

ORIGINAL ARTICLE

Asthma diagnosis in a child and cessation of smoking in the child's home: the PIAMA birth cohort

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Second hand smoke (SHS) exposure is associated with increased incidence and severity of childhood asthma. We investigated whether, in turn, asthma diagnosis in a child is associated with cessation of smoking exposure in the child's home. In the PIAMA birth cohort ($n = 3963$), parents reported on smoking in their home and on asthma diagnosis in their child, annually from birth to 8 years. We used generalized estimating equations to assess the association between asthma diagnosis in a child and cessation of smoking in the child's home. Among children with residential SHS exposure, smoking stopped in 23.7% of the homes of children with newly diagnosed asthma as compared with 16.2% of the homes of children without asthma diagnosis ($P = 0.014$). For children with an asthma diagnosis, the relative risk of smoking cessation in their home was 1.36 (one-sided 95% confidence interval: 1.09, inf.) and changed little after adjustment for maternal education, parental allergy and child's age. In most smokers' households (76.3%), smoking continued when the child got an asthma diagnosis. Nevertheless, an asthma diagnosis in the child increased the probability of a smoke-free home for the child and its parents and siblings. Cross-sectional associations between SHS exposure and asthma may underestimate true associations, because exposure may have been reduced following diagnosis of the disease.

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INTRODUCTION

It is well established that second hand smoke (SHS) exposure of children is associated with asthma exacerbations,¹ with increased prevalence² and incidence³ of asthma and wheeze, and with chronic respiratory symptoms in non-smoking adults.⁴

In this study, we considered the possibility of a “feedback loop” in the SHS-childhood asthma association, in the sense that an asthma diagnosis may result in reduction of SHS exposure. From adult studies, it is known that incidence or diagnosis of a serious disease, may increase the probability of quitting smoking among patients as compared with people unaffected by disease.^{5–8} It is unknown whether the occurrence of a disease is also an incentive for close relatives of a patient to refrain from smoking for the benefit of the patient. It seems likely that this may be the case, especially if disease occurs in a child. On the other hand, there is evidence (reviewed in refs 9,10) that interventions aimed to make smoking parents quit smoking in the interest of their child, are mostly unsuccessful.

In this study, we hypothesize that parents who are told by a doctor that their child has asthma, may be more inclined to reduce their child's exposure to SHS than parents of a child without an asthma diagnosis. More specifically, our study question is whether a doctor's diagnosis of asthma in a child increases the likelihood of cessation of smoking in the child's home.

MATERIALS AND METHODS

Study Design and Population

We used data from the Prevention and Incidence of Asthma and Mite Allergy (PIAMA) study, a population-based birth cohort study investigating the influence of lifestyle and environment on asthma development. Details of the study design have been published previously.¹¹ Pregnant women were recruited from the general population and their children ($n = 3963$) were born in 1996/1997. For this study, we used data from questionnaires on family characteristics, lifestyle and health that were completed annually by the parents during the first 8 years of life. The study protocol was approved by the Medical Ethical Committees of the participating research institutes and all parents gave written informed consent.

Data and Study Variables

We used doctor's diagnosis of asthma in the child as the independent variable and smoking cessation in the child's home as the dependent variable. Parents reported doctor's diagnosis of asthma ever (a positive answer to the question “Did a doctor ever diagnose asthma in your child? yes/no”) in each of the annual questionnaires completed at ages 1–8 years and smoking in the child's home (any smoking in the home at least once a week, yes/no) in the questionnaires completed at 3 months and at 1–8 years of age. We defined smoking cessation in the child's home as change from a home with smoking in a specific follow-up (t_0) to a home without smoking in the subsequent follow-up (t_1).

Statistical Analysis

The majority of the PIAMA participants (60.2%) never reported smoking in the home once a week or more during the entire study period

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Table 1. Questionnaires completed and prevalence of smoking in the child's home per follow-up; and numbers of observations included in the study from the respective follow-ups (t_0), and the next follow-up (t_1).

