"warning labels," but vague phrases such as "for you" are still being used, which should be removed.

Moreover, the phrase, "try not to bother those around you when smoking," is permitted, but it would be better if the phrase, "do not bother those around you when smoking," was used.

Of the eight types of label language required under Article 36 of the Order for the Enforcement of the Tobacco Business Act, there are many that address the harmfulness of tobacco. However, regarding its addictiveness, the language is inadequate. Examples of such inadequate language include, "although different people will have different results, nicotine can cause people to be addicted to smoking," and "the harmful health effects and addictiveness may be worse among minors." Yet, one of the reasons minors reach for their first cigarette is that they underestimate their addictiveness. In other words, they assume they will be capable of quitting at any time. Of course, conveying the proper information regarding tobacco's addictive nature is necessary, but more language regarding tobacco's addictive nature must be included on the warning labels on cigarette packs themselves.

The imposition of the duty to put warning labels on tobacco products raises issues of economic freedom. However, considering the health of smokers and the harmful health effects of secondhand smoking on others, to prevent minors from smoking, such imposition should be constitutionally permissible. Further, as proper decisions cannot be made without having accurate information on which to base them, one could say that smokers were deprived of their actual right to choose by their lack of accurate information[28]. Additionally, a major reason that consumers are not given accurate information on the harmful and addictive properties of tobacco is that the entire tobacco industry is united in manipulating tobacco information to hide the truth from consumers[29].

28) *See* Yoshio Isayama, 1999, *The Modern Tobacco War (Gendai Tabako Senso)*, Iwanami Shoten, p. 12.ff.

29) See Philip J Hilts, 1996, *Smokescreen: The Truth behind the Tobacco Industry Cover-up*, Addison Wesley Reading. This book is based on the accounts of a whistleblower from inside the tobacco industry and uses secret internal documents. This book reveals that tobacco companies have been aware of the addictiveness of tobacco and the dangers of nicotine for quite some time, and have manipulated nicotine concentrations (by increasing them) to get smokers addicted.

Additionally, *see* ASH (Action on Smoking and Health), 1998, *Tobacco Explained*, available at http://www.ash.org.uk/files/documents/ASH_599.pdf (last visited April 12, 2014). This book is a compilation of internal document evidence from U.S. and European tobacco companies which was made public during tobacco litigation in the United States by the British NGO, ASH. It was compiled by ASH and can be viewed on their webpage, available at http://www.ash.org.uk/information/tobacco-industry/tobaccochronology. The webpage for the World Health Organization (WHO)'s World No Tobacco Day 2001 contains "Tobacco Explained", available at http://www.who.int/tobacco/media/en/TobaccoExplained.pdf#search='TobaccoExplainedwho. It was intended to expose the immoral business strategies of the tobacco industry to the world (last visited April 14, 2014). If you read these internal documents, you will understand that the tobacco industry was clearly aware of the harms of tobacco use. Further, for a robust collection of internal documents from the tobacco industry, visit the University of California, San Francisco (UCSF)'s library website, available at http://www.library.ucsf.edu/tobacco (last visited April 14, 2014). Also, *see* the Journal of the Japan Society for Tobacco

Hence, to provide consumers with accurate information on the harmful and addictive properties of tobacco, it is absolutely necessary that the law impose a duty regarding the language used to label cigarette packs.

2. Regulating Tobacco Product Names

Japan's *Policy on Advertising by Tobacco Manufacturers*, formulated in accordance with Article 40.2 of the Tobacco Business Act, authorizes the use of words such as "low tar," "light," "ultra light," and "mild" on cigarette packs, as long as "to avoid misunderstandings among consumers, such packaging contains clear language to the effect that the cigarettes contained within are not less harmful than other cigarettes." However, descriptive adjectives such as "mild" and "light" provide the false impression to consumers that certain products cause fewer health risks than other products, and therefore such descriptive adjectives should be banned from tobacco product names. Ultimately, names with descriptive adjectives such as "mild" and "light," in the context of product selection, constitutes a failure to provide accurate information.

Therefore, words such as "low tar," "light," "ultra light," "mild," and any other language that are likely to cause misunderstanding among consumers regarding the harmful relationship between cigarettes and health should not be used in tobacco product names.

The WHO Framework Convention on Tobacco Control requires that signatory nations ensure that the packaging and labels on cigarette packs sold within their borders do not promote the sale of tobacco products through the use of means likely to cause misunderstanding regarding the properties, harmful health effects, dangers, and/or emissions of cigarettes (e.g., labels with descriptive adjectives such as "low tar," "light," and "ultra light") (Article 11.1).

On August 8, 2012, the JT Group announced that it was globally renaming its Mild Seven brand to "Mevius." However, the various individual Mevius products still have names such as "Mevius Light," "Mevius Super Light," and "Mevius Extra Light." Further, JT's other cigarette products have names such as "Seven Star," "Peace," and "Hope." However, according to the WHO Framework Convention on Tobacco Control, product names such as "Mevius Light," "Mevius Super Light," and "Mevius Extra Light" can be considered violations of the announcement. It should also be considered whether product names such as "Seven Star," "Peace," and "Hope" constitute "language likely to cause misunderstanding in consumers regarding the relationship between tobacco consumption and health."

Control, 2000, *Judging the Tobacco Industry: Japan's Tobacco War (Tabako Sangyo wo Sabaku, Nihon Tabako Senso)*, Jissensha.

3. Induction through Economic Methods

Smokers also generate various societal costs by smoking. Since smokers create a heavier economic burden on society than nonsmokers, principles of societal fairness are highlighted. Smokers may present the argument that they pay the tobacco tax. However, as the estimated societal and national healthcare costs of tobacco use are approximately JPY 7 trillion per year, these costs are not fully covered by the tobacco tax. Accordingly, it is necessary to introduce economic methods (economic disincentives for smokers and economic incentives for nonsmokers). Possible economic methods include 1) fire insurance, 2) life insurance, 3) automobile insurance, 4) rental and hotel fees, and 5) surcharges for smoking seats at eating and drinking establishments.

