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Indoor exposure to environmental tobacco smoke and dampness: Respiratory symptoms in Sardinian children—DRIAS study ☆

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ABSTRACT

Indoor exposures at home, environmental tobacco smoke (ETS) and mould/dampness adversely affect respiratory health of children. Disturbi Respiratori nell'Infanzia e Ambiente in Sardegna (DRIAS) (Respiratory Symptoms in children and the Environment in Sardegna, Italy) aims at relating the prevalence of respiratory and allergic symptoms to indoor exposures in Sardinian children.

DRIAS, a cross-sectional investigation of respiratory symptoms/diseases, used a modified version of ISAAC questionnaire, included 4122 children attending 29 primary schools in the school year 2004–2005.

If both parents smoke the prevalence for current wheeze and current asthma is almost doubled in comparison with never smokers, for persistent cough and phlegm a role is suggested when only mother smokes. Among mothers smoking in pregnancy, the prevalence of current wheeze and current asthma is increased. Exposure to ETS and family atopy have a joint effect resulting in an almost tripling of prevalence for current wheeze and more than four times for current asthma. Exposure to "dampness" (mould or dampness) both during the first year of life and currently is associated with increased prevalence of current wheeze, persistent cough or phlegm and current rhino-conjunctivitis; if exposure is only during the first year of life a doubling or more of prevalence is observed for current wheeze, current asthma, and persistent cough or phlegm.

DRIAS results add evidence to the causal role of childhood exposure to ETS in the development of respiratory symptoms (cough, phlegm, and wheezing) and asthma. The joint effect of ETS and family atopy is corroborated. The results strengthen the evidence for a causal association between "dampness" and respiratory health, pointing to its possible independent role in causing asthma, a long-lasting exposure entails a doubled prevalence for both asthmatic and bronchitis symptoms.

1. Introduction

Respiratory health of children is adversely affected by indoor exposures at home, mainly deriving from parental smoking and mould or dampness exposure.

In 2002, a comprehensive quantitative review concluded that the evidence that the environmental tobacco smoke (ETS) exposure in childhood causes chronic respiratory symptoms (cough, phlegm, and wheezing) is strong and consistent, ETS has a causal role in asthma, especially in its induction (Jaakkola and Jaakkola, 2002). The role of ETS in increasing wheeze and asthma risk is consistent with investigations in Italy (SIDRIA 1 and 2-Studi Italiani sui Disturbi Respiratori nell'Infanzia e l'Ambiente) (Agabiti et al., 1999; De Sario et al., 2005) and in other countries (Spengler et al., 2004; Raherison et al., 2007). For smoking in pregnancy, ETS exposure may predict the development of asthma later in life (Jaakkola and Jaakkola, 2002). Successive studies showed that maternal smoking during pregnancy increases the risk of wheeze

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and asthma (Spengler et al., 2004; Zlotkowska and Zejda, 2005; Zacharasiewicz et al., 2000), observation in agreement with Italian investigations (Agabiti et al., 1999; De Sario et al., 2005).

The studies on the relationship of ETS to childhood eczema and rhinitis are not consistent (Raherison et al., 2007; Zacharasiewicz et al., 2000; Kramer et al., 2004).

Many investigations observed an association between home mould or dampness and respiratory symptoms. For indoor dampness, there is sufficient evidence for exacerbation of asthma symptoms, but whether exposure to dampness also causes development of asthma is still unclear (Douwes and Pearce, 2003; IOM report-Institute of Medicine (US), 2004). Also in Italy, SIDRIA 2 observed an increased prevalence for asthma and wheeze (Simoni et al., 2005).

Rhino-conjunctivitis and eczema as well as cough/phlegm are positively related to home mould/dampness in a number of epidemiological investigations (Zacharasiewicz et al., 2000; Simoni et al., 2005; McNally et al., 2001).

The aim of the present study, named Disturbi Respiratori nell'Infanzia e Ambiente in Sardegna (DRIAS)—Respiratory Symptoms in children and the Environment in Sardegna, Italy) is to relate the prevalence of respiratory and allergic symptoms to indoor exposures to ETS and mould or dampness in children living in Sardegna, a southern Italian island. DRIAS results contribute to the knowledge on adverse effects of indoor exposures on respiratory health of children providing evidence from southern Mediterranean populations not previously investigated for these age groups. Sardinian populations have genetic homogeneity, they also share much of the same environment which reduces non-genetic sources of variation, these characteristics make them appropriate for the study of asthma, a multifactorial disease influenced by genetic and environmental factors (Balaci et al., 2007).