Follow-up (t_0)	Questionnaires completed	Homes with smoking	Homes with smoking and child never diagnosed with asthma at t_0	Data available from questionnaire at t_1
	n	n (% ^a)	n	n
3 months	3,934	1,129 (29)	1,129	1,056
1 year	3,746	1,058 (28)	1,005	954
2 years	3,740	1,054 (28)	975	929
3 years	3,694	981 (27)	882	805
4 years	3,563	852 (24)	753	711
5 years	3,518	835 (24)	728	691
6 years	3,472	753 (22)	660	607
7 years	3,374	641 (19)	525	475
Total			6,657	6,228

^aPercentage of respondents.

Table 2. Cessation of smoking in homes where the child was diagnosed with asthma between one follow-up (t_0) and the next follow-up (t_1) compared with cessation of smoking in homes where the child was not diagnosed with asthma during the same period.

Follow-up (t_0)	Smoking in the home at t_0 but not at t_1		Smoking in the home at t_0 , but not at t_1 and not at t_2^a	
	Child diagnosed with asthma between t_0 and t_1	Child not diagnosed with asthma between t_0 and t_1	Child diagnosed with asthma between t_0 and t_1	Child not diagnosed with asthma between t_0 and t_1
	% (n/N)	% (n/N)	% (n/N)	% (n/N)
3 months	25 (14/56)	14.6 (146/1,000)	16.4 (9/55)	9.6 (90/936)
1 year	12.5 (3/24)	12 (112/930)	13.6 (3/22)	7.8 (68/874)
2 years	26.1 (6/23)	16.8 (152/906)	23.8 (5/21)	12.5 (102/818)
3 years	31.6 (6/19)	17.6 (138/786)	23.5 (4/17)	12.7 (93/730)
4 years	20 (3/15)	14.4 (100/696)	21.4 (3/14)	10.8 (71/657)
5 years	11.7 (2/12)	16.1 (109/679)	0 (0/9)	11.8 (73/618)
6 years	30.8 (4/13)	22.9 (136/594)	30.8 (4/13)	17.4 (93/535)
7 years	28.6 (2/7)	19 (89/468)	—	—
Total	23.7 (40/169) ^b	16.2 (982/6,059) ^b	18.5 (28/151) ^c	11.4 (590/5,168) ^c

^aOnly t_0 observations up to age 6 years were included in this analysis and children who were diagnosed with asthma between t_1 and t_2 ($n = 97$) were excluded from this analysis. ^b P for association = 0.014 (based on one-sided Wald test, taking into account correlations between observations in the same child). ^c P for association = 0.005 (based on one-sided Wald test, taking into account correlations between observations in the same child).

and were therefore not included in this study. Table 1 shows the numbers of questionnaires available at the child's ages of 3 months–8 years, and the numbers of observations that were available for this analysis.

As a starting point (t_0) for our analyses, we identified all observations from children aged 3 months–7 years, who were living in a home with smoking and who had never been diagnosed with asthma: a total number of 6,657 observations (Table 1). For these observations, we then considered the situation at the next follow-up (t_1) and assessed the dependent variable smoking cessation in the child's home (i.e., smoking stopped between t_0 and t_1). For this analysis, 6,228 of the 6,657 observations were eligible; for the remaining 429 observations data were missing at t_1 . We calculated the percentage of homes that became smoke-free in the year in which the child was diagnosed with asthma ($t_0 - t_1$) and compared this to the percentage of homes that became smoke-free without the child having been diagnosed with asthma in that year. A one-sided Wald test was used to test the null hypothesis that these two percentages were equal against the alternative hypothesis that the percentage of smoking cessation at home in the year of an asthma diagnosis was larger than the percentage of smoking cessation at home in the absence of an asthma diagnosis.