4. Nonsmoking Support Measures

Smokers underestimate the danger of becoming addicted and find themselves unable to quit after smoking for a certain period. Once addicted, warning labels and economic incentives do little to help smokers quit. Accordingly, mechanisms are needed to support the efforts of those who are trying to quit smoking (smoking cessation support measures). As a practical matter, this could include funding for smoking cessation treatment by physicians, and private sectors measures such as providing a nonsmoking bonus to nonsmoking workers and allowing workers to use sick leave to visit smoking cessation clinics.

Incidentally, smoking cessation treatment has been covered by health insurance since April 2006, if it meets certain conditions. However, health insurance coverage is limited to outpatient treatments, and, even when receiving such outpatient treatments, patients (former smokers) must pass a four-factor test to be eligible for coverage. These four factors require that patients 1) themselves wish to quit smoking (not family members), 2) test positive for at least five factors in a nicotine dependence test, 3) must score at least a 200 based on the formula of (number of years smoked) x (cigarettes smoked per day), and 4) are capable of consenting to the treatment and treatment-related documents. Only those who satisfy all of these requirements may receive outpatient smoking cessation treatment (Notice from the Director of the Medical Care Division of the Ministry of Health, Labor and Welfare's Insurance Bureau, "Regarding Issues to Consider in Implementation of System for Calculating Reimbursement of Medical Fees")[30] (March 5, 2008, *Hoihatsu* No. 0305001). In addition, treatment is only covered for the first five treatments. Any further treatment is not covered (at patient's expense). However, in the future, in addition to enabling health insurance coverage for more than outpatient smoking cessation care, the conditions for receiving outpatient care should be relaxed, more patients should be approved, and the number of covered treatments should be increased. Furthermore, to

30) *See* the website of the Ministry of Health, Labor and Welfare, available at http://www.mhlw.go.jp/topics/2008/03/dl/tp0305-1d.pdf (last visited April 14, 2014).

secure the necessary funding, the tobacco tax must be substantially increased.

V Dramatic Reform of Tobacco Regulation

Parts II through IV dealt with concrete tobacco measures that can be taken given Japan's current legal system. However, the future legal challenges facing Japan in the realm of tobacco regulation must include dramatic reforms in addition to more actual measures. Accordingly, Part V will not be premised on the existing legal system; instead, to explore ideal solutions, the need for more dramatic reforms will be considered.

1. Repeal of the Tobacco Business Act

The Tobacco Business Act has no stated purpose of protecting the health of the nation's citizens. Rather, the statute is intended to develop the tobacco industry, secure funds for the government, and contribute to the national economy, irrespective of any harm to the health of the national citizenry. Lacking such protective purpose, the statute serves as a means for accumulating funds through an aggressive policy of tobacco industry expansion at the expense of national health. However, the purposes of the Tobacco Business Act are irreconcilable with the promotion of the general health and welfare of a nation's citizens. Accordingly, as long as the Tobacco Business Act exists, one cannot expect the country to achieve a policy that will promote the general health and welfare of the nation's citizens. To achieve such a policy, the Tobacco Business Act must first be repealed.

2. Establishment of a Comprehensive Tobacco Control Law

The current Tobacco Business Act should be repealed and a comprehensive Tobacco Control Law enacted in its place. The current Tobacco Business Act actually regulates tobacco in many ways, but usually laws are structured so that 1) they define the law's policy purpose in the first section of the law, and 2) starting with Article 2 of the law in question, they establish means for achieving that policy purpose. If that were the case, the provisions of the current Tobacco Business Act, starting with Article 2, would be to develop the tobacco industry, secure government revenue, and promote the national economy. Therefore, amending the law from the perspective of protecting the health of the nation's citizens would raise several problems. Accordingly, the current Tobacco Business Act should be repealed and replaced with a comprehensive Tobacco Control Law formally devoted to protecting the health of the nation's citizens.

3. Changing Jurisdiction from the Ministry of Finance to the Ministry of Health, Labor and Welfare

Not a single aspect of the Tobacco Business Act regulates the tobacco business in the interest of protecting health. One reason for this is that the government ministry with exclusive jurisdiction over supervision of the tobacco business, the Ministry of Finance,

obtains tax revenue from tobacco.

Yet, the tobacco problem is a health problem and supervision of the tobacco industry should be conducted by the Ministry of Health Labor and Welfare, not the Ministry of Finance. Accordingly, jurisdiction over tobacco supervision should be transferred from the Ministry of Finance to the Ministry of Health, Labor and Welfare. If such a transfer is difficult to implement, at the very least, the Ministry of Health, Labor and Welfare should have joint jurisdiction with the Ministry of Finance. Hence, at least, the legal system should be restructured to give the Ministry of Health, Labor and Welfare substantive authority over tobacco control by, at the very least, giving the Ministry of Health, Labor and Welfare joint jurisdiction over tobacco supervision and the previously proposed Tobacco Control Law.

4. Restrictions on Shares of JT Held by the National Government

One of the reasons for Japan's slow progress on tobacco issues is that the national government owns half of the shares of JT. However, the party previously in power, the Democratic Party of Japan, when compared with the Liberal Democratic Party, had fewer ties to the Ministry of Finance and JT, and it was expected that they would implement reforms to ban smoking. Then, the Liberal Democratic Party regained power, but the national government should not own shares of JT[31].

5. Enacting a Comprehensive Secondhand Smoke (passive exposure to tobacco smoke, passive smoke) Prevention Law

(A Law Restricting Smoking Areas)

As previously discussed, if Article 25 of the Health Promotion Act and Article 68.2 of the Industrial Safety and Health Act could be amended to institute a complete ban on indoor smoking, one could expect a significant drop in indoor smoking and concomitant increase in the prevention of secondhand smoking (passive exposure to tobacco smoke, passive smoke). Even if it were to be amended, the laws would still not affect outdoor smoking. However, currently, the prospects for amending the Articles and regulating outdoor street smoking are equally bleak.