2. Materials and methods

2.1. Study subjects

All children attending 29 primary schools located in nine villages in the South–West area of Sardegna-Italy, were included in DRIAS study and investigated during the school year 2004–2005. In Italy, children start primary school at 5 years of age and primary school is compulsory for 5 years. Study subjects amounted to 4122 children.

2.2. Study design

A cross-sectional investigation of respiratory symptoms/diseases was carried out in the period January–April 2005. A self-administered questionnaire, a modified version of ISAAC questionnaire, was filled in by the parents. The questionnaire consisted of five sections on: (i) respiratory and general health of the child; (ii) information on pregnancy and early years of life; (iii) life style habits of the child such as physical exercise and TV watching; (iv) educational, occupational, health characteristics and smoking habits of mother and father; and (v) home characteristics such as presence of mould/dampness, pets, and crowding.

2.3. Outcomes

The possible effects of ETS and mould/dampness exposure on the symptoms/diseases listed below were investigated on the basis of answers to specific questions (questions in brackets).

1. *Current wheeze* (has your child had wheezing or whistling in the chest when breathing in the last 12 months?)
2. *Current asthma* (has your child ever had asthma? and has your child had wheeze or whistle/dyspnea with wheeze/whistle or wheeze after exercise in the past 12 months?)
3. *Persistent cough or phlegm* (has your child ever had cough or phlegm for 4+ days/week apart from cold for 3+ months in the past 12 months?)
4. *Current rhino-conjunctivitis* (has your child had frequent sneezes or runny/stuffy nose, apart from flu or cold, with itching watery eyes in the past 12 months?)
5. *Eczema* the questions in the questionnaire related to eczema were:
   (i) Has your child ever had an itchy rash which was coming and going for at least 6 months?
   (ii) Has your child had this itchy at least once in the last 12 months?
   (iii) Has this itchy rash at any time affected any of the following places: the folds of the elbows, behind the knees, in front of the ankles, under the buttocks, or around the neck, ears or eyes?
   (iv) Has your child ever had eczema?

Prevalence of positive answers to these questions were, respectively, 19.5%, 12.3%, 8.9%, and 23.6%. Eczema was operationally defined as a "yes" answer to both questions (i) and (iii) because they provide a better estimate of prevalence, while direct question about eczema may overestimate the prevalence due to self-diagnosis.

2.4. Exposures

Information on child's indoor exposure was derived from questionnaire answers. Passive smoking was investigated with reference to current wheeze, *current* asthma, and persistent cough/phlegm, the effect of exposure to mould/dampness was studied for all the above-listed symptoms/diseases.

Passive smoking was defined as parents' smoking status and intensity. Parental smoking status was categorized into five mutually exclusive levels: both parents never smokers (i.e. lifetime nonsmokers), both parents past smokers, only child's father current smoker, only child's mother current smoker, both parents current smokers. For smoking intensity maternal and paternal smoking was classified in five groups as never (i.e. lifetime non smokers), former smokers (10 or less cigarettes per day, 11 or more cigarettes per day), current smoker (10 or less and 11 or more cigarettes per day). Maternal smoking in pregnancy was classified as: never smoker, non smoker in pregnancy and smoker in pregnancy. Family atopy was defined as parents or siblings history of asthma or eczema or hay fever.

Exposure to mould or dampness was classified as never, only early, only current and both early and current, on the basis of answers to the questions: "have you seen mould/dampness/fungi on the walls or on the ceiling of your child's bedroom (a) in the first year of your child's life? (only early exposure), (b) recently?" (only current exposure) and (c) both (both early and current). Thirty-nine subjects were excluded from the analysis because they answered "do not remember" with regard to past exposure. Valid answers about this exposure were available for 85% of children.

The confounders were selected on the basis of epidemiological evidence on the association with respiratory health outcomes or study characteristics (e.g. person compiling the questionnaire). Parental education was classified into four categories: "none or elementary", "junior high", "high school degree", and "university degree or higher". Pet ownership was defined as "ever had a dog/cat in the home". Crowding was defined as the average number of people per room. Presence of gas water heater, gas cooker, moisture, dog, and cat were dichotomous variables.