As several observations per child were included in the analysis, we expected the observations not to be independent. Therefore, we used generalized estimating equations (GEE) to estimate the relative risk (RR) and the 95% one-sided confidence interval (CI) for the association between an asthma diagnosis and cessation of smoking in the home. A log function

for binary data was used and an autoregressive (AR(1)) serial correlation structure was chosen to account for possible dependence of observations on the same child. On the basis of evidence from the literature, the following covariates were considered as potential confounders: maternal education (as a "summary indicator" of the family's health related lifestyle; categories high, intermediate or low), maternal and paternal allergy (defined as having any of the following: asthma ever, current house dust mite allergy, pet allergy or a nasal allergy, such as hay fever) and child's age at the time of smoking cessation in the home.

In additional analyses, we assessed the persistence of non-smoking in homes that became smoke-free. We calculated the percentage of homes that became smoke-free in the year in which the child was diagnosed with asthma ($t_0 - t_1$) and that were still smoke-free 1 year later (at t_2) and compared this with the percentage of homes that were smoke-free at both t_1 and at t_2 without the child having been diagnosed with asthma up to t_2 . Also for the outcome "smoke-free home at both t_1 and at t_2 " we conducted a GEE analysis as described above.

In addition to assessing the influence of potential confounders, we also investigated, within the group of children with an asthma diagnosis, the possible role of asthma severity in smoking cessation in the home, using inhalation corticosteroids (ICS) use as an indicator of asthma severity. Data on ICS use were available only from the age of 3 years onward, consequently children < 2 years old at t_0 were excluded from this analysis.

Although attrition was low in the PIAMA cohort, we considered the possibility of selective loss-to-follow-up of households where smoking

Table 3. RRs (one-sided 95% CI) of smoking cessation in the child's home for children with newly diagnosed asthma (children without asthma diagnosis are the reference group).

Model ^a	RR (1-sided 95% CI) (n = number of observations used in the analysis)	
	Smoking in the home at t_0 but not at t_1	Smoking in the home at t_0 , but not at t_1 and not at t_2
Asthma diagnosis in the child		
Crude	1.36 (1.09, inf.) (n = 6228)	1.58 (1.13, inf.) (n = 5319)
Adjusted for:		
Maternal education	1.38 (1.10, inf.) (n = 6182)	1.62 (1.16, inf.) (n = 5298)
Maternal allergy	1.34 (1.07, inf.) (n = 6228)	1.55 (1.11, inf.) (n = 5319)
Paternal allergy	1.35 (1.08, inf.) (n = 6220)	1.53 (1.09, inf.) (n = 5311)
Child's age	1.41 (1.13, inf.) (n = 6228)	1.67 (1.19, inf.) (n = 5319)

Abbreviations: CI, confidence interval; RR, relative risks. ^aCovariates were included in the regression model one at a time because the number of degrees of freedom was insufficient to estimate the regression coefficients with enough precision with all covariates in the model at the same time.

continued despite an asthma diagnosis in the child. We conducted a sensitivity analysis in order to assess the magnitude of the bias that such selective loss-to-follow-up might have caused.

Code Availability

The data and the computer code underlying the findings presented in this paper are available on request. Requests can be submitted to the PIAMA Principal Investigators. Their names and e-mail addresses are listed on the PIAMA website (<http://piama.iras.uu.nl/index-en.php#collaboration>).

RESULTS

At t_0 , all children were exposed to SHS at home and none of them had been diagnosed with asthma. At t_1 , for the majority of observations (81.5%) the situation was unchanged: smoking in the home had continued and the child had not been diagnosed with asthma. A total of 169 children (2.7%) had been diagnosed with asthma at t_1 (Table 2). At nearly every age, the incidence of smoking cessation was higher in homes of children with newly diagnosed asthma than in homes of children without asthma diagnosis (Table 2). Overall, in the group of 169 children with newly diagnosed asthma between t_0 and t_1 , smoking in the home had stopped in that year in 23.7% (40/169) of the homes, as compared with a percentage of 16.2% (982/6059) smoking cessation if the child did not get an asthma diagnosis (Table 2).