If the previously mentioned amendments to Article 25 of the Health Promotion Act and Article 68.2 of the Industrial Safety and Health Act, together with regulations of street smoking, were to be enacted, it would result in actual restrictions on areas where smoking is permitted. However, the various laws would still have limited scope. I predict that even if these laws were to be enacted, nonsmokers would still be unable to avoid secondhand smoke (passive exposure to tobacco smoke, passive smoke) in their daily lives.

31) *See* Satoshi Kitamura, 2009, "Towards Preventing Secondhand Smoking: In Order to Not Lag Behind the Rest of the World (Judo Kitsuen Boshi ni mukete: Sekai no Choryu ni Okurenai tameni), *Japanese Journal of Tobacco Control*, Vol. 4, No. 5, p. 117.ff.

Accordingly, to prevent secondhand smoking (passive exposure to tobacco smoke, passive smoke) in the future, comprehensive laws and ordinances restricting smoking areas are desirable[32].

6. Repudiating the Right to Smoke

Is there an actual right to smoke?

Tobacco companies entice minors into nicotine dependence and use them as profit-generating customers for a prolonged period, devising clever and ingenious branding strategies. In reality, the decisions to start smoking and to continue smoking are affected by the addictive nature of tobacco and by the various influences of the tobacco companies. Smoking is not simply a matter of free personal choice[33]. Notably, tobacco is uniquely addictive, making it extremely difficult for people to stop using once consumed regularly for a certain time. Accordingly, it becomes exceedingly difficult for smokers to control themselves and exercise true free will.

Therefore, for smokers who are trying to quit, but cannot, we should not speak of a "right to smoke." It is better to proceed as though no such right exists and to build a consensus to repudiate the so-called "right to smoke." Since April 2006, smoking cessation treatment, provided it meets certain conditions, has been covered by health insurance. Such treatment is premised on the belief that does not consider smoking to be a mere lifestyle choice, but a life-threatening disease known as nicotine dependence, which requires medical treatment. In other words, such a belief repudiates the very concept of a right to smoke.

7. Complete Prohibition of the Sale of Tobacco

Smokers do not smoke as an exercise of free will. Their bodies have become pathetically dependent on nicotine. Tobacco companies such as JT profit by making smokers nicotine dependent. This business model is similar to that employed by organized criminal organizations which inject people with methamphetamines to get them addicted and are forced to buy their product at a high price. Accordingly, we should consider the argument that we should outlaw the sale of tobacco completely and create an environment wherein tobacco is not available even if smokers wish to smoke, instead of simply telling smokers to stop smoking[34]. Eventually, despite the fact that the dangers of tobacco are

32) In Japan also, on November 21, 1978, the Committee of Legislators for Securing Rights for Nonsmokers submitted a bill entitled the "Act Regarding Spatial Restrictions on Smoking." Further, in March 2010, the Japan Society for Tobacco Control drafted a bill entitled, "Act for Preventing Secondhand Smoking in the Workplace and Other Public Spaces," and petitioned the ruling administration (including the Prime Minister and Minister of Health, Labor and Welfare) to enact it.

33) *See* Slade, *supra* note 19, pp. 78-83.

34) *See* Abe, *supra* note 1, p. 44, Takao Tanase. "U.S. Tobacco Litigation and Tobacco Policy: The Gap between the Right to Smoke and Smoking Prohibition (Beikoku Tabako Sosho no Tenkai to Tabako Seisaku: Kitsuen

visible to all, the simple question remains, why does the national government permit the sale of tobacco? It is difficult to answer this question accurately. Yet, accurate information regarding tobacco is not provided, and even when smokers do appreciate the risks of smoking and try to quit, the vast majority of them fail. Therefore, regardless of concepts like "the right" to smoke, for smokers who appreciate the risks of smoking and yet still cannot quit, we should consider forcefully urging smokers to quit by using government regulation to absolutely prohibit the sale of tobacco[35].

However, tobacco is addictive because once consumed for a certain period, it becomes exceedingly difficult to discontinue. It is incorrect to state that smokers are exercising absolute control and their freedom to choose to smoke tobacco. Due to tobacco's addictive nature and the way it impairs the ability of smokers to make decisions for themselves, we should adopt a paternalistic attitude toward this issue and strive to preserve a bare minimum of societal morals. The possession and sale of tobacco should be strictly regulated under the Narcotics and Psychotropic Control Act similar to methamphetamines and opium.

Personally, I believe that if you really care about others, then you should oppose the sale of poisonous, addictive substances which cause people to suffer. You should want JT to immediately withdraw from the tobacco industry. If the purpose of a business is to provide more of the things people want—love, money, freedom, and time—or to remove the things people do not want—stress, conflict, discord, and anxiety— (or to do both),then there certainly is a right to conduct business[36]. However, tobacco steals love, money, freedom, and time from smokers (and nonsmokers as well) on the one hand, and burdens society as a whole with stress, conflict, discord, and anxiety (when smokers think they are relieving stress by smoking, they are really just relieving nicotine cravings which are caused by nicotine dependence) on the other. With the aforementioned characteristics, this peculiar consumer good has 100 demerits and zero merits. We should be moving toward outright a prohibition of this consumer product in the future.

8. Reform of the Tobacco Manufacturing, Marketing, and Sales System

The tobacco industry is powerful, and has the support of labor unions, farmers, shippers, related businesses, and smokers. It also conducts lobbying activities. Although the various tobacco companies are mutually competitive, they will be united in their opposition to anti-smoking activities and devise a means of opposing any policy aimed at reducing smoking areas and will continue to market their product.

The tobacco industry, which deliberately created the market for their product, the

Jiyu to Kitsuen Kinshi to no Hazama)", Takao Tanase ed., 2000, *Sociolegal Study of Tobacco Litigation (Tabako Sosho no Hoshakaigaku)*, Sekaishisosha, p. 3.ff.