2.5. Statistical analyses

We studied the relationship between indoor pollutant exposures and respiratory outcomes (symptoms/diseases) using a generalised estimating equation (GEE) approach to take into account correlation between observations (Liang and Zeger, 1986). We fitted GEE logistic regression models with the presence/absence of the symptom as a dependent variable. An exchangeable correlation structure between children within the same school were assumed, while children from different schools were considered independent. Robust estimator of standard errors was used (White, 1982).

GEE logistic regression models applied to respiratory outcomes and ETS exposure (Tables 3–6) took into account the following potential confounding variables: age, gender, parental education, crowding, presence of gas water heater, use of gas cooker, questionnaire's compiler, and presence of moisture in child's bedroom.

We also performed an analysis to study the joint effect of ETS exposure, defined as at least one parent current smoker, and family atopy (Table 7) introducing an interaction term in our model.

Models for mould or dampness exposure (Table 8) were adjusted by: age, gender, parental education, crowding, presence of gas water heater, use of gas cookers, presence of dogs or cats at home, and questionnaire's compiler. When analyzing exposure to ETS in pregnancy, we excluded 179 and 52 questionnaires without information, respectively, on natural parents and mother.

All statistical analyses were performed using STATA 9.2 (StataCorp., 2005).

3. Results

Response rate is 84.4% (3477 out of 4122 distributed questionnaires were filled in and returned). Questionnaires without

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answers to one or more of the main core questions of ISAAC questionnaires (lifetime presence of wheezing, rhinitis, and eczema symptoms) were excluded from the analysis. Valid questionnaires with adequate information for the analysis were available for 3455 children (83.8%). Information about siblings was not registered. On the basis of data on address and parents' birth, it is approximately estimated that the cohort included about 94% (3264) independent households and less than 200 children with a sibling.

Table 1 shows general characteristics of children included in the DRIAS study. The age range of children is 5–12 years, the median age is 8 years (not in Table). Table 2 describes the distribution of exposures to ETS and mould or dampness.

Prevalence rate for symptoms considered in DRIAS (Table 3) is 8.45% (95% confidence interval (CI) 7.52%–9.30%) for current wheeze and 3.88% (95% CI 3.23%–4.52%) for *current* asthma. For persistent cough or phlegm, the prevalence is 3.18% (95% CI 2.59%–3.76%), for current rhino-conjunctivitis and eczema, respectively, 6.92% (95% CI 6.07%–7.76%) and 8.94% (95% CI 8.01%–9.94%).

Exposure to parental smoking (Table 4) entails an increased prevalence for current wheeze and *current* asthma when both parents are smokers and never smokers are used as reference. For persistent cough or phlegm, a role is suggested for "only mother smoking".

The odds ratios (ORs) for smoking intensity of father and mother using never smokers as reference are reported in Table 5. For fathers current smoker, the prevalence of current *asthma*

Table 1
Information on 3455 children and distribution of covariates used in the models

Characteristics	Number	%
Age		
<8 years	1260	36.5
Missing	3	<0.1
Gender		
Male	1800	52.1
Female	1655	47.9
Questionnaire's compiler		
Mother	1713	49.6
Father	114	3.3
Both	1452	42.0
Missing	176	5.1
Mother's education		
Primary school or no education	281	8.1
Junior high school	1733	50.2
High school	1115	32.3
University degree	239	6.9
Missing	87	2.5
Father's education		
Primary school or no education	404	11.7
Junior high school	1804	52.2
High school	942	27.3
University degree	177	5.1
Missing	128	3.7
Presence of dog		
Never	1685	48.8
First year	177	5.1
Last year	416	12.6
Other	248	7.2
Missing	929	26.9
Presence of cat		
Never	1704	49.3
First year	108	3.1
Last year	333	9.6
Other	150	4.3
Missing	1160	33.6
Presence of gas cooker		
Yes	3343	96.8
No	70	2.0
Missing	42	1.2
Presence of gas heater		
Yes	344	10.0
No	3020	87.4
Missing	91	2.6
Family atopy	1616	46.8

Table 2
Distribution of exposures: ETS, smoking during pregnancy and mould or dampness