The GEE analysis showed a relative risk of smoking cessation in their home of 1.36 (one-sided 95% CI: 1.09, inf.) for children with an asthma diagnosis as compared with the reference group of children without asthma diagnosis. Adjustment for potential confounders (one at a time) did not substantially change the strength of this association (Table 3). Asthma severity, as indicated by ICS use, did not seem to substantially contribute to smoking cessation in homes of children with an asthma diagnosis: of the children with an asthma diagnosis and whose homes became smoke-free 48% (11/23) were ICS users as compared with 44% (28/64) of the children with an asthma diagnosis whose homes did not become smoke-free.

Most of the homes that became smoke-free between t_0 and t_1 were still smoke-free at the subsequent follow-up, 1 year later (t_2). This was the case in 80% (28 out of 35 with complete follow-up data) of the homes where the child was diagnosed with asthma and in 71% (590 out of 835 with complete follow-up data) of the homes where the child was not diagnosed with asthma. In additional analyses, we used smoking in the home at t_1 and at t_2 (instead of smoking in the home at t_1 only) as dependent variable to obtain insight into "long term" smoking cessation. Of the homes where children were diagnosed with asthma 18.5% became smoke-free in the year of the asthma diagnosis and were still smoke-free at the next follow-up, 1 year later. In homes where the child was not diagnosed with asthma, this percentage was

11.4% (Table 2). GEE analysis showed that compared with children without asthma diagnosis, children with an asthma diagnosis had a relative risk of living in a smoke-free home at t_1 and t_2 of 1.58 (one-sided 95% CI: 1.13, inf.). Adjustment for potential confounders (one at a time) did not substantially change the strength of this association (Table 3).

As a sensitivity analysis, we assessed the possible impact of selective loss-to-follow-up on our results in a hypothetical scenario. For the 429 families with missing data at t_1 we assumed incidence of asthma diagnosis to be 2.7%, as in the total study population. The number of asthma diagnoses in this group would then be 12. If, in this sub group with missing data, smoking continued in the homes of all 12 children with an asthma diagnosis, but stopped in 16.2% of those without an asthma diagnosis (as in the total study population with complete data), the incidence of asthma-related smoking cessation in the total population would be 22.1% (instead of the 23.7% observed in our data set) as compared with 16.2% in homes where the child did not get an asthma diagnosis (P for association = 0.050, based on one-sided Wald test taking into account correlations between observations in the same child).

DISCUSSION

We found that in smokers' households an asthma diagnosis in a child increases the likelihood of smoking cessation in the home. The majority of parents of children with a new asthma diagnosis in the last 12 months still reported smoking in the home, but 23.7% of them reported their home to be smoke-free now. In families where the child did not get an asthma diagnosis, 16.2% of the homes became smoke-free during the study period. In the families where the child was diagnosed with asthma 18.5% of the homes became smoke-free and were still smoke-free 1 year later, as compared with 11.4% in the absence of an asthma diagnosis.

We observed that smoking cessation in the home also occurred in families where the child did not get an asthma diagnosis during the study period (1996–2005). This observation is in line with results from a Dutch trend study conducted over the period 1996–2009¹² that coincided with national policy measures to discourage smoking and a campaign aimed to reduce SHS exposure of young children (but not specifically focused on children with respiratory symptoms).¹³ This trend study showed that in households with a 0–4-year-old child, the percentage with a smoking family member decreased from 48% in 1996 to 33% in 2009.¹² However, SHS exposure in children is still an important problem in the Netherlands, as was shown in a recent pilot study for a new intervention program.¹⁴

We extended the evidence on smoking cessation in adults who are themselves confronted with a disease,^{5–8} by addressing the

question of reduction of SHS exposure if a child is diagnosed with a disease. Previous studies addressed the issue of parental smoking cessation to protect child health mainly in the context of interventions to encourage parents to quit smoking for their children's benefit. A Cochrane review⁹ and a systematic review and meta-analysis¹⁰ of such intervention trials both concluded that the majority of the interventions they reviewed were unsuccessful. Our study differed from the studies covered in these reviews in that we did not conduct an intervention and have no information on the advice parents were given by their physician. Instead, we considered a doctor's diagnosis of asthma as a potential trigger for smoking cessation in the child's home. Despite these differences, our finding that the majority of smokers' homes did not become smoke-free when the child was diagnosed with asthma is in line with the conclusions of the reviews. We could not identify any pediatric studies with a design similar to ours, that is, prospective studies with a control group of children who did not get (a diagnosis of) asthma.