35) *See* Tanase, *supra* note 34, p. 3.ff.

36) *See* Chris Guilleabeau (Translated by Naoyuki Honda), 2013, *The Hundred Dollar Startup*, Asuka Shinsha, p. 57.ff.

practice and custom of smoking itself, through aggressive sales and marketing, must absolutely be reformed; however, the government which should be regulating such sales and marketing practices (particularly the Ministry of Finance) is itself an interested party in the survival and development of the tobacco industry, making the prospects for effective tobacco regulation unlikely. The legal system governing the manufacture, marketing, and sale of tobacco, which perpetuates the custom of smoking and resultant health damage, must be changed[37]. As stated previously, it is important to reduce the number of smokers, but to do that, it will be necessary to achieve structural changes in the current manufacturing, marketing, and sales system in which the national government is united with the tobacco industry[38].

VI Conclusion

As mentioned previously, the so-called "right to smoke," as a natural right, is intrinsically limited to the extent to which it does not harm the health or survival of others. Furthermore, based on the WHO Framework Convention on Tobacco Control, measures must be taken to prevent secondhand smoking (passive exposure to tobacco smoke, passive smoke), prevent smoking by minors, and reduce the number of current smokers. I also believe that more dramatic reforms are needed. For the current Japanese society, stronger governmental regulation of tobacco is an absolute necessity. If one truly understands the addictive and harmful nature of tobacco, then one should understand the need and desirability of a fair legal system that can guide our society away from tobacco usage as quickly as possible. The day when that happens cannot come too soon.

(Appendix)

This study is the compilation of the results of research conducted from FY 2008–2011 under scientific research grants from the Japan Society for the Promotion of Science (B) under the research topic "Law and Policy of Tobacco Regulation" (Topic Number 20730007). It also contains the results of research conducted under an FY 2013–2014 Ministry of Health, Labor and Welfare science research grant (Project for General Research on Measures Against Circulatory Conditions, Diabetes and Lifestyle-Related Diseases) research topic "General Research on Countermeasures Based on the WHO Framework Convention on Tobacco Control" (Representative: Dr. Masakazu Nakamura) (Topic Number H25 *Junkanki To* (*Seishu*) *Ippan* 010).

37) *See* Iwao Sato, 2000, "Changes in Tobacco Litigation and the Identity of the Movement (Tabako Sosho no Henyo to Undo no Identity), Tanase ed., *supra* note 34, p. 95.ff.

38) *See* Simon Chapman, 2007, *Public Health Advocacy and Tobacco Control*, Blackwell Publishing, pp. 172-197.

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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):

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

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.

[2] 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.

[3] 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.

[4] 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.

3. Results

The baseline characteristics of study subjects according to the parental smoking categories are shown in Table 2. All covariates except for child's sex show statistically significant differences; i.e., maternal smoking groups showed lower birthweight, less exclusive breastfeeding, younger parental age, and lower equivalent household income.

The prevalence of outcomes within defined time durations are shown in Table 3 (numbers in Supplementary Table 2[5]). For example, during the period 0.5 < –2.5 years, the prevalence of physician visits and hospitalization for asthma was 6.7% and 1.7%, respectively.

Table 4 show adjusted ORs with 95% CIs for asthma according to parental smoking categories. Corresponding PARP and PAF are shown in Supplementary Tables 3 and 4[6]. From the tobacco control perspective, both parents smoking indoors at home had 31%, 18% and 19% higher odds for physician visits for asthma than no parental smoking, at ages 0.5 < –2.5 years, 2.5 < –4.5 years and 4.5 < –8 years, respectively. Similarly, both parents smoking indoors had 54%, 43% and 72% higher odds for hospitalization for asthma, respectively. Maternal non-smoking and paternal smoking (both indoor and outdoor) showed non-significant but positive ORs for asthma compared with no paternal smoking, except for one category (i.e., maternal non-smoking and paternal indoor smoking for hospitalization at age 0.5 < –2.5 years).

The percentage (PARP) attributable to both parents smoking indoors at home ranged from 1.9% to 3.2% for physician visits and from 4.5% to 7.3% for hospitalization. The number (PAF) attributable to both parents smoking indoors at home was 7630 (95% CI: 3420, 11, 840) for physician visits and 3330 (1160, 5510) for hospitalization at age 0.5 < –2.5 years. In total, PARP of total parental smoking ranged from 3.9% (95% CI: 0.7, 7.0) to 7.8% (3.9, 11.8) for physician visits and from 8.3% (2.2, 14.3) to 18.2% (7.7, 28.8) for hospitalization.

From the clinical perspective, compared with both parents smoking indoors at home, both parents smoking outdoors at home showed non-significant but decreased odds for asthma, ranging from 0.81 to 0.94 for physician visits and from 0.74 to 0.99 for hospitalization. Maternal non-smoking and paternal smoking (both indoor and outdoor) had significantly lower odds for asthma than parents smoking indoors at age 0.5 < –2.5 years with some non-significant results in other age categories. The percentage attributable to the risk reduction from parents smoking indoors (PARP) was 3.7% (95% CI: –2.1%, 9.6%) for parents smoking outdoors, and 5.6% (95% CI: 1.9%, 9.3%) for maternal non-smoking and paternal indoor smoking, for hospitalization at age 0.5 < –2.5 years.

4. Discussion

Using Japanese nationally representative large cohort data, we found an elevated association with asthma among the children who were exposed to SHS from parental smoking. This is consistent with the findings of preceding studies in Japan and other areas (USDHHS, 2006). The primary new findings of the present study were, (i) compared to children whose parents do not smoke tobacco, children whose parents smoke indoors at home showed increased association with having asthma in terms of both outpatient visits and hospitalization, with a maximum 70% odds increase; (ii) the association with asthma for children whose fathers but not mothers smoke, either indoors or outdoors, did not noticeably increase compared to those with non-smoking parents; however, when the mothers also smoked, the association noticeably increased; and (iii) parental indoor smoking at home is a stronger risk factor for children's asthma than outdoor smoking but the differences in odds between indoor and outdoor smoking were not statistically remarkable, suggesting that outdoor smoking at home may not significantly prevent children's asthma.

