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ORIGINAL ARTICLE

How home-smoking habits affect children: a cross-sectional study using urinary cotinine measurement in Italy

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Abstract
Objectives To assess the impact of different home-smoking rules and smoking habits of cohabitant on environmental tobacco smoke (ETS) exposure of children.
Methods Information about 396 Italian children (5–11 years old) and cohabitants' smoking habits was collected by a questionnaire. Exposure assessment was performed by determination of urinary cotinine (u-cotinine).
Results Median u-cotinine concentrations in children significantly increased in a similar fashion as theoretical ETS exposure increase: cohabitants do not smoke (1.79 μg/g creatinine), cohabitant(s) smoker(s) never smoke at home (2.84), smoke at home only when children are out (3.90), and smoke at home even if children are in (6.02). Median u-cotinine levels of exposed children were associated to the strength of cohabitant's smoking behaviours when smoker(s) consume daily a high number of cigarettes ($\geq$ 20) respect to light consumption (1–9) (4.52 and 3.24 μg/g creatinine).
Conclusions The magnitude of ETS exposure in children is correlated with smoking habits and home-smoking precautions adopted by their cohabitants. Educational interventions on parents are essential to increase their awareness about ETS exposure and to teach correct behaviours to protect health of kids, especially in household environment.

Keywords Children · Passive-smoking exposure · Secondhand smoke · Thirdhand smoke · Urinary cotinine

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Introduction

The well-known health problems associated with passive smoking, or environmental tobacco smoke (ETS) exposure (IARC 2004), have led numerous countries, including Italy, to introduce restrictions or complete bans on smoking in public areas. The Italian government established an official ban on smoking in any indoor public place on January 10, 2005.

However, this type of ban does not guarantee full protection from ETS exposure for non-smokers who live with smokers. This issue is of particular concern when the non-smoker is a child for two reasons: (1) in any community, children are the most susceptible population to the harmful health effects caused by ETS exposure (Adgent 2006; Asomaning et al. 2008; Cheraghi and Salvi 2009; Muller 2007), and (2) the greatest proportion of children's ETS exposure occurs in the household environment, because that is where they spend most of their time (McNabola and Gill 2009).

Some researchers have hypothesised that bans on smoking in public places could adversely affect children' health by shifting smoking into the domestic environment. Contrary to this assertion, Hyland et al. (2008) showed that at-home-smoking habits were similar in Ireland, which had introduced a smoking ban, and in the United Kingdom, which did not have such a ban at the time the research was conducted.

In addition, several surveys conducted in the United Kingdom (O'Dowd 2005), Canada, the United States, and Australia (Borland et al. 2006) have shown an increase in the prevalence of smoke-free homes in the recent years. The authors of these studies observed that making public places smoke-free seems to encourage smokers to also make their homes smoke-free or to at least adopt protective behaviours towards non-smoking adults or children, such as smoking at home only when non-smoking cohabitants are not home or are in separate rooms.

Further studies have been performed to evaluate the impacts of maintaining a smoke-free home and of adopting other smoking precautions in the household on the ETS exposure of children by measuring salivary and/or urinary cotinine (u-cotinine). Cotinine is the main metabolite of nicotine and is a proven biomarker for assessing passive-smoking exposure (Benowitz 1996; Haufroid and Lison 1998; Keskinoglu et al. 2007). The results of these studies indicated that, for those who live with smokers, having a smoke-free home or a home where other protective measures are taken offers appreciable, but not complete, protection against passive smoking (Jarvis et al. 2009; Johansson et al. 2004).

These findings may be associated with the newly defined issue of "thirdhand" smoke (THS), which is the residue from tobacco smoke that persists on the clothing and hair of smokers, on environmental surfaces, and in dust long after a cigarette has been extinguished (Winickoff et al. 2009). Thirdhand smoke contains many different chemicals that are re-emitted as a gas, either directly or as a result of reacting with oxidants or other compounds to form secondary contaminants, some of which are carcinogenic or toxic to humans (Burton 2011; Destaillats et al. 2006; Matt et al. 2011; Petrick et al. 2011; Sleiman et al. 2010).

The term "thirdhand smoke" is derived from "second hand smoke" (SHS), which is "the combination of smoke emitted from the burning end of a cigarette or other tobacco products and smoke exhaled by the smoker" (WHO 2007). Passive smoking is the combination of SHS and THS exposure (Protano and Vitali 2011).