Risk factor	Number	%
Mother's smoking		
Never	1846	53.4
Former	756	21.9
Former 1–10 cigarettes per die	484	14.0
Former 11+ cigarettes per die	213	6.2
Missing data on smoking intensity	59	1.7
Current	775	22.4
Current 1–10 cigarettes per die	461	13.3
Current 11+ cigarettes per die	270	7.8
Missing data on smoking intensity	44	1.3
Missing data on smoking status	78	2.3
Mother's smoking in pregnancy		
Yes	325	9.4
No	2894	83.8
Missing	236	6.8
Father's smoking		
Never	1033	29.9
Former	1037	30.0
Former 1–10 cigarettes per die	293	7.6
Former 11+ cigarettes per die	564	16.3
Missing data on smoking intensity	180	5.2
Current	1242	36.0
Current 1–10 cigarettes per die	382	11.1
Current 11+ cigarettes per die	777	22.5
Missing data on smoking intensity	83	2.4
Missing data on smoking status	143	4.1
Mother and father smoking		
Never smokers	695	22.0
Ex smokers	1004	29.1
At least one smoker	1538	44.5
Missing	218	6.3
Only father smoker	736	21.3
Only mother smoker	249	7.2
Both smokers	479	13.9
Mould or dampness		
Never	2075	60.0
Ever	1302	37.7
Missing	78	2.3
Only current	297	8.6
Only early, during first year of child's life	324	9.4
Both current and early	237	6.9

Table 3
Prevalence of respiratory outcomes: number of cases, prevalence rates and 95% CI

Symptoms/diseases	*N*	Prevalence rates	95% CI
Current wheeze	292	8.45	7.52–9.3
Current asthma	134	3.88	3.23–4.52
Persistent cough or phlegm	110	3.18	2.59–3.76
Current rhino-conjunctivitis	239	6.92	6.07–7.76
Eczema	309	8.94	8.01–9.94

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Table 4
Prevalence, number of cases, estimated ORs and 95% confidence intervals (95% CI) for the association between parental (paternal and maternal) smoking exposure and selected respiratory symptoms/diseases in DRIAS children

Parental smoking	Current wheeze				Current asthma				Persistent cough or phlegm			
	Prevalence %	Cases *N*	OR[a]	95% CI	Prevalence %	Cases *N*	OR[a]	95% CI	Prevalence %	Cases *N*	OR[a]	95% CI
Never smokers ($n = 621$)	6.6	41	1	–	2.9	18	1		3.2	20	1	–
Ex smokers ($n = 877$)	8.0	70	1.21	0.81–1.82	4.0	35	1.39	0.78–2.50	2.6	23	0.76	0.41–1.41
Only father smoker ($n = 636$)	8.3	53	1.29	0.84–1.99	4.7	30	1.80	0.98–3.30	3.0	19	0.80	0.42–1.54
Only mother smoker ($n = 210$)	11.0	23	1.69	0.98–2.91	4.8	10	1.73	0.77–3.86	5.2	11	1.45	0.67–3.13
Both smokers ($n = 388$)	12.1	47	2.03	1.30–3.18	4.9	19	1.96	1.00–3.84	3.4	13	0.95	0.46–1.94

[a] OR adjusted for gender, age, parental education, crowding, presence of moisture in child's bedroom, presence of gas heater/cooker, family atopy.

Table 5
Prevalence, estimated ORs and 95% confidence intervals (95% CI) for the association between paternal and maternal smoking intensity and selected respiratory symptoms/diseases in DRIAS children