To date, there is insufficient evidence as to whether parental outdoor smoking at home (i.e., hypothesized smoke-free homes) or maternal postnatal smoking cessation decreases children's asthma compared with parental indoor smoking (Blizzard et al., 2003; Carlsson et al., 2013; Emmons et al., 2001). In this cohort study, parental outdoor smoking at home showed a decreased association with childhood asthma compared with parental indoor smoking. However, the decrease was not significant. This non-significance may indicate the limited impact of parental outdoor smoking on childhood asthma, although both maternal and paternal outdoor smoking at home might be an alternative option to reduce harm for children (Blizzard et al., 2003). This result may be due to the relatively small sample size (for outdoor smoking parents), or inappropriate outdoor smoking habits which did not reduce risk compared with indoor smoking (e.g., near-door smoking; Blizzard et al., 2003; Chapman, 2007; Halterman et al., 2007).

Furthermore, the results from maternal non-smoking and paternal indoor or outdoor smoking versus parental indoor smoking suggest that maternal smoking cessation may decrease the association with childhood asthma, especially for hospitalization at 0.5 < –2.5 years (35% reduction). This may be because children spend more time at home with the mother than the father. For example, some mothers live apart from their partner during the perinatal and child-care-leave period in Japan (Ohga et al., 2005). Although a randomized intervention study is required to validate this hypothesis, such an intervention may be difficult because of ethical and sample size problems (Carlsson et al., 2013; Emmons et al., 2001). Therefore, this observational study may provide the best evidence and policy implications in this field of research. Thus, this study suggests that parents, especially mothers, who smoke should be strongly supported to stop smoking (Committee on Substance Abuse, 2001).

4.1. Population impacts

Our results confirmed the results from previous studies (Burke et al., 2012), although some adjusted estimates of the association were not significant. If our results indicate truly causal associations, our estimation of population attributable risks can be interpreted as that if all parents in Japan quit smoking, hospitalization of children due to asthma could be reduced by 8.3%, 9.3% and 18.2%, respectively, for the three age ranges 0.5 < –2.5, 2.5 < –4.5 and 4.5 < –8 years. If parents smoke outdoors rather than indoors, the reductions would be 3.7%, 0.8% and 0.1%, respectively. In summary, the numbers of excess cases of hospitalizations due to asthma and the number of preventable pediatric asthma hospitalizations, were simulated to be 4970, 4950 and 10,940, respectively. Because these numbers are not small, there is room to prevent childhood asthma by advancing tobacco control measures including parental smoking cessation support.

4.2. Magnitude and persistence of the risk

Exposure to SHS in early life had a substantial association with both physician visits and hospitalization for asthma. The magnitude of the association with hospitalization was relatively higher than that with physician visits. Whether smoking causes asthma or only exacerbates latent and controlled symptoms remains controversial

[5] 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.

[6] 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.

Table 2
Baseline characteristics of study subjects according to parental smoking categories at baseline, $n = 42{,}768$.

Characteristics	Parental smoking categories					
	No parental smoking ($n = 15{,}649$)	No maternal smoking and parental non-indoor smoking ($n = 9619$)	No maternal smoking and paternal indoor smoking ($n = 11{,}043$)	Parental non-indoor smoking ($n = 1685$)	Parental indoor smoking ($n = 4772$)	*P* value[a]
Child's male gender (%)	51.9	51.7	52.1	50.0	52.9	0.3515
Atopy (%)	13.7	13.7	13.2	11.1	11.2	<0.0001
Birth weight, mean (SD)	3061 (407)	3047 (420)	3071 (411)	3009 (419)	3020 (425)	
Low birth weight (%)	6.6	7.6	6.6	8.7	8.3	<0.0001
Exclusive breastfeeding (yes) (%)	25.2	23.8	22.7	10.0	10.7	<0.0001
Number of sibling						<0.0001
1 Or more (%)	49.9	43.3	56.7	38.9	55.6	
Population dense						<0.0001
Low (%)	19.1	22.0	23.1	23.7	24.6	
Middle (%)	34.2	35.7	35.7	31.2	31.2	
High (%)	46.7	42.3	41.2	45.0	44.2	
Mother's age, mean (SD)	31.1 (4.2)	30.0 (4.3)	30.0 (4.4)	28.1 (4.6)	28.2 (4.6)	<0.0001
16–19 Years-old (%)	0.2	0.5	0.6	1.8	1.6	
20–29 Years-old (%)	36.0	45.5	45.9	61.7	61.4	
30–39 Years-old (%)	61.1	52.4	51.5	35.5	35.6	
40–49 Years-old (%)	2.6	1.6	2.0	1.0	1.4	
Father's age, mean (SD)	33.4 (5.2)	31.9 (5.3)	32.2 (5.6)	29.8 (5.9)	30.2 (5.9)	<0.0001
18–19 Years-old (%)	0.1	0.2	0.2	1.0	0.7	
20–29 Years-old (%)	22.5	34.1	33.1	52.3	49.6	
30–39 Years-old (%)	65.8	57.6	56.8	41.2	43.0	
40–49 Years-old (%)	11.0	7.7	9.1	4.8	5.8	
50–71 Years-old (%)	0.6	0.4	0.8	0.8	0.9	
Equivalent household income						<0.0001
First quartile (lowest) (%)	16.5	21.3	27.1	33.2	38.3	
Second quartile (%)	21.0	22.8	26.2	24.6	25.5	
Third quartile (%)	25.1	24.0	21.9	18.5	17.1	
Forth quartile (highest) (%)	31.7	25.8	18.2	16.1	10.5	
Missing (%)	5.7	6.2	6.7	7.5	8.5	

Abbreviation: SD, standard deviation.
Missing value number was 187 for breastfeeding, and 2755 for equivalent household income.
[a] *P* values calculated from chi-square test using categorical data.