To the best of our knowledge, no data exist regarding on the impact of policies adopted by Italian smokers for smoking at home on children's exposure to SHS and THS.

The present study was conducted with a group of Italian children (5–11 years old) using u-cotinine determination in order to:

- assess how the smoking habits of cohabitants predict ETS exposure levels;
- quantify the effectiveness of home-smoking policies adopted by smokers with respect to ETS exposure;
- identify the possible individual contributions of SHS and THS to overall ETS exposure.

Methods

Study population and design

The study population consisted of all of the students in three primary-school districts located, respectively in northern, central, and southern area of Latium region (central Italy), comprising a total of 665 children aged 5–11 years.

All of the students and their parents received information about the research goals and plan and were invited to take part in the cross-sectional study, which was conducted on Wednesdays (a typical weekday) during the winter season of the academic years 2007–08 and 2008–09.

Information about cohabitants' smoking habits and precautions taken by at-home smokers as well as detailed information about the sociodemographic characteristics of the children and their families, the children's Wednesday activities, and household characteristics was collected using a self-administered questionnaire, previously validated, filled out by each child's parents.

Each participant's level of ETS exposure was estimated using an analytical determination of the cotinine level in a urine sample collected at the end of the sampling day. The urinary sample was taken at the last time of the day each participant urinated just before going to sleep in polypropylene bottle; then, the sample was immediately stored in the refrigerator at 4°C. The next morning, the sample was placed into a polystyrene cooler containing an ice pack and was delivered to the research team.

Covariates and ETS exposure data gathered by questionnaire

The answers to the first questions, which were about the gender of the child and the age (as defined by the primary-school grade they were attending), were classified as 0 = male and 1 = female and as 0 = first, second, or third grade (the younger group) and 1 = fourth or fifth grade (the older group). The sizes of the children's homes (expressed in cubic metres) were gathered using an open question.

The presence of cohabitant smokers was assessed with the question, "Are there smokers living with the child?" The possible responses were "Yes" and "No". If the response was "Yes", the child was considered to be exposed to ETS, and the respondent was invited to answer some other questions:

"How many cohabitant smokers live with the child?" (An open numeric answer, which was categorised in the analysis as 0 = 1 cohabitant smoker and 1 = more than 1 cohabitant smoker);

''Do(es) the cohabitant smoker(s) smoke inside the home in which the child lives?'' (Yes or no);

''Do cohabitants smoke inside the home when the child is present?'' (Yes or no).

The formulation of these questions was non-specific (avoiding terms such as "precautions" or "preventive measures") to encourage responses that would be as honest as possible. We classified the responses as 0 = no and 1 = yes.

The entire sample was divided into four groups on the basis of cohabitant smokers' behaviours and home-smoking rules:

- Children not living with smoker(s);
- Children living with smoker(s), with a total home-smoking-restriction (cohabitant smokers do not smoke at home);
- Children living with smoker(s), with a partial home-smoking-restriction (cohabitants smoke inside the home only if the child is out);
- Children living with smoker(s), with no home-smoking-restriction (cohabitants smoke inside the home even if the child is present).

Finally, the respondents were asked two questions about the average cigarettes consumption of each cohabitant smoker:

"On average, how many cigarettes are smoked by the cohabitant smoker(s) in the course of a weekday?" (Open numeric answer);

"On average, how many cigarettes are smoked by the cohabitant smoker(s) in the course of a weekday inside the home?" (Open numeric answer).

The responses to each of these questions were analysed as a single continuous variable; i.e., the smokers were added together for each question, but the questions were analysed separately (if there was more than one cohabitant smoker, the numbers of cigarettes consumed by all of the smokers were added together). In our evaluation of how the intensity of cohabitants' smoking habits affected the children's ETS exposure, the combined total daily consumption of cigarettes was categorised as:

- 0–9 cigarettes consumed daily by the cohabitant smokers: light consumption;
- 10–19 cigarettes: moderate-consumption;
- ≥20 cigarettes: heavy-consumption.

ETS exposure level as measured by u-cotinine

Urinary cotinine and urinary creatinine (u-creatinine) in the urine samples were measured using the procedures outlined in previous publications (Manini et al. 2008; Protano et al. 2010); in brief, about 2 mL of spot urine sample was partitioned into plastic tubes for u-cotinine and u-creatinine determinations. All samples were coded and then frozen at −20°C until analysis. The samples were analysed within 30 days from sampling.

