, we examine whether the effect of family meals and parent–child conversations (hypothesized protective factors) on smoking experimentation is moderated by each parents smoking status (hypothesized risk factors). These aims were addressed with data from a longitudinal study that attempted to address some of the methodological shortcomings of past research (i.e. by using a prospective design and a large sample).

Methods

Study design and recruitment

Data come from two cohorts participating in the British Youth Panel Survey (BYPS; 1999–2002 and 2000–2003), an annual survey of children resident with participants of the British Household Panel Survey (BHPS).

A complete description of BHPS procedures and measures has previously been reported. Briefly, however, the BYPS was annual survey of individuals aged 16 years and over in a representative sample of private households in England, Wales and Scotland. At the inception of the study, members were selected via a two-stage, stratified clustered probability sample. At annual intervals, efforts were made to re-interview all original sample members. Children resident with BHPS participants were introduced to the study at age 11, then leave to join the adult BHPS when they reach age 16. This means that around 17–20% of participants join or leave the study at each wave, with a core group remaining for a maximum of 4 years [36].

Data from participants when introduced to the study were used as the first experimentation with smoking typically occurs in early adolescence [12, 13]. As an explicit aim of the study was to investigate parent-specific interactions and factors which predict the onset of experimentation, children who declared themselves to have already smoked, or were from a single-parent family, were excluded from analyses.

Procedure

Data in the BYPS were collected by adolescents listening to questions recorded on a personal stereo, away from parents and researchers involved in data collection and completing a questionnaire that only contained response categories for answers without the questions. This strategy was used so that children could control the pace of the interview and their confidentiality was preserved if another household member scanned their responses. No incentives were provided for participation. Informed consent was obtained from parents and children also gave their assent. The BYPS investigators complied, in full, with the Ethical Guidelines of the Social Research Association.

Participants

Across the two cohorts, of the 1736 (818 boys and 918 girls) participants who entered the study at age 11, the averaged attendance rate at age 12 was 1696

(97.6%), 1689 (97.3%) at 13 and 1699 (97.8%) at 14. Of these, 143 (8.2%) participants reported that they had already smoked by the time they entered the study and 30 (1.7%) were from a single parent family and were therefore excluded.

Of those remaining, 1555 (89.5% of those recruited at 11 years of age) provided annual data on smoking status (such that smoking experimentation over the 3-year interval could be assessed) and had complete data on all individual predictors. The average age of participants at entry was 11.26 (SD = 0.65) years of age. The characteristics of these children are summarized in Table I. Ninety-five percent of the sample was White (97.3%), 0.8% described themselves as mixed race and 1.9% as Asian. Participants came from diverse economic (monthly household income in £: Mean = 2858.16; SD = 2068.36; Median = 2504.63) backgrounds.

A comparison between those who were recruited to those in the analytical sample, showed that participants in the analytical sample ate less frequently with their family, had less frequent mother–child and father–child conversations and spent more time with their friends and out after 9 p.m. (all P 's < 0.05). However, participants who dropped out of the study and their parents were not more likely to report smoking (Supplementary data available at *Health Education Research* online).

Measures

Demographics

Two items recorded participants' gender and date of birth. Household income was calculated using parents' responses to the questions, 'On average, what was your weekly or monthly income from this job/business over the last 12 months?' and 'Was that weekly or monthly income?' (averaged across parents). These responses were linked to participants responses using the cross-wave personal identifier of mothers (IMPID) and fathers (IFPID) included in the BYPS dataset at each wave.

Smoking experimentation

Smoking experimentation was assessed with the question, 'Have you ever tried a cigarette, even if

it was only a single puff?’ Response categories were: yes (1) and no (0).

Frequency of supportive parent–child conversations

The frequency of father–child and mother–child conversations was assessed using two items. Two questions asked, ‘How often do you talk to your mother, about things that matter to you?’ and ‘How often do you talk to your father, about things that matter to you?’ Response options on these questions were a four-point scale ranging from hardly ever (1), less than once a week (2), more than once a week (3) and most days (4).

Frequency of parent–child quarrels

The frequency of parent–child quarrels was also assessed using two items. These questions asked, ‘Most children have occasional quarrels with their parents. How often do you quarrel with your mother?’ and ‘Most children have occasional quarrels with their parents. How often do you quarrel with your father?’ Response options on these questions were a four-point scale ranging from hardly ever (1), less than once a week (2), more than once a week (3) and most days (4).