(Baena-Cagnani et al., 2009). SHS exposure due to parental smoking may exacerbate rather than induce children's asthma, potentially because tobacco smoking is associated with a reduction in asthma medication efficacy (Chaudhuri et al., 2003).

The impact of SHS on asthma remained significant even among children aged 4.5 < –8 years, regardless of the potential underestimation due to parental smoking change. SHS exposure in early childhood may have a long-lasting or cumulative impact on the induction and the severity of asthma, through biological pathways in children's growth such as premature lung (Alati et al., 2006; Royal College of Physicians, 2010).

4.3. Limitations and strengths

There are several limitations to this study. First, the data used in the study were based on self-reported smoking status and medical care for childhood asthma, which may lead to misclassification (Miller, 2001), although previous studies showed that self-reported SHS exposure correlated well with biomarker concentrations (Brunekreef et al., 2000; Johansson et al., 2005). Use of the simple question of "Do you smoke indoors at home?", could lead to misclassification of indoor or outdoor smoking status, resulting in underestimation. However, parents showed a high match percentage in response to the question (i.e., both indoor and outdoor, see Table 1) and only the matched parents were used in the analyses. Lack of information on smoking of other household members and self-reported parental smoking with no consideration for smoking status after first survey may lead to underestimation. Furthermore, our questions did not directly measure parental smoking in the presence of their children. This also may lead to an underestimation, explaining the higher likelihood of asthma attacks due to smoking by mothers than fathers. Parental smoking data were also collected in the 5th (i.e. 4.5 years old) survey. Of mothers who did not smoke when the child was 0.5 years, 3.0% of mothers in "no parental smoking" at baseline smoked when the child was 4.5 years. Of mothers who smoked when the child was 0.5 years, 84.5% of mothers in "both parental indoor smoking" at baseline continued to smoke when the child was 4.5 years (77.9% of those

Table 3
Number and prevalence[a] of outcomes within defined time duration.

Age groups (defined time duration)	No.[b]	Prevalence of physician visits for asthma		Prevalence of hospitalization for asthma	
		No.	%	No.	%
0.5 < –2.5 Years (2 years)	41,182	2746	6.67	682	1.66
2.5 < –4.5 Years (2 years)	38,977	3525	9.04	571	1.46
4.5 < –8 Years (3.5 years)	36,483	4361	11.95	361	0.99

Abbreviation: No., number.
[a] Each individual was counted once even if he/she suffered more than once within the defined time duration.
[b] Number of subjects who responded at least once within the corresponding time duration.

Table 4
Results of multivariate logistic regression for physician visit and hospitalization for asthma according to parental smoking categories.

Parental smoking categories	Physician visit for asthma			Hospitalization for asthma		
	0.5 < –2.5 Years	2.5 < –4.5 Years	4.5 < –8 Years	0.5 < –2.5 Years	2.5 < –4.5 Years	4.5 < –8 Years
	AOR[a] (95% CI)	AOR[a] (95% CI)	AOR[a] (95% CI)	AOR[a] (95% CI)	AOR[a] (95% CI)	AOR[a] (95% CI)
Tobacco control perspective						
No parental smoking (reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Maternal non-smoking and paternal outdoor smoking	1.11 (0.99, 1.23)	1.07 (0.98, 1.18)	1.04 (0.95, 1.13)	1.11 (0.90, 1.37)	1.13 (0.90, 1.41)	1.21 (0.91, 1.62)
Maternal non-smoking and paternal indoor smoking	1.08 (0.97, 1.20)	1.01 (0.92, 1.11)	1.05 (0.96, 1.14)	1.00 (0.81, 1.23)	1.04 (0.83, 1.29)	1.22 (0.92, 1.60)
Both parental outdoor smoking	1.15 (0.93, 1.43)	1.10 (0.91, 1.34)	0.97 (0.80, 1.17)	1.14 (0.75, 1.71)	1.33 (0.86, 2.06)	1.70 (1.02, 2.83)
Both parental indoor smoking	1.31 (1.14, 1.49)	1.18 (1.04, 1.34)	1.19 (1.06, 1.33)	1.54 (1.21, 1.96)	1.43 (1.08, 1.90)	1.72 (1.22, 2.43)
Clinical perspective						
Both parental indoor smoking (reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Both parental outdoor smoking	0.88 (0.70, 1.11)	0.94 (0.76, 1.16)	0.81 (0.66, 1.001)	0.74 (0.48, 1.13)	0.93 (0.58, 1.48)	0.99 (0.57, 1.70)
Maternal non-smoking and paternal indoor smoking	0.83 (0.72, 0.95)	0.86 (0.75, 0.97)	0.88 (0.78, 0.994)	0.65 (0.51, 0.83)	0.72 (0.54, 0.97)	0.71 (0.50, 0.999)
Maternal non-smoking and paternal outdoor smoking	0.85 (0.74, 0.97)	0.91 (0.80, 1.04)	0.87 (0.77, 0.99)	0.72 (0.56, 0.93)	0.79 (0.59, 1.06)	0.71 (0.49, 1.01)
No parental smoking	0.77 (0.67, 0.88)	0.85 (0.75, 0.96)	0.84 (0.75, 0.95)	0.65 (0.51, 0.83)	0.70 (0.53, 0.92)	0.58 (0.41, 0.82)

Abbreviations: AOR, adjusted odds ratio; CI, confidence interval.
[a] Adjusted for child's sex, child's atopy, birthweight, breast feeding, number of siblings, population density, maternal age, paternal age and equivalent household income.

in "both parental outdoor smoking"). The time of parental behavior change was unknown; misclassification due to these parental smoking changes was not extremely large; and the trend of change according to parental smoking categories might result in conservative results. Therefore, smoking information from the 5th survey was not used in the current study. However, a future study that considers smoking behavior changes is necessary. Second, information on "prenatal" smoking was not available. Generally, few mothers change their smoking behavior in the perinatal period. If mothers stop smoking in pregnancy, most of them relapse soon after the birth (Shih et al., 2008). The lack of information on prenatal smoking may lead to an underestimation of SHS exposure, although several reviews have concluded that prenatal smoking data are not necessary to elicit the adverse effect of SHS (USDHHS, 2006; Vork et al., 2007). Third, the risk of asthma may be determined by unmeasured factors including genetics, which might be associated with both smoking and development or severity of asthma. For example, a history of parental asthma is one of the major risk factors for childhood asthma (Arshad et al., 2005). However, we were able to adjust the child's atopy variable, because this is suggested as a key confounding factor, complementing the lack of information of parental allergy (Vork et al., 2007).