Urinary cotinine was determined by isotopic dilution liquid chromatography tandem mass spectrometry (LC–MS–MS). Before analyses, urine samples were added with the internal standard (cotinine-d_3) and centrifuged at 3,000 g for 10 min. Chromatography was performed on an Atlantis®dC_{18} column (100 × 2.0-mm i.d., 3 μm; Waters, Milford, MA, USA) using variable proportions of 10 mM aqueous formic acid (pH 3.75) and methanol. Elution program: 12% methanol, hold for 12 min; from 12 to 70% methanol in 2.5 min (linear gradient); 70% methanol, hold for 1 min. The flow-rate was 0.2 ml/min and the injection volume 30 μl. Analytes were ionised in positive-ion mode and the transitions chosen for selected reaction monitoring detection of cotinine and its internal standard were *m/z* 177 → 80 and *m/z* 180 → 101, respectively. The limit of detection was 0.2 μg/l (20 μl injected), the coefficient of variation of the method (expressed as %CV) was below 2% for all intra- and inter-day determinations.

Urinary creatinine was measured by the method of Jaffe (Henry 1974). Urinary cotinine concentrations were expressed in micrograms/gram of creatinine to adjust for urine dilution.

Statistical analyses

The statistical analyses were conducted using the SPSS software package (Version 14.0 for Windows, Chicago, IL).

As the u-cotinine results were not normally distributed, analyses were conducted using non-parametric techniques. Mann–Whitney tests were used to assess differences in the concentrations of u-cotinine between the following groups:

- children not living with smoker(s) and children living with smoker(s) groups;
- children living with one smoker and children living with more than one smoker groups.

Kruskal–Wallis test was used to explore differences in the u-cotinine levels for children living with smoker(s), on the basis of the different smoking rules at home and the daily-cigarette-consumption of cohabitant smoker(s).

Even, since the total number of cigarettes consumed in a day and at home was not normally distributed, Kruskal–Wallis test was used to examine differences in the amount of the total number of cigarettes consumed in a day between total home-smoking-restriction, partial home-smoking-restriction, and no home-smoking-restriction groups. Mann–Whitney test was used to assess the differences in the mean total numbers of cigarettes consumed at

home between the partial home-smoking-restriction and the no home-smoking restriction groups.

In all, two forward multiple-linear-regression analyses were conducted to estimate the independent effects of cohabitant smokers' behaviours on the children's u-cotinine excretion. The first model was used to test the independent effects of home-smoking-restriction-related strategies adopted by cohabitant smokers (complete, partial or no restriction) on u-cotinine excretion, taking the children not living with smoker(s) group as the reference group. The second model (involving only the children living with smokers) was used to examine the contributions to ETS exposure of home-smoking rules (taking the total home-smoking-restriction group as the reference group), number of cohabitant smokers, and intensity of cohabitant smokers' smoking habits (taking the light daily-cigarette-consumption group as the reference group), as measured by the u-cotinine excretion.

The two-tailed significance threshold chosen for all of the statistical tests was $p \leq 0.05$. Linear-regression analyses were conducted using a significance level of 0.05 for entry and a level of 0.10 for removal from the models. The goodness of fit of the models was assessed using adjusted R^2.

Results

In total, out of 665 children 501 took part in the research, which constituted a response rate of 75%. However, 46 urine samples were rejected because of unsatisfactory sealing of sample containers; therefore, analytical determinations of u-cotinine and u-creatinine were performed for 455 samples.

In addition, we excluded 59 children who had at least one parent who was not Italian from the data analysis to avoid interference from well-known ethnic differences in the metabolism and excretion of u-cotinine (Benowitz et al. 1999; Pérez-Stable et al. 1998). In the end, the analysis was conducted using the data on 396 children.

The descriptive characteristics of the study subjects are given in Table 1. The sample was well-balanced with respect to gender and age. Responses to the question regarding the children's activities on the sampling day revealed that they spent the greatest proportion of their time in indoor environments (school, home, and other indoor settings).

The percentage of children who were exposed to ETS was similar to the percentage of unexposed children (45.1% vs. 54.9%). Approximately two-thirds of the children had cohabitant smokers who usually smoked inside the home. The mean overall cigarette-consumption level for cohabitant smokers was 16.6 per day, of which 34%, on average, was smoked inside the home.