Family meal frequency

Frequency of family meals was assessed with the item, ‘In the past 7 days, how many times have you eaten an evening meal together with your family?’ The four response options were: none (1), 1 or 2 (2), 3–5 (3) and 6 or 7 times (4).

Frequency of time spent with friends

Time spent with friends was assessed using one item, ‘Thinking back over the last 7 days, how many times have you gone out with friends?’, with a four-point response scale ranging from none (1), once or twice (2), 3–5 (3) to 6 or more times (4).

Parental monitoring

Parental monitoring was assessed using the item, ‘In the past month, how many times have you

stayed out after 9.00 p.m. at night without your parents knowing where you were?’, with four response categories ranging from never (1), 1 or 2 (2), 3–9 (3) to, 10 or more times (4).

Parental smoking status

Maternal and paternal smoking status was assessed using parents responses to two questions in the adult survey, ‘Do you smoke cigarettes at all nowadays?’, response categories were: No (0) and Yes (1) and another question to identify their sex ‘Please tick if you are male or female’.

Data analysis strategy

Two 3-year cohorts running from 1999 to 2002 and 2000 to 2003 were combined and all analyses are based on this combined cohort. Univariate and multivariate logistic regression models were produced for the risk of smoking experimentation (remained a non-smoker versus experimented with smoking) in the combined cohort over 3 years. Analysis was conducted in two steps.

First, the relationship between each of the putative baseline risk factor and the onset of smoking was investigated using individual logistic regression models to examine these relationships without complications arising from multicollinearity. These models examined the univariate relation between each risk and protective factor and the onset of smoking experimentation over a 3-year period. Categorical predictors were modelled using indicator contrasts with the lowest values acting as a reference category (e.g. family meals = none; paternal conversations = hardly ever). Odds ratio’s (ORs) and 95% confidence intervals (95% CIs) were reported for the largest comparison (e.g. family meals = none versus 6 or 7 times) and *P*-values were for the global effect of the variable reflecting the significance of comparisons to all possible categories.

Interactions between parental risk and protective factors with participant gender were also examined. Interactions terms for participant gender and maternal smoking status (repeated for fathers), the frequency of mother–child conversations (repeated for fathers) and mother–child quarrels (repeated

for fathers) were derived. Each parent-child variable was entered with gender and the relevant interaction term to examine whether each hypothesized risk and protective factor had a different effect in males and females. A number of hypothesized interactions between risk and protective factors were also modelled. Maternal smoking status with the frequency of mother-child conversations, the frequency of mother-child quarrels and family meal frequency (repeated for paternal smoking status with: father-child conversations, father-child quarrels and family meal frequency).

Second, the risk and protective factors that showed significant univariate relationships were included in a multivariate logistic regression to assess the unique effect of each predictor controlling for the effect of other predictors. Interactions that showed significant effects were also included in the multivariate model.

Results

A minority of participants 143 (8.2%) who reported smoking at baseline were excluded from analysis. The number of participants who had experimented with smoking increased over the course of the study: over 1 year 253 (14.6%), 2 years 473 (27.2%) and over 3 years 700 (40.3%) participants had smoked. The majority of parents of participants also reported that they were non-smokers. At baseline, 436 (13%) fathers and 418 (12.4%) mothers were smokers. One hundred and three (3.1%) participants had two parents who smoked.

Table I shows boys reported having more frequent conversations with fathers ($\chi^2 = 30.28$; $P < 0.001$), spending more time with friends ($\chi^2 = 14.46$; $P = 0.002$) and experienced lower levels of monitoring than girls ($\chi^2 = 33.68$; $P < 0.001$). Conversely, girls reported having more conversations with mothers than boys ($\chi^2 = 30.28$; $P < 0.001$) and no meaningful difference were found in the frequency of arguments with each parent or family meals. After stratifying the analysis by parental smoking status, no difference was found across boys and girls in the frequency of conversations (mothers, $\chi^2 = 2.53$; $P =$

0.47 and fathers, $\chi^2 = 3.91$; $P = 0.27$) or arguments (mothers, $\chi^2 = 1.74$; $P = 0.63$ and fathers, $\chi^2 = 1.22$; $P = 0.75$) between parents who smoked and did not smoke.

