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Maternal and paternal indoor or outdoor smoking and the risk of asthma in their children: A nationwide prospective birth cohort study☆

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ABSTRACT

Background: Little is known about the differential impact of combinations of parental smoking behavior (indoor or outdoor smoking, or not smoking) on preventing childhood asthma. Our objective was to examine the association between parental smoking behavior and children's asthma.
Methods: A nationally representative population-based birth cohort of 40,580 babies, aged 0.5 years in 2001 (response rate, 87.8%), was studied to estimate adjusted odds ratios of combinations of maternal and paternal indoor or outdoor smoking at home for physician visits and hospitalization for childhood asthma up to 8-years-old, and population attributable fractions.
Results: Odds of hospitalization for asthma among children whose father alone smokes indoors at home did not largely increase (up to 20%). However, if the mother also smokes indoors at home, the odds strongly increased. After adjusting for demographic, perinatal and socioeconomic factors, the increase in odds for children whose father and mother both smoke indoors compared to children with non-smoking parents was 54% (95% confidence interval: 21–96%), 43% (8–90%) and 72% (22–143%) for children aged 0.5 < –2.5, 2.5 < –4.5 and 4.5 < –8 years-old, respectively. The odds ratios of smoking outdoors did not largely differ from those of smoking indoors. Our estimation of population attributable fractions revealed that if all parents in Japan quit smoking, hospitalization of children for asthma could be reduced by 8.3% (2.2–14.3%), 9.3% (0.9–17.6%) and 18.2% (7.7–28.8%), respectively.
Conclusions: Parental indoor smoking at home increased and exacerbated children's asthma. Smoking at home, whether it is indoors or outdoors, may increase the risks for asthma attacks of their children.

1. Introduction

Children are likely to be exposed to second hand tobacco smoke (SHS) at home (U.S. Department of Health and Human Services [USDHHS], 2006). A study in Japan has shown that 64.8% of 6-month old children live with smoking parent(s), and of those, 57.9% of parents smoke indoors at home (Kaneita et al., 2006). Although many previous studies have revealed the risk of SHS for childhood asthma (Royal College of Physicians, 2010; USDHHS, 2006), a recent review by Burke et al. (2012) showed several evidence gaps in this field of research. There has been no prospective study of the risk of paternal smoking for asthma in children aged 2 years or less and only one study for children aged 3–4 years. Further, a wide range of estimated effect size of postnatal maternal smoking on incidence of childhood asthma was observed, indicating a need to confirm the results. One objective of our study was to approach these gaps. A previous study by Kanoh et al. (2012), using data from the Longitudinal Survey of Newborns in the 21st Century, reported a positive hazard risk between parental smoking and childhood asthma incidence. However, they did not focus on the gaps (i.e., did not use

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corresponding age categories) and did not use severity of asthma as an outcome. Furthermore, although parents are encouraged to smoke outdoors or not to smoke (Committee on Substance Abuse, 2001), the difference in the contribution of these parental smoking behaviors to the risk reduction of asthma among their children has not been sufficiently evaluated (Blackburn et al., 2003; Blizzard et al., 2003; Leung et al., 2004). Thus, the main objective of this study was to assess whether and how parental smoking behaviors, combined with indoor smoking status at home, were associated with the development and severity of childhood asthma from very young ages to 8 years old, using data from a large nationally representative birth cohort study.

2. Methods

2.1. Study population

The data used for this study were taken from the Longitudinal Survey of Newborns in the 21st Century which was conducted by the Japanese Ministry of Health, Labour, and Welfare from 2001 to 2009 (Ministry of Health, Labour and Welfare, 2013). The study sample included all infants born in Japan during the periods January 10–17, 2001, and July 10–17, 2001 using the national birth record (n = 53,575). Questionnaires were mailed when the infants were 0.5 years of age. The total number of respondents was 47,015 (response rate, 87.8%). Follow-up surveys were conducted at the ages of 1.5, 2.5, 3.5, 4.5, 5.5, 7, and 8 years, among the remaining subjects who responded at least once to two recent, consecutive surveys. Respondent numbers (%) of each survey were: 43,925 (82.0%), 42,812 (79.9%), 41,559 (77.6%), 39,817 (74.3%), 38,537 (71.9%), 36,785 (68.7%) and 36,136 (67.4%), respectively. Details of the study are available elsewhere (Fujiwara et al., 2013; Kaneita et al., 2006; Ministry of Health, Labour and Welfare, 2013; Yamakawa et al., 2013).

The analysis was limited to cases where both parents lived together with the subjects at the time of the first survey (n = 45,903). Of these, subjects without information on parental age (n = 181), parental smoking (n = 1141) or birth weight (n = 13) were excluded from the analyses. As assumed interchangeability of twins and other multiple birth children, all such subjects were excluded (n = 937), leaving a total of 43,653 subjects (see Supplementary Fig. 1[1]). Data were used with permission from the Japanese Ministry of Health, Labour, and Welfare. The analyses of national survey data were considered to be exempt from the need for ethical review according to the Epidemiological Research Guidelines.

2.2. Outcomes

We defined two self-reported dichotomous outcomes: (1) physician visit for asthma as an indicator for prevalence (including induction and/or persistence) of current asthma ("Has the child been seen by a physician because of asthma in the last year?") and (2) hospitalization for asthma as an indicator for prevalence of current severe asthma ("Has the child been hospitalized for asthma in the last year?"). Data for each of these outcomes were obtained at the ages of 1.5, 2.5, 3.5, 4.5, 5.5, 7, and 8 years (2nd to 8th surveys), and pertain to the previous 12 months (outcomes on 7th survey only pertain to 18 months). Because the phenotype of asthma is potentially different according to age at diagnosis (for example, early-onset transient or persistent asthma; Burke et al., 2012; Horner and Strunk, 2007), we divided the outcomes into the following three age categories according to previous studies (Burke et al., 2012; USDHHS, 2006): children aged 0.5 < –2.5 years (data from 2nd to 3rd surveys), those aged 2.5 < –4.5 years (4th to 5th surveys), and those aged 4.5 < –8 years (6th to 8th surveys).