Table 1 Basic characteristics of the study population (Latium region, Italy; winter season of the academic years 2007–08 and 2008–09)

Characteristic	*N*	Value
Gender (%)		
Male	197	52.3
Female	180	47.7
Grade in primary school (%)		
First, second or third	212	55.9
Fourth or fifth	167	44.1
Time spent in indoor vs. outdoor environments on the sampling day prior to the time of urine collection (Min; mean ± SD)		
At school (indoor environment)	377	444.7 ± 74.2
Home and other indoor environments	377	156.1 ± 147.1
Outdoor environments	377	29.5 ± 52.1
Home size (m^3; mean ± SD)	340	312.4 ± 181.5
Children ETS-exposure status (%)		
Living with smoker(s)	172	45.1
Not living with smoker(s)	209	54.9
At-home-smoking rules of cohabitant(s)[a] (%)		
Total home-smoking-restriction	58	33.7
Partial home-smoking-restriction	48	27.9
No home-smoking-restriction	66	38.4
Number of cohabitant smokers[a] (%)		
1	107	63.3
>1	62	36.7
Number of cigarettes smoked in a day by cohabitants[a] (Mean ± SD)		
Overall	163	16.6 ± 12.2
At home	108	8.4 ± 7.1
Daily-cigarette-consumption[a] (%)		
Light (0–9 cigarettes)	49	30.1
Moderate (10–19 cigarettes)	46	28.2
Heavy (≥20 cigarettes)	68	41.7

ETS environmental tobacco smoke

[a] Only includes responses for children exposed to ETS

A summary of the statistics on u-cotinine levels for all of the children combined and for each of the four ETS-exposure-status groups is given in Table 2. The median u-cotinine concentration value for the whole sample was 2.59 μg/g creatinine (interquartile (IQ) range = 1.51–4.23), whereas the median concentrations for the children not living with smoker(s) and those living with smoker(s) taken separately were 1.79 and 3.90 μg/g creatinine, respectively ($p < 0.001$). Urinary cotinine median levels increased significantly in a similar pattern as the levels of ETS exposure revealed by the questionnaire increased: children not living with smoker(s) <<children living with smoker(s) total home-smoking-restriction <<partial home-smoking-restriction <<no home-smoking-restriction.

The relationship between differences in the mean numbers of cigarettes consumed in a day and home-

Table 2 Summary of statistics on urinary cotinine (u-cotinine) concentrations (expressed as μg/g creatinine) for all participants and for subgroups stratified according to environmental tobacco smoke (ETS) exposure and the smoking habits of their cohabitants (Latium region, Italy; winter season of the academic years 2007–08 and 2008–09)

	N	Missing values	Arithmetic mean	95% CI	Median	IQ range	*p* Value
All children	396		4.37		2.59	1.51–4.23	
ETS-unexposed	209	15	2.40	2.14–2.66	1.79	1.30–2.88	<0.001[a]
ETS-exposed	172		6.65	5.40–7.89	3.90	2.22–7.07	
Children living with smoker(s)	172						
No. of cohabitant smokers							
1	107	0	5.34	4.01–6.64	3.51	2.01–5.70	0.008[a]
>1	62		8.69	6.11–11.19	5.05	2.63–9.71	
1. Total home-smoking-restriction	58	3	3.34	2.60–4.08	2.84	1.56–3.86	<0.001[b]
2. Partial home-smoking-restriction	48		5.49	4.29–6.70	3.90	2.66–6.41	
3. No home-smoking-restriction	66		10.40	7.54–13.26	6.02	2.88–12.30	
Daily-cigarette-consumption of cohabitant smoker(s)							
1. Light (0–9)	49	9	5.18	3.51–6.86	3.24	1.84–5.39	0.022[b]
2. Moderate (10–19)	46		5.05	3.35–6.74	3.73	2.23–5.71	
3. Heavy (≥20)	68		8.76	6.01–11.31	4.52	2.64–9.03	

[a] Mann–Whitney test

[b] Kruskal–Wallis test

smoking rules was also examined. We found that the median total number of cigarettes consumed in a day by cohabitant smokers who did not smoke at home at all, by cohabitant smokers who smoked at home only when the child was not at home, and by cohabitants who smoked at home even when the child was there were 10.0, 15.0, and 17.5 respectively ($p = 0.034$). Every day, cohabitant smokers who smoked at home only when the child was out consumed, on average, 8.0 cigarettes at home, whereas cohabitants who smoked at home even when the child was there consumed, on average, 8.9 cigarettes at home ($p = 0.768$).