Univariate relationships with smoking experimentation

The ORs and CIs for the largest contrast and P -values for the global effect for each variable in these univariate models are reported in Table II.

As hypothesized, maternal (OR = 2.03; 95% CI = 1.40–2.94) and paternal smoking status (OR = 1.48; 95% CI = 1.10–2.01) were associated with a significantly increased risk of experimenting with smoking over the follow-up period. The frequency of quarrels between children with their mothers and fathers had a non-significant effect, as did the frequency of mother-child conversations. Frequent father-child conversations (OR = 0.56; 95% CI = 0.42–0.76) were associated with a reduced risk of smoking experimentation over 3 years. Low levels of parental monitoring (OR = 2.76; 95% CI = 1.35–5.64), frequent time spent with friends (OR = 2.70; 95% CI = 1.99–3.65), being older (OR = 1.51; 95% CI = 1.30–1.76) and female (OR = 0.58; 95% CI = 0.48–0.71) were also associated with an increased risk of smoking experimentation.

Most interactions were non-significant. The only significant interaction was between maternal smoking status and participant gender (OR = 0.39; 95% CI = 0.18–0.87), such that mothers who smoked increased the risk of girls, but not boys, experimentation.

Multivariate model for smoking experimentation

Table III shows the multivariate model that tested whether factors which demonstrated a significant univariate relationship with smoking experimentation remained significant once entered simultaneously.

Spending a lot of time with your friends (OR = 2.72; 95% CI = 1.93–3.82), having a mother who smoked (OR = 3.23; 95% CI = 1.61–6.45), being older (OR = 1.45; 95% CI = 1.22–1.71) or female (OR = 0.55; 95% CI = 0.44–0.68) were all

Table I. *Baselinerisk and protective factors of study participants*

Baseline risk and protective factors	Boys (<i>N</i> = 737)		Girls (<i>N</i> = 818)	
	<i>N</i>	%	<i>N</i>	%
Mother–child conversations				
Most days	250	33.9	357	43.6
More than once a week	156	21.2	174	21.3
Less than once a week	136	18.5	156	19.1
Hardly ever	195	26.4	131	16.0**
Father–child conversations				
Most days	159	21.6	132	16.1
More than once a week	139	18.9	125	15.3
Less than once a week	150	20.4	202	24.7
Hardly ever	289	39.2	359	43.9*
Mother–child arguments				
Most days	84	11.4	81	10.0
More than once a week	115	15.7	136	16.6
Less than once a week	170	23.0	185	22.6
Hardly ever	368	49.9	416	50.8
Father–child arguments				
Most days	49	6.6	46	5.7
More than once a week	84	11.4	87	10.6
Less than once a week	162	22.0	172	21.0
Hardly ever	442	60.0	513	62.7
Family meal frequency				
None	68	9.2	67	8.2
1–2	151	20.5	183	22.4
3–5	198	26.8	195	23.8
6–7	320	43.5	373	45.6
Time spent with friends				
None	197	26.7	216	26.4
1–2	208	28.2	336	41.1
3–5	170	23.1	152	18.6
6 or more	162	22.0	114	13.9**
Parental monitoring				
Never	623	84.5	765	93.5
1–2	87	11.8	37	4.5
3–9	18	2.4	12	1.5
10 or more	9	1.2	4	0.5**

P* < 0.01; *P* < 0.001

independently associated with an increased risk of smoking experimentation over 3 years. The main effects of gender and maternal smoking were qualified by a significant interaction between these variables (OR = 0.25; 95% CI = 0.10–0.70), such that having a mother who smoked was associated with an increased risk of experimenting with smoking over 3 years for girls, but not boys. Again, frequent conversations with fathers were the only protective factor (OR = 0.58; 95% CI = 0.42–0.79), with paternal

smoking and the degree of parental monitoring having a non-significant effect on experimentation.