Parental smoking	Current wheeze				Current asthma				Persistent cough or phlegm			
	Prevalence %	Cases *N*	OR[a]	95% CI	Prevalence %	Cases *N*	OR[a]	95% CI	Prevalence %	Cases *N*	OR[a]	95% CI
Father												
Never smoker ($n = 860$)	7.0	60	1	–	2.8	24	1	–	3.5	30	1	–
Ex smoker <10 cig/die ($n = 263$)	7.6	20	1.11	0.65–1.90	5.3	14	1.94	0.96–3.94	3.8	10	1.26	0.59–2.67
Ex smoker 11+ cig/die ($n = 487$)	7.4	36	1.10	0.71–1.71	3.7	18	1.40	0.73–2.66	2.5	12	0.67	0.32–1.39
Current <10 cig/die ($n = 318$)	10.4	33	1.36	0.85–2.19	6.0	19	2.14	1.10–4.14	3.5	11	0.96	0.46–2.02
Current 11+ cig/die ($n = 665$)	9.3	62	1.22	0.81–1.83	4.1	27	1.60	0.87–2.94	3.0	20	0.75	0.39–1.42
Mother												
Never smoker ($n = 1558$)	7.3	113	1	–	3.3	51	1	–	2.8	43	1	–
Ex smoker <10 cig/die ($n = 415$)	7.5	31	0.95	0.60–1.48	4.3	18	1.28	0.72–2.30	3.4	14	1.25	0.65–2.40
Ex smoker 11+ cig/die ($n = 185$)	10.8	20	1.26	0.71–2.23	6.5	12	2.06	1.02–4.16	2.7	5	0.91	0.32–2.63
Current <10 cig/die ($n = 362$)	11.0	40	1.43	0.94–2.17	5.0	18	1.50	0.83–2.71	3.3	12	1.34	0.67–2.67
Current 11+ cig/die ($n = 221$)	13.6	30	1.69	1.03–2.75	4.1	9	0.85	0.35–2.07	4.5	19	1.66	0.77–3.58

[a] OR adjusted for gender, age, parental education, crowding, presence of moisture in child's bedroom, presence of gas heater/cooker, family atopy and spouse smoking.

Table 6
Prevalence, estimated ORs and 95% confidence intervals (95% CI) for the association between smoking exposure in pregnancy and selected respiratory symptoms/diseases in DRIAS children

Maternal smoking in pregnancy	Current wheeze				Current asthma				Persistent cough or phlegm			
	Prevalence %	Cases *N*	0	95% CI	Prevalence %	Cases *N*	OR[a]	95% CI	Prevalence %	Cases *N*	OR[a]	95% CI
Never smoker ($n = 1589$)	7.2	115	1	–	3.3	52	1	–	2.8	44	1	–
Nonsmoker in pregnancy ($n = 961$)	9.6	92	1.35	1.01–1.81	4.9	47	1.55	1.03–2.34	3.0	29	1.09	0.68–1.77
Smoker in pregnancy ($n = 280$)	12.9	36	1.79	1.19–2.70	5.7	16	1.84	1.02–3.34	5.4	15	1.71	0.92–3.18

[a] OR adjusted for gender, age, parental education, crowding, presence of moisture in child's bedroom, presence of gas heater/cooker, family atopy.

among *light* smokers (10 or less cigarettes/day) is higher than among smokers of 11+ cigarettes/day, a similar observation is made for current wheeze. For persistent cough or phlegm no consistent pattern emerges.

For mothers current smokers of less than 10 cigarettes/day the prevalence of *current* asthma is higher than among the smoker of 11+ cigarettes/day. This compares with a prevalence among former smokers of 11+ cigarettes/day higher than *light* smokers. Among mothers current smokers prevalence for current wheeze raises with increasing numbers of cigarettes smoked; for persistent cough or phlegm, the prevalence in current smokers is higher in smokers of 11+ cigarettes/day among *heavy* than *light* smokers.

Results for smoking in pregnancy are presented in Table 6. After adjustment, current wheeze and *current* asthma have a higher prevalence among women smoking during pregnancy compared with never smokers. We found similar results for persistent cough or phlegm. In comparison with never smokers an increase is also present among women who abstained from smoking during pregnancy for current wheeze and *current* asthma.

The study results show a joint effect of ETS exposure and family atopy (Table 7) for current wheeze and *current* asthma. When both factors are present, the prevalence for current wheeze is almost tripled and for *current* asthma more than four times using the absence of both factors as reference. Among children with family atopy and no ETS exposure, prevalence is augmented for current wheeze and *current* asthma. For ETS exposure on its own, an increase in prevalence is present for current wheeze and *current* asthma.

In Table 8 the results for exposure to mould or dampness are presented. When exposure to mould or dampness occur *both* early and currently, an increased prevalence is measured for current wheeze, persistent cough or phlegm and current rhino-conjunctivitis.