The results of the univariate analyses were confirmed by the first linear-regression model (Table 3); the various types of home-smoking behaviour were associated with significant increases in the children's u-cotinine levels in comparison with children not living with smoker(s).

In the second multivariate-regression model (also shown in Table 3), the significant predictors of higher levels of u-cotinine excretion were having partial- ($\beta = 1.797$; $p = 0.002$) or no home-smoking-restriction ($\beta = 2.425$; $p < 0.001$) (in comparison with having a total home-smoking-restriction) and heavy daily-cigarette-consumption by cohabitants ($\beta = 1.452$; $p < 0.010$). Age, gender, home size and number of cohabitant smokers did not have significant effects on u-cotinine excretion.

The presented models explained a percentage of the variability in u-cotinine levels equal to 31 and 21% (first and second model, respectively).

Discussion

Principal findings and synthesis with prior research

In the present study, we found that only one-third of children living with smokers had a total restriction on smoking in the home is similar to the findings of studies conducted in other countries (Borland et al. 2006).

The second relevant result of this study relates to the impact of different home-smoking rules on the nicotine uptake of children. The impact of at-home-smoking practices on children's ETS exposure is highlighted by the significant and progressive increases in u-cotinine levels from children not living with smoker(s) to children living with smoker(s) who do not smoke at home to children living with smoker(s) who only smoke at home when the child is not there, and finally to children living with smoker(s) who smoke at home even if the child is in.

In addition, in comparing the three groups of children who live with smokers, we found that the ETS exposure was directly related to the home-smoking rules that parents reported in their responses to the questionnaires. The lowest levels of exposure were found among children living in domestic environments where there was complete smoking-restriction, children whose cohabitants observed partial smoking-restriction evidenced median levels of exposure, and the highest levels of exposure were found among children living in homes without any smoking-restriction. This finding is in agreement with previous

Table 3 Differences in urinary cotinine (u-cotinine) levels (ln u-cotinine expressed as μg/g creatinine) for children whose cohabitants have different at-home-smoking habits in comparison with children whose cohabitants do not smoke group (Model 1) and for different at-home-smoking habits among those children who live with smoker(s) (Model 2) (Latium region, Italy; winter season of the academic years 2007–08 and 2008–09)

Independent variable	B (regression coefficient)[a]	95% CI[a]	*t* Statistic	*p*
MODEL 1[b]				
Children not living with smoker(s) (reference group)	1			
Children living with smoker(s) with total home-smoking-restriction	1.259	1.003–1.587	1.946	0.050
Children not living with smoker(s) with partial home-smoking-restriction	2.375	1.842–3.059	6.716	<0.001
Children not living with smoker(s) with no home-smoking-restriction	3.307	2.672–4.092	11.045	<0.001
MODEL 2[c]				
Home-smoking rules				
Total home-smoking-restriction (reference group)	1			
Partial home-smoking-restriction	1.797	1.251–2.581	3.200	0.002
No home-smoking restriction	2.425	1.747–3.367	5.340	<0.001
Daily-cigarette consumption				
Heavy (≥ 20 cigarettes)	1.452	1.093–1.929	2.601	0.010

[a] Values were converted back to the original state by using the anti-log, EXP()

[b] Final forward linear-regression model with the participants' ages, genders, home sizes and the home-smoking habits of their cohabitants entered in step 1; constant = 1.891; final adjusted *R* equal to 0.307

[c] Final forward linear-regression model with the participants' ages, genders, home sizes, the home-smoking habits of their cohabitants, the number of cohabitant smokers, and the cohabitants' daily-cigarette-consumption entered in step 1; constant = 2.065; final adjusted *R* equal to 0.218

studies (Akhtar et al. 2009; Matt et al. 2004) and highlights two critical realities:

- The domestic environment is an important source of ETS exposure, even for Italian children.
- Smoking at home only when children are not there or smoking only outside the home gives smokers a false perception that they are fully protecting the children's health.

This last point is supported by evidence that children can be exposed not only to SHS but to THS as well. Thirdhand smoke is a major public health concern because it highlights the impossibility to maintaining a safe level of exposure to tobacco smoke and also because nicotine residues in the domestic environment can react with ambient nitrous acid to form new tobacco-specific, carcinogenic nitrosamines (Sleiman et al. 2010).