2.3. Parental smoking

Parental smoking status data were collected in the first (i.e., 0.5 years old) and 5th survey. Only the first survey data were used because it was simpler and led to a conservative estimate (additionally explained in the limitation section below). Maternal and paternal smoking behavior at age 0.5 years was categorized into 3 levels: noncurrent smoker, current smoker who did not smoke indoors at home (i.e., outdoor smoker), and current smoker who smoked indoors at home (i.e., indoor smoker). Among all nine (3 × 3) combinations of parental smoking (Table 1), major five categories which included more than 1000 eligible subjects were used as exposure level variables: i.e., (i) no parental smoking (n = 15,649, 35.9%), (ii) maternal non-smoking and paternal outdoor smoking (n = 9619, 22.0%), (iii) maternal non-smoking and paternal indoor smoking (n = 11,043, 25.3%), (iv) parental outdoor smoking (n = 1685, 3.9%), and (v) parental indoor smoking (n = 4772, 10.9%). Because analyses were limited to these exposure populations, the number of remaining baseline subjects was 42,768 (98.0%).

Table 1
Number (%) of parental smoking combinations at baseline.

	Father		
	No smoking	Outdoor smoking	Indoor smoking
Mother			
No smoking	15,649 (35.9)[a]	9619 (22.0)[a]	11,043 (25.3)[a]
Outdoor smoking	207 (0.5)	1685 (3.9)[a]	199 (0.5)
Indoor smoking	330 (0.8)	149 (0.3)	4772 (10.9)[a]

[a] Used in the study.

The parental smoking variable was used in two ways. First, no parental smoking was used as a reference category to examine whether children who had been exposed to SHS were more likely to develop outcomes than those not exposed. This included comparisons not only between parental smoking versus no parental smoking but also between paternal smoking with nonsmoking mother versus no parental smoking, which provided a tobacco control perspective (Tabuchi et al., 2013). Second, parental indoor smoking was used as a reference category to examine whether children who had lower SHS exposure (due to outdoor smoking or maternal nonsmoking) have a lower association with outcomes than those who are exposed to parental indoor smoking. This was based on a clinical perspective, assessing a quasi-intervention effect on child's asthma of non-indoor smoking. However, this was an observational study and a full intervention is necessary to test the hypothesis (Parsons et al., 2010; Tabuchi et al., 2013).

2.4. Statistical analyses

The basic characteristics were tabulated according to the parental smoking categories. A chi-square test was used to compare the difference in subject characteristics between the parental smoking categories. The prevalence of outcomes within each outcome age category (i.e., within defined time durations) was calculated.

A multivariate logistic regression was used to estimate adjusted odds ratios (ORs) with 95% confidence intervals (CIs) for each outcome according to each age category. Subjects who responded at least once within each outcome age category were analyzed. In line with previous studies (Kaneita et al., 2006; USDHHS, 2006; Vork et al., 2007), we used child's sex, maternal age, paternal age, low birthweight (yes or no), number of siblings (0 or ≥1), breastfeeding (exclusive or not), child's atopic dermatitis history (at least one physician visit for atopic dermatitis followed before the age of 8 years, yes or no), residential population density (tertiles), and equivalent household income (quartiles) as potential confounders in the analyses. Although characteristics of subjects who participated in the study differ significantly from those who did not respond at follow-up survey (see Supplementary Table 1[2]), the non-response was sufficiently explained by above covariates in the logistic model. Therefore, the complete-case analysis in the logistic model can be regarded as appropriate with assumption that data are missing at random (Graham, 2012). Details of covariates and non-response analysis are available elsewhere[3] (Fujiwara et al., 2013; Little et al., 2012).

To estimate population impact, population attributable risk percent (PARP) was calculated based on the relative risk (RR) of asthma associated with exposure to parental smoking, which was approximated from the adjusted ORs (Rothman et al., 2008) we estimated, and the prevalence of each combination of smoking statuses of parents in the total Japanese population, derived from the data we used in this study (P; see Table 1), using the following formula (Inoue et al., 2012; Tamakoshi et al., 2009):

$$\mathrm{PARP}_i = P_i \times \frac{(\mathrm{RR}_i - \mathrm{RR}_{\mathrm{no}})}{(1 \times \mathrm{RR}_{\mathrm{no}} + P_i \times (\mathrm{RR}_i - \mathrm{RR}_{\mathrm{no}}))}$$

where i denotes parental smoking (indoor or outdoor smoking) and no denotes no parental smoking. Population attributable fractions (PAF), the estimated number of preventable cases of asthma in children, were also calculated by multiplying PARP and the expected number of children with asthma in all of Japan. The latter value was obtained by multiplying the asthma incidence rate in this study and the population size reported from the Japanese national census in 2000 (Ministry of Internal Affairs and Communications, 2000). Additional methodology details are available in the supplementary material[4].

Probability values for statistical tests were two-tailed, and P < 0.05 was regarded as statistically significant. All statistical analyses were carried out using the SAS statistical package version 9.2 (SAS Institute, Inc., Cary, NC, USA).

[1] Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi:10.1016/j.drugalcdep.2014.12.001.

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