Despite these limitations, this study has the strengths of large sample size with generalizability and a prospective design, which allows for examination of detailed parental smoking categories and the association with asthma with multiple covariate adjustments. To our knowledge, this is the first study to focus on the impact of different combinations of maternal and paternal smoking and indoor or outdoor smoking at home on asthma. Use of a nationally representative sample to estimate the population impact is another strength of the study. Moreover, because there has been no prospective study on the impact of paternal smoking on asthma for children aged 2 years or less and only 1 study for children aged 3–4 years (Burke et al., 2012), our findings contribute to filling an evidence gap on paternal smoking and childhood asthma.

4.4. Conclusions

We found that parental smoking especially maternal smoking, either indoor or outdoor at home, is harmful for the development and severity of childhood asthma. Smoking at home, whether it is indoor or outdoor, may increase the risks for asthma attacks of their children.

Author disclosures

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Contributors

All authors contributed to and have approved the final manuscript.

Conflict of interest statement

All authors declare that they have no conflicts of interest.

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Appendix A. Supplementary data

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Are secondhand smoke-related diseases of children associated with parental smoking cessation? Determinants of parental smoking cessation in a population-based cohort study

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ABSTRACT

Objective. Little is known about whether secondhand smoke (SHS)-related diseases of young children, such as asthma, induce parental smoking cessation during the early child-rearing period. Our objective was therefore to show the association in addition to other potential determinants of parental cessation.

Methods. We analyzed data from the Longitudinal Survey of Newborns in the 21st Century in Japan, from 0.5 years ($N = 47{,}015$) to 4.5 years ($N = 39{,}817$), having selected participants whose parents smoked at baseline (maternal smoking $N = 8{,}037$; paternal smoking $N = 28{,}486$). Multivariable log-binomial regression models were used to calculate the prevalence ratios for parental smoking cessation according to the onset of SHS-related diseases of their children, using inverse probability weight to account for non-response at follow-up.

Results. A total of 16.7% of smoking mothers and 14.5% of smoking fathers had stopped smoking at follow-up. The onset of SHS-related children's diseases was not statistically significantly associated with either maternal or paternal smoking cessation after multivariable adjustments. Strong determinants were, for example, number of cigarettes smoked per day and partner's smoking status during follow-up.

Conclusion. SHS-related children's diseases were not associated with parental smoking cessation. It may therefore be necessary to provide additional support for parental smoking cessation within their child's medical care setting.

Introduction

Smoking is an addictive behavior, and factors that contribute to smoking cessation have been investigated (Vangeli et al., 2011). Becoming a parent is one opportunity to quit smoking (Pollak et al., 2010). Children's exposure to secondhand smoke (SHS) due to parental smoking significantly increases SHS-related diseases such as asthma, middle ear diseases, respiratory illness including infection, and all-cause hospitalizations (Pattemore, 2013; Royal College of Physicians, 2010; U.S. Department of Health and Human Services[USDHHS], 2006). Therefore, it is thought that the medical care for children's SHS-related diseases setting offers a unique opportunity for health care professionals to advise parents to quit smoking (Stein et al., 2000). More specifically, parents may quit smoking if their child develops SHS-related diseases such as asthma (Stein et al., 2000). However, to the best of our knowledge, few studies have investigated whether, or to what extent, the onset of SHS-related disease in a child was associated with smoking cessation by the parents (Rattan et al., 2013).

According to national statistics in Japan, adult smoking prevalence decreased from 48% to 32% among men and from 14% to 10% among women between 2001 and 2010 (Ministry of Health, Labour and Welfare[MHLW], 2014). The number of 6-month-old children living with a smoking mother also decreased from 17% to 7%, while those living with a smoking father decreased from 63% to 42% between 2001and 2010 (MHLW, 2013). Although parental smoking prevalence has thus declined, many children continue to be exposed to SHS at home. Therefore, it is important to monitor parental smoking status and know the determinants of parental smoking cessation, including the SHS-related disease status of their children, to inform tobacco control measures that protect children's health in early life. However, studies that examine parental smoking cessation after childbirth are scarce (Hauge et al., 2013; Rattan et al., 2013).

The Longitudinal Survey of Newborns in the 21st Century is a nationally representative longitudinal study, which reports parental smoking status when the child is 0.5 years old (baseline), and again at 4.5. Further, this study includes details of the SHS-related disease status of

Abbreviations: SHS, secondhand smoke; PR, prevalence ratio; CI, confidence interval.

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the children. Thus, the data provided us with an opportunity to achieve our objective, which was to investigate whether the onset of SHS-related diseases in their child would induce parental smoking cessation in addition to other potential determinants, using a nationally representative sample.

Methods

Study subjects

The data used for this study were taken from the Longitudinal Survey of Newborns in the 21st century, which was conducted by the Japanese MHLW from the fiscal year 2001 to 2005 (MHLW, 2013). The study included all infants born in Japan during the periods January 10–17, 2001, and July 10–17, 2001 using national birth records (n = 53,575). Questionnaires were mailed to the households when the infants were 0.5 years of age (at July for children born in January and at January for children born in July). The total number of respondents was 47,015 households (response rate, 87.8%). Follow-up surveys were conducted at the ages of 1.5, 2.5, 3.5, and 4.5 years. Respondent numbers (households) for each survey were 43,925, 42,812, 41,559, and 39,817, respectively. Details of the study are available elsewhere (Fujiwara et al., 2013; Kaneita et al., 2006; MHLW, 2013; Yamakawa et al., 2013).