Finally, we found that the u-cotinine levels of children living with smoker(s) increase in direct proportion to the intensity of the smoking habits of the cohabitant smokers; this finding is especially significant among children who live with smokers who consume a high number of cigarettes daily (≥20). This significant relationship shows that heavy smokers, in addition to risking adverse effects on their own health, are endangering people living in the same environment.

The possible contributions of SHS and THS to children's ETS exposure levels

The differences in u-cotinine levels that we found among the four groups of participants described above are presumably attributable to a combination of SHS and THS, which likely contribute to total ETS exposure in variable proportions, depending on the habits of and the precautions adopted by cohabitant smokers.

However, it should be noted that a small amount of cotinine is always present in human bodily fluids because of the consumption of foodstuffs containing nicotine (e.g., potatoes, tomatoes, eggplant, or beverages) (Domino et al. 1993). The exact quantity of u-cotinine derived from dietary nicotine is not well-defined and very difficult to evaluate: a range from 0.6 to 6.2 μg/L of possible values for urinary cotinine concentrations was calculated based on estimated average and maximal consumption of food and beverages containing nicotine and cotinine (Davis et al. 1991). These results are in line with u-cotinine levels we found in the group of children not living with smoker(s).

Possible sources of nicotine for the study participants are listed in Table 4. Taking nicotine ingested in food and resulting from occasional ETS exposure unrelated to cohabitant smokers as the sources of the base amount of u-cotinine excreted by both children not living and children

Table 4 The possible contributions of secondhand (SHS) and thirdhand smoke (THS) to children's urinary cotinine levels (Latium region, Italy; winter season of the academic years 2007–08 and 2008–09)

Possible contributors to nicotine uptake						
Group	Dietary intake of nicotine	Occasional passive smoking through exposure to non-cohabiting smokers	SHS from cohabitants occurring outside the home	THS from contaminated hair and clothing	THS from household surfaces and dust	SHS from cohabitants smoking at home
Children not living with smoker(s)	+	+				
Children living with smoker(s)						
Total home-smoking-restriction	+	+	+	+		
Partial home-smoking-restriction	+	+	+	+	+	
No home-smoking-restriction	+	+	+	+	+	+

living with smoker(s), we can assume that the nicotine intake of the latter group is also affected by SHS from cohabitants smoking outside the home, THS from contaminated hair and clothing and from household surfaces and dust, and SHS from cohabitants smoking at home. Which of these sources is relevant depends on the smoking policy adopted in the participants' homes.

Strengths and weaknesses of the study

To the best of our knowledge, the present study is the first to examine the impact of the home-smoking rules of cohabitant smokers on a large sample of Italian children, using a proven biomarker of exposure to tobacco smoke such as u-cotinine as an objective parameter.

There are several methodological limitations that should be considered in interpreting the findings of this study.

First, urine samples were collected only once from each child (at the end of the sampling day), so possible changes in u-cotinine excretion over time could not be examined. However, previous studies have indicated that although multiple-occasion urine sampling does provide highly accurate estimates of an individual child's exposure to nicotine, cotinine measurements from single urine samples provide a very accurate estimation of a child's recent exposure (2–3 days) (Matt et al. 2007). Second, the findings of the present study should be confirmed by conducting similar studies in different seasons and with children living in other areas of Italy. In addition to the variables investigated in the present study, u-cotinine concentrations may also be affected by the size of each room in a child's house and the level of ventilation (Blackburn et al. 2003). However, we did not collect any data regarding these potentially variables. Besides, final adjusted R^2 was quite low for both models; in our opinion, residual variability in u-cotinine levels could be explained by other determinants of exposure, evidenced in previous studies but not considered in the present one (parental education, socio-economic status, genetic polymorphism, etc.) (Mannino et al. 2001).

Implications for policy-makers

Our findings suggest that the u-cotinine levels of Italian children are correlated with the smoking habits of and home-smoking precautions adopted by their cohabitants. As there is a constant policy debate about possible strategies for limiting ETS exposure, especially among children, this is an issue with major public health significance.

For this reason, in addition to adopting smoking bans for workplaces and public places, educational interventions on parents are essential to increase their awareness of the negative impacts of ETS exposure in childhood and promoting behaviours that will better protect the children's health.

Ethical standards. The experiments performed in the present research comply with the current laws of Italy, in which they were performed.

Conflict of interest The authors declare that they have no conflict of interest.

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