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Table 7
Joint effect of ETS exposure and family atopy

ETS exposure[a]	Atopy	*N*	%	Current wheeze		Current asthma	
				OR[b]	95% CI	OR	95% CI
No	No	977	28.3	1		1	
Yes	No	861	24.9	1.4	0.9–2.1	1.4	0.7–2.9
No	Yes	940	27.2	1.8	1.2–2.7	2.8	1.5–5.3
Yes	Yes	677	19.6	2.7	1.8–4.0	4.6	2.5–8.5

[a] At least one parent smoker.
[b] OR adjusted for gender, age, parental education, crowding, presence of moisture in child's bedroom, presence of gas heater/cooker.

Table 8
Prevalence, number of cases, estimated ORs and 95% confidence intervals (95% CI) for the association between mould or dampness exposure and respiratory symptoms/diseases in DRIAS children

	Current wheeze			
	Prevalence %	Cases *N*	OR[a]	95% CI
Mould or dampness exposure				
Never ($n = 1183$)	6.6	78	1	–
Only current ($n = 189$)	7.9	15	1.31	0.84–2.05
Only early ($n = 202$)	15.0	31	1.96	1.34–2.88
Both ($n = 142$)	16.9	24	2.41	1.59–3.65
Current asthma				
Never ($n = 1183$)	3.0	35	1	–
Only current ($n = 189$)	4.4	8	1.25	0.66–2.34
Only early ($n = 202$)	9.9	20	1.95	1.15–3.30
Both ($n = 142$)	7.7	11	1.67	0.90–3.10
Persistent cough or phlegm				
Never ($n = 1183$)	2.1	25	1	–
Only current ($n = 189$)	4.2	8	2.10	1.09–4.06
Only early ($n = 202$)	5.9	12	2.42	1.33–4.39
Both ($n = 142$)	4.9	7	2.40	1.24–4.66
Current rhino-conjunctivitis				
Never ($n = 1183$)	6.2	73	1	–
Only current ($n = 189$)	8.5	16	1.61	1.04–2.49
Only early ($n = 202$)	8.9	18	1.09	0.68–1.76
Both ($n = 142$)	14.1	20	2.08	1.32–3.28
Eczema				
Never ($n = 1183$)	8.0	95	1	–
Only current ($n = 189$)	13.8	26	1.34	0.90–2.00
Only early ($n = 202$)	12.9	26	1.30	0.88–1.92
Both ($n = 142$)	10.6	15	1.04	0.65–1.65

[a] OR adjusted for gender, age, parental education, person who filled the questionnaire, crowding, presence of passive smoking, presence of gas cooker/heater, presence of dog/cat, family atopy (asthma, rhinitis, eczema).

Exposure to mould or dampness *only* in the first year of child's life ("only early") increases prevalence for current wheeze, *current* asthma, and persistent cough or phlegm. *Only current exposure* seems to play a role for rhino-conjunctivitis and to a lesser extent for eczema.

4. Discussion

In the present cross-sectional study, an association was observed between parental smoking and current wheeze and *current* asthma, the prevalence is doubled if both parents smoke, for persistent cough or phlegm the major role is for "only mother smoker". An increased prevalence for current wheeze and *current* asthma is observed for smoking in pregnancy. Exposure to ETS and family atopy have a joint effect with an almost tripled prevalence for current wheeze and more than four times for *current* asthma. The prevalence is doubled or more for current wheeze, persistent cough or phlegm, and current rhino-conjunctivitis when exposure to mould or dampness occur *both* early and currently. Exposure to "dampness" *only* during the first year of life entails a doubling of prevalence for current wheeze and *current* asthma, nearly 2.5 times for persistent cough or phlegm.

The 8.45% (95% CI 7.5–9.3) prevalence rate of current wheeze in DRIAS children is similar to that measured in SIDRIA, 8.6% (Galassi et al., 2006) and in other European investigations (Spengler et al., 2004; Zlotkowska and Zejda, 2005; Maziak et al., 2003; Garcia-Marcos et al., 2004; Braun-Fahrlander et al., 2004); the same applies to rhino-conjunctivitis, with a prevalence rate equals to 6.92% (95% CI 6.07–7.76), in SIDRIA 6.8% and to eczema, 8.9% (95% CI 8.01–9.94) in DRIAS, and 10.4% in SIDRIA (Galassi et al., 2006).