Discussion

The primary aim of this study was to test whether parent-specific interactions, namely the frequency of supportive conversations and quarrels with each parent, were associated with the onset of smoking

Table II. *Univariate relationships between risk and protective factors with smoking experimentation*

Baseline risk and protective factors	Smoking experimentation—3 years		
	OR	95% CI	<i>P</i> *
Parental variables			
Mother–child arguments	1.22	0.84–1.80	0.10
Father–child arguments	1.34	0.96–1.87	0.24
Mother–child conversations	0.75	0.56–1.00	0.08
Father–child conversations	0.56	0.42–0.76	<0.001
Paternal smoking	1.48	1.10–2.01	0.01
Maternal smoking	2.03	1.40–2.94	<0.001
Interactions			
Maternal smoking × sex	0.39	0.18–0.87	0.02
Paternal smoking × sex	0.17	0.35–1.20	0.17
Mother–child conversations × sex	0.88	0.48–1.61	0.84
Father–child conversations × sex	0.74	0.40–1.37	0.30
Mother–child arguments × sex	0.63	0.36–1.11	0.39
Father–child arguments × sex	0.84	0.43–1.65	0.76
Family meals × maternal smoking	6.71	1.53–29.45	0.08
Family meals × paternal smoking	3.23	0.97–10.77	0.25
Mother–child conversations × maternal smoking	1.56	0.45–5.45	0.85
Father–child conversations × paternal smoking	2.11	0.64–6.98	0.41
Family and peer processes, demographic variables			
Family meal frequency	0.76	0.53–1.08	0.29
Time spent with friends	2.70	1.99–3.65	<0.001
Parental monitoring	2.76	1.35–5.64	0.002
Age	1.51	1.30–1.76	<0.001
Sex	0.58	0.48–0.71	<0.001
Household income	0.86	0.64–1.14	0.57

Largest contrast reported; lowest values used as reference categories (e.g. none: family meals; hardly ever: paternal communication). **P*-value for global effect of variable; significant relationships at *P* < 0.05 are in bold.

experimentation in early adolescence. Across a 3-year period, neither the frequency of quarrels between children and parents nor supportive mother–child conversations were significantly associated with smoking experimentation. However, the frequency of supportive father–child conversations was consistently associated with a reduced risk of experimentation across all models. These findings suggest that the family could be a significant agent in the shaping of health-related behaviours in adolescence.

The finding that frequent father–child conversations protected adolescents against experimenting was a unique result. It contrasts findings gathered in another prospective cohort that found frequent

smoking specific mother–child or father–child conversations did not impact upon the likelihood of smoking in middle adolescence [21]. However, this study focused on the frequency of smoking-specific conversations, not those which adolescents felt were supportive, as operationalized in the current study. The definition used in the current study is more analogous to measures of family connectiveness, which have been linked to reductions in smoking frequency [37]. However, this does not explain why father–child conversations in particular were influential, particularly when mothers were found in this and other studies to talk more frequently to their children than fathers [26]. Perhaps the type of

Table III. Multivariate relationships between risk and protective factors with smoking experimentation

Baseline risk and protective factors	Onset—3 years (<i>N</i> = 1555)		
	OR	95% CI	<i>P</i> *
Parental variables			
Paternal communication	0.58	0.42–0.79	0.001
Paternal smoking	1.29	0.81–2.06	0.29
Maternal smoking	3.23	1.61–6.45	0.001
Interactions			
Maternal smoking × Sex	0.25	0.10–0.71	0.01
Family and peer processes, demographic variables			
Parental monitoring	2.11	0.95–4.66	0.17
Time spent with friends	2.72	1.93–3.82	<0.001
Age	1.45	1.22–1.71	<0.001
Sex	0.55	0.44–0.68	<0.001

Largest contrast reported; reference categories (e.g. communication: hardly ever; non-smoking: monitoring: never; time spent with friends: non; sex: male).

**P*-value for global effect of variable; significant relationships at *P* < 0.05 are in bold

conversations mothers have with children are not conducive to conveying a protective effect. Consistent with this conjecture, quarrels with mothers were more frequent than fathers in the present study (quarrels on most days: mother: 10.4% and fathers: 6.3%). Taken together, these results suggest that interventions, which attempt to delay smoking experimentation, should attempt to enhance the quality and frequency of father–child conversations.