Important strengths of our study were the prospective design and the availability of annually repeated measurements. This enabled us to assess pre- and post-diagnosis residential SHS exposure from year to year over a period of 8 years, avoiding diagnosis-related reporting bias that may occur in retrospective studies. In addition, our population-based cohort included a large group of subjects without asthma diagnosis, so that we could take the general trend in smoking prevalence into account. Furthermore, we validated the parental reported data on smoking in the home with measurements of air nicotine concentrations in sub groups of households when the children were 3 months¹⁵ and 4 years old.¹⁶ Agreement between questionnaire reports and air nicotine levels was good (11.5% misclassified) and there was no indication that parents of symptomatic children under-reported smoking.¹⁶ Our sensitivity analysis on the potential impact of missing data showed that it is unlikely that our findings are explained by selective loss-to-follow-up.

A number of limitations of the study have to be considered as well. An important point of concern in all questionnaire based studies is the possibility of selective under- or over-reporting by respondents. Although our validation study¹⁶ did not show evidence for selective under-reporting of smoking in the home by parents of children with respiratory symptoms, we cannot exclude the possibility that parents of children with an asthma diagnosis under-reported smoking in their home. The PIAMA cohort has a baseline study population of $n=3,963$, but the majority of families lived in smoke-free homes throughout the study period and were not included in this study. As a result, our final study population was too small to conduct more detailed analyses, such as age specific analyses. Furthermore, we did not know the exact timing of the asthma diagnoses and of smoking cessation. We know whether or not these events took place in the last 12 months, but we cannot be certain that smoking cessation in the home always followed the diagnosis of asthma when both occurred in the last 12 months. However, it is reasonable to assume that an asthma diagnosis is preceded by a period of recurrent symptoms and even if smoking cessation in the home did take place before the diagnosis, it may still be related to the occurrence of symptoms before the diagnosis. Our results are limited to cessation of smoking in the child's home and we do not have information on SHS exposure of the child elsewhere. Also, we do not know whether the children with a parental reported doctor's diagnosis of asthma really had asthma. In young children, asthma cannot be diagnosed reliably and also errors in parental reporting cannot be excluded. However, in the context of the present study, the perception of the diagnosis by the parents is the factor that is most relevant for their subsequent behavior.

Having considered these limitations and uncertainties, our interpretation of the study's results is that an asthma diagnosis in

a child may give parents an additional incentive to create a smoke-free home environment for their child. At the same time, however, the results show that smoking continued in the majority of smokers' households in spite of an asthma diagnosis in the child. Together these observations indicate that health care providers must continue to stress the importance of a smoke-free home for children's health and should offer parents the support they need to achieve that.

From a methodological perspective, our findings indicate that cross-sectional associations between SHS exposure and asthma are likely to underestimate true associations, because asthma patients' exposure may have been reduced following diagnosis of the disease. We revealed a mechanism that may cause smoking to be less common in homes of children with asthma, which, in observational studies, could even mistakenly be interpreted as a "protective" effect of tobacco smoke exposure on asthma prevalence. In the PIAMA study, we previously showed that smoking was less common among parents with asthma or allergy than among parents without these conditions, so that children with increased asthma risk due to their family history, were more likely to be born and grow up in a home without SHS exposure.¹⁷ Results of the present study add to these earlier findings that also an asthma diagnosis in a child may increase the probability of a smoke-free home for the child as well as for the child's parents and siblings. For future studies, we therefore recommend prospective designs that take into account the feedback loop indicated in our study.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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REFERENCES