As far as smoking intensities of father and mother are concerned, DRIAS results for current wheeze and *current* asthma are in overall agreement with Italian SIDRIA 1 and 2 studies (Agabiti et al., 1999; De Sario et al., 2005). Given that fathers are less present at home, the observation of an increased prevalence for smoking father suggest that this variable is a proxy of ETS at home. The lack of association with mothers currently "*strong smokers*" could be explained by *avoiding bias* leading mothers of asthmatic children to abstain from smoking when the child is present; the possibility of such a bias is supported by the increased prevalence when mothers are former "*strong smokers*".

In the present study, the increased prevalence of current wheeze and *current* asthma with smoking in pregnancy is consistent with the Italian cross-sectional investigation SIDRIA 1 (Agabiti et al., 1999) and other studies (Jaakkola and Jaakkola, 2002; Spengler et al., 2004; Raherison et al., 2007; Mommers et al., 2005; Jaakkola et al., 2006) adopting both cross-sectional (Raherison et al., 2007; Zlotkowska and Zejda, 2005; Pattenden et al., 2006; Jaakkola et al., 2006) and analytical design (Jaakkola and Gissler, 2004; Mommers et al., 2005). ETS exposure in pregnancy can predict the later development of asthma possibly through adverse effects on foetal growth and decreased development of lung function (Jaakkola and Jaakkola, 2002; Jaakkola et al., 2006); the independent role of ETS in pregnancy is documented in a US study where there is no exposure subsequent to the one in pregnancy (Gilliland et al., 2001).

The present results and the available evidence point to the need for smoking control programmes to reduce children's exposure to ETS, given that these programmes have been shown to work (De Sario et al., 2005).

The results on the joint effect of ETS and family atopy are consistent with studies conducted in Norway (Jaakkola et al., 2001) and Sweden (Rönmark et al., 1999). In the first cohort investigation (Jaakkola et al., 2001), the OR for asthma in presence of both family atopy and ETS is 2.68 (95% CI 1.70–4.22), also in the Swedish cross-sectional study (Rönmark et al., 1999) an increased asthma prevalence is present (OR 6.88 (95% CI 4.03–1.75). In a cross-sectional study of adults never smokers, the prevalence of asthma among those with ETS exposure in infancy and a family history of asthma is 13.2% versus 7.6% among those not so exposed (Larsson et al., 2001).

In DRIAS an increased prevalence for current wheeze, *current* asthma, persistent cough or phlegm and current rhino-conjunctivitis is observed when exposure to mould or dampness occurs *both* in the first year of life and in the past year as well when occurring: these results point to the need for mould/dampness control actions (Kercsmar et al., 2006). The importance of *only early* exposure to "dampness" entailing a doubled prevalence for

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current wheeze and *current* asthma is in agreement with SIDRIA2 cross-sectional investigation (Simoni et al., 2005) and other similar surveys (Li and Hsu, 1996; Bornehag et al., 2005), as well as case-control studies (Mommers et al., 2005; Li and Hsu, 1996; Bornehag et al., 2005; Pekkanen et al., 2007).

The strong effect of *only early* exposure on current wheeze or *current* asthma observed in DRIAS was present also among Scandinavian children (with an older asthmatic sibling) for exposure in the first two years of life (Forsberg et al., 1997), this result is in line with the hypothesis that indoor mould/dampness exposure has an independent effect on the development of asthma as also suggested by a recent cohort study (Jaakkola et al., 2005). Also SIDRIA 2 measured an increased wheeze prevalence for *early* mould exposure (Rusconi et al., 2005); fungi in moulds could be acting as irritant or increasing susceptibility to infections (Stark et al., 2003).

The comparison of DRIAS results for wheeze and asthma when exposure occurs *currently* (independent from the presence/absence of early exposure) with other studies adopting the same design and exposure definition, shows an agreement on the presence of this association (Dales et al., 1997; Forsberg et al., 1997; Yang et al., 1997; Jedrychowski and Flak, 1998; Spengler et al., 2004; Simoni et al., 2005).

For persistent cough or phlegm, DRIAS is in overall agreement with previous studies (Simoni et al., 2005; Cuijpers et al., 1995; Yang et al., 1997) where an effect was present regardless of timing of exposure.

An association of mould or dampness with current rhino-conjunctivitis has been reported by other authors in Italy (Simoni et al., 2005) and abroad (Zacharasiewicz et al., 2000; Li and Hsu, 1996; Bornehag et al., 2005). For eczema, the observation of an increase in prevalence was in agreement with previous investigations (Simoni et al., 2005; McNally et al., 2001).