A secondary aim was to examine the possibility of interactions between parent–child interactions with participant gender in the prediction of smoking experimentation. The only significant interaction replicated results from a small number of prospective studies focusing on the frequency of smoking [30–32], suggested mothers have a more significant effect on girls than boys smoking behaviour. Stratifying the frequency of parent–child conversations by gender shows that although mothers communicated more frequently in the present study with their daughters than their sons (43.6% versus 33.9% on most days), they were not more likely to have been exposed to maternal smoking than boys. An alternative explanation is that girls are more likely to

model mothers' than fathers' behaviour [38], which may in turn increase the likelihood that girls adopt maternal than paternal behaviours.

A final aim was to investigate how interactions between risk and protective factors were associated with smoking experimentation. Within the multivariate model, there were no significant interactions among risk and protective factors, suggesting risk factors (e.g. parental smoking) and protective factors (e.g. supportive parent–child conversations), operate independently to affect the likelihood of experimentation. These findings suggest that interventions that encourage parents to engage in conversations about smoking with adolescents may be ineffective [26] because they only target the frequency of smoking specific rather than general emotional tone of father–child conversations, and ignore risk factors (e.g. parental smoking), which might conceivably increase the likelihood of experimentation. These findings lend support to advocates of universal interventions, which target both parents and children in an attempt to prevent adolescent smoking.

With respect to the more established risk factors we examined. There was no evidence that the frequency of family meals was associated with the risk of starting smoking. These findings contrast past prospective research examining the frequency of smoking [6, 7, 39], and research which has found girls gain more from attending meals than boys [40]. They suggest that the effect of family meals may be limited to slowing increases in the intensity of smoking rather than delaying onset. More importantly, they also suggest that the effect of family meals in past studies [6, 7, 40] may have been due to meal frequency acting as a proxy protective factor for omitted covariates, such as the amount time spent with friends which was significantly associated with smoking experimentation in the current study.

As expected, time spent with peers emerged as one of the most potent and consistent risk factors for smoking experimentation. These results are consistent with studies which have found that high levels of parental monitoring reduce the risk of smoking during adolescence by reducing association with smoking peers [22, 35]. As these results

take into account the effect of socialization with peers after 9 p.m., it suggests that this influence operates within a wider population of adolescents who socialize with peers before 9 p.m. It adds additional evidence to the assertion that peer-factors, in this case the frequency of socialization with peers, are potent risk factors for smoking experimentation in early adolescence [35, 41, 42], even after accounting for a host of parent–child and familial influences.

Although the use of a prospective design and a large sample increases the confidence that can be placed in the results, it is important that the limitations of this study are acknowledged. First, this study relies on self-report data such that responses on items of smoking experimentation may be underestimated due to reporting bias, which may have inflated the magnitude of effects. Second, a number of single-item measures were used, which meant that the reliability and validity of measurement could not be investigated. Third, the rates of parental smoking were lower than would be expected at the time of the study (UK adult smoking prevalence: 26% (2001) and 24% (2005) [43]; current study: 13%), however, these are global rates rather than rates for individual parents, which may be somewhat lower. Fourth, we did not include single parent families in the analysis as the previous research indicated factors such as parental monitoring vary across single and dual parent families [42]. We therefore suggest that these results are only be generalized to children in contact with both parents. Finally, the use of a longitudinal design does not preclude third-variable explanations, where a shared causal variable influences both the risk factor and promotes smoking experimentation.

Conclusions and implications for practice

The implication of these results for interventions is that programmes that increase father–child conversations about important issues might be a powerful way to help delay smoking experimentation. Interventions that have been successful in delaying smoking experimentation during adolescence, such as the Strengthening Families Programme [43], emphasize improving parenting skills, including

parent–child conversations. Some of the success of these programs may be attributed to changing the frequency of meaningful conversations between children and fathers. The current findings also suggest a universal prevention approach should be taken to encourage parental cessation, to in turn reduce the likelihood that children adopt this behaviour. This would have the dual public health benefits of promoting cessation across parent and adolescent populations and may increase the impact of health promotion messages provided by parents to children.

In conclusion, these results provide support for the assertion that the frequency of supportive father–child conversations, time spent with peers and maternal smoking are significantly associated with the risk of smoking experimentation in early adolescence. However, our findings failed to support claims that the frequency of family meals, parent–child quarrels and mother–child conversations is associated with smoking experimentation. These findings lend support to those who advocate a universal approach to smoking prevention in adolescents and highlight the neglected role of fathers in influencing the adoption of health behaviours in adolescence.

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Conflict of interest statement

None declared.

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