- 1 Chilmonczyk BA, Salmun LM, Megathlin KN, Neveux LM, Palomaki GE, Knight GJ *et al.* Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. *N Engl J Med* 1993; **328**: 1665–1669.
- 2 Strachan DP, Cook DG. Health effects of passive smoking. 6. Parental smoking and childhood asthma: longitudinal and case control studies. *Thorax* 1998; **53**: 204–212.
- 3 Burke H, Leonardi-Bee J, Hashim A, Pine-Abata H, Chen Y, Cook DG *et al.* Prenatal and passive smoke exposure and incidence of asthma and wheeze: systematic review and meta-analysis. *Pediatrics* 2012; **129**: 735–744.
- 4 David GL, Koh W-P, Lee H-P, Yu MC, London SJ. Childhood exposure to environmental tobacco smoke and chronic respiratory symptoms in non-smoking adults: The Singapore Chinese Health Study. *Thorax* 2005; **60**: 1052–1058.
- 5 Falba T. Health events and the smoking cessation of middle aged Americans. *J Behav Med* 2005; **28**: 21–33.
- 6 Keenan PS. Smoking and weight change after new health diagnoses in older adults. *Arch Intern Med* 2009; **169**: 237–242.
- 7 Manschot A, van Oostrom SH, Smit HA, Verschuren WM, Picavet HS. Diagnosis of diabetes mellitus or cardiovascular disease and lifestyle changes—The Doetinchem Cohort Study. *Prev Med* 2014; **59**: 42–46.
- 8 van Gool CH, Kempen GI, Penninx BW, Deeg DJ, van Eijk JT. Chronic disease and lifestyle transitions: results from the Longitudinal Aging Study, Amsterdam. *J Aging Health* 2007; **19**: 416–438.
- 9 Baxi R, Sharma M, Roseby R, Polnay A, Priest N, Waters E *et al.* Family and carer smoking control programmes for reducing children's exposure to environmental tobacco smoke. *Cochrane Database Syst Rev* 2014; **3**: CD001746.
- 10 Rosen LJ, Noach MB, Winickoff JP, Hovell MF. Parental smoking cessation to protect young children: a systematic review and meta-analysis. *Pediatrics* 2012; **129**: 141–152.

- 11 Wijga AH, Kerkhof M, Gehring U, de Jongste JC, Postma DS, Aalberse RC *et al*. Cohort profile: the prevention and incidence of asthma and mite allergy (PIAMA) birth cohort. *Int J Epidemiol* 2013; **43**: 527–535.
- 12 Crone MR, Nagelhout GE, van den Burg I, HiraSing RA. Passive smoking in young children in the Netherlands sharply decreased since 1996. *Nederlands Tijdschrift voor Geneeskunde* 2010; **54**: A1658.
- 13 Crone MR, Reijneveld SA, Willemsen MC, Sing RA. Parental education on passive smoking in infancy does work. *Eur J Public Health* 2003; **13**: 269–274.
- 14 Hutchinson SG, Mesters I, van Schayck CP, Dompeling E. Prevention of passive smoking exposure in children at risk for asthma: results of a pilot study. *Eur Respir J* 2011; **38**: 1548.
- 15 Brunekreef B, Leaderer BP, van Strien R, Oldenwening M, Smit HA, Koopman L *et al*. Using nicotine measurements and parental reports to assess indoor air: the PIAMA birth cohort study. Prevention and incidence of asthma and mite allergy. *Epidemiology* 2000; **11**: 350–352.
- 16 Gehring U, Leaderer BP, Heinrich J, Oldenwening M, Giovannangelo ME, Nordling E *et al*. Comparison of parental reports of smoking and residential air nicotine concentrations in children. *Occup Environ Med* 2006; **63**: 766–772.
- 17 Wijga A, Smit HA, Brunekreef B, Gerritsen J, Kerkhof M, Koopman LP *et al*. Are children at high familial risk of developing allergy born into a low risk environment? The PIAMA Birth Cohort Study. *Clin Exp Allergy* 2001; **31**: 576–581.