The main limitations of the present study are the cross-sectional design and the self-reporting of both health outcomes and independent variables considered as exposure or confounding variables.

Because of the high response rate (84.4%) and the low percentages of missing values of both covariates and exposure variables (22 observations), selection bias should not be a major problem. As far as respiratory symptoms are concerned, the validity of self-reported information for asthma is documented (Pekkanen et al., 2005), even if, as in any cross-sectional study, the possibility of recall bias cannot be excluded. Parents of symptomatic children may be more inclined to under-report their smoking habits, while for exposure to mould or dampness under or over-reporting is possible.

In the present study exposure variables were not confirmed by objective measurements, on this topic there is evidence documenting that questionnaire can give a valid estimate of residential ETS exposure among children (Strachan and Cook, 1997).

Exposure to mould or dampness in DRIAS also lacks of objective measurements. In addition the wording of the question makes it impossible to distinguish between mould or dampness; however, a strong correlation has been shown between these two factors objectively assessed (Williamson et al., 1997). The fact that the information is for child's bedroom, a location where he/she presumably spend most of the time, stands for the validity of the exposure information (Pekkanen et al., 2007). Although lacking of objective measurements DRIAS results are in agreement with other studies, cross-sectional (Spengler et al., 2004; Zlotkowska and Zejda, 2005; Simoni et al., 2005; Li and Hsu, 1996; Bornehag et al., 2005) or case-control (Mommers et al., 2005; Pekkanen et al., 2007). Exposure information was based either on self-reported information on mould or dampness in the home (Spengler et al., 2004; Mommers et al., 2005; Li and Hsu, 1996), in child's bedroom (Bornehag et al., 2005) or on the evaluation of moisture damage in child's bedroom from trained engineers (Pekkanen et al., 2007).

In conclusion, the present study adds evidence to the causal role of exposure to ETS in childhood in the development of chronic respiratory symptoms and asthma (Jaakkola and Jaakkola, 2002), and confirms its role in increasing the risk for cough and phlegm (Strachan and Cook, 1997). The observation of the joint effect of ETS and family atopy on wheeze and asthma is in agreement with the hypothesis that asthma development results from the effects of environmental stimuli in genetically susceptible individuals, as demonstrated in a limited number of studies on gene–environment interaction (Kurz and Ober, 2007; Yang et al., 2007). The importance in understanding such interaction lays in the possible improvement of preventive strategies and in targeted interventions in children with asthma (Yang et al., 2007).

The respiratory risk from exposure to ETS during pregnancy (Jaakkola and Jaakkola, 2002; Agabiti et al., 1999; Spengler et al., 2004; Raherison et al., 2007; Zlotkowska and Zejda, 2005; DiFranza et al., 2004) is corroborated by DRIAS results, its independent role as risk factor (Zlotkowska and Zejda, 2005; DiFranza et al., 2004) is confirmed, the observation of an effect among women not smoking in pregnancy (Agabiti et al., 1999) is repeated. The adverse respiratory effects of prenatal and postnatal exposure to passive smoking calls for action to reduce such exposure, properly considering that the overall evidence of effectiveness is insufficient and the evidence to recommend one specific strategy ahead of changing background social trends is also insufficient (Roseby et al., 2003).

The results of DRIAS investigation strengthen the evidence for a causal association between "dampness" and respiratory health, possibly not only exacerbating but playing an independent role in causing asthma (Douwes and Pearce, 2003; IOM report-Institute of Medicine (US), 2004), they also document that a long-lasting exposure (*both* early and current) exerts an important role for both asthmatic and bronchitis symptoms. The mechanism linking dampness to asthma is not clear, but preventive measures should be taken considering the available evidence of a consistent association between reported mould/dampness and increased prevalence of bronchial hyper responsiveness (Hagmolen of ten Have et al., 2007) and of airflow obstruction in presence of objectively confirmed dampness areas (Williamson et al., 1997). Furthermore a population-based cohort study documented the independent effect of parental atopy and exposure to moulds on asthma development (Jaakkola et al., 2005). Preventive measures against dampness in homes should be adopted without delay also because their efficiency has been recently documented in prospective randomized control trial of children with asthma (Kercsmar et al., 2006).

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