The analysis focused on parents who smoked at the time of the first surveys (n = 8172 for mother and n = 29,220 for father). Of those, subjects without information on parental age (n = 0 for mother and n = 148 for father) or child's birthweight (n = 1 for mother and n = 7 for father) were excluded from the analyses. Subjects of multiple births (n = 134 for mother and n = 581 for father) were also excluded because of the low reliability of the data. The number of remaining subjects was 8,037 mothers and 28,486 fathers at baseline (Fig. 1), to be analyzed separately. Data were used with permission from the Japanese MHLW. The analyses of national survey data were considered to be exempt from the need for ethical review according to the Epidemiological Research Guidelines.

Variables

Parents were asked whether they smoked (current smoker or not) when their child was 0.5 years old (baseline; 1st survey) and 4.5 years old (5th survey). As smoker (baseline) who did not smoke at the time of the 5th survey was defined as a case of smoking cessation.

The following characteristics were considered to be potential confounding factors on the basis of their potential association with parental smoking behavior and were categorized as (i) characteristics of smoking parent, (ii) characteristics of partner, (iii) characteristics of household, and (iv) characteristics of child. The characteristics of smoking parent included the following: the number

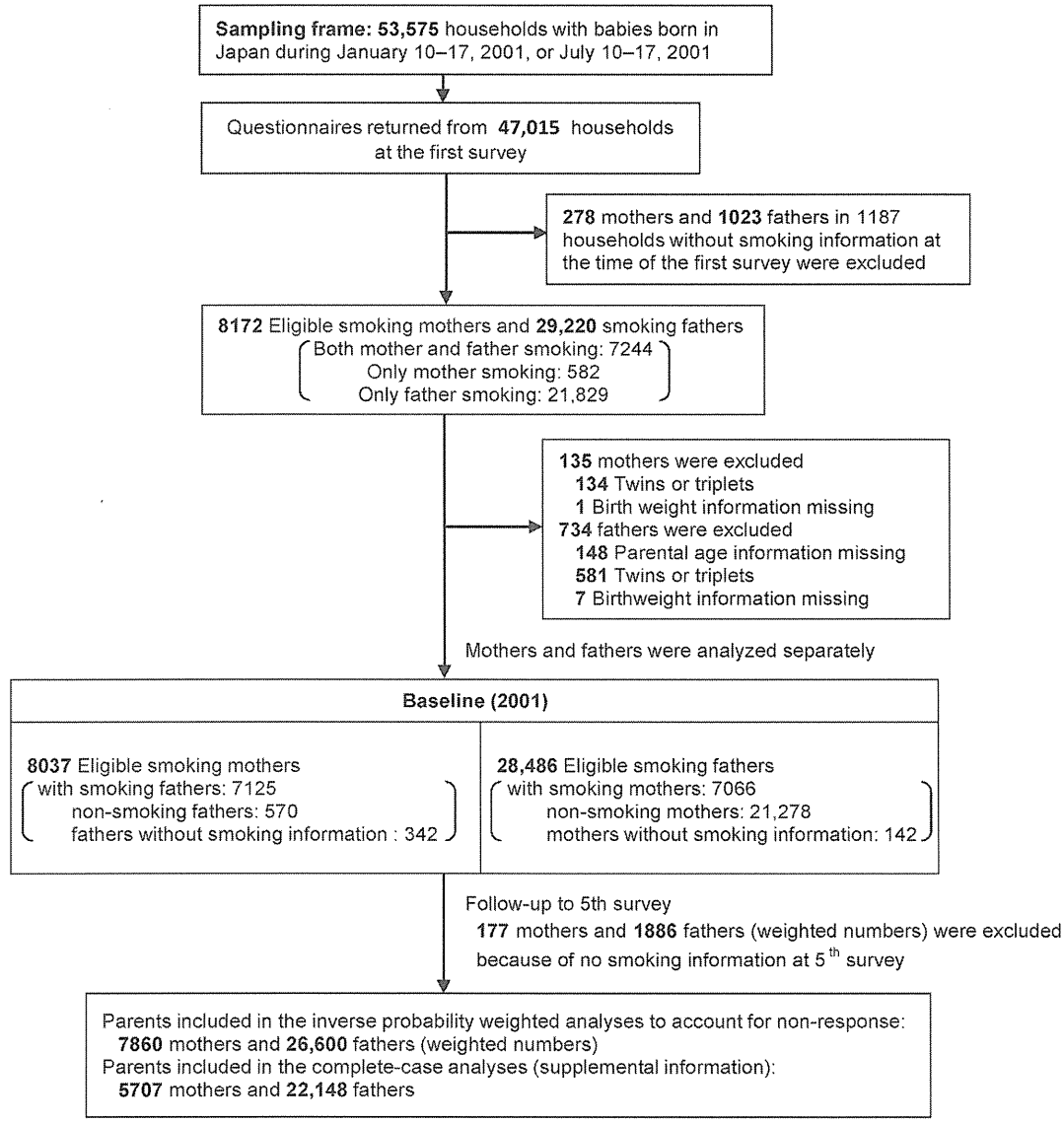

Fig. 1. Flow diagram for study subjects; Japan, 2001–2005.

of cigarettes smoked per day (1 to 9, 10, or 11 or more for the mother and 1 to 19, 20, or 21 or more for the father; 1st survey); indoor smoking (yes or no; 1st survey); parental age group (16–19, 20–29, 30–39, or 40–49 years old for mother, and 18–19, 20–29, 30–39, 40–49, or 50–71 years old for father, 1st survey); parental education (high school or less, technical school or junior college, or university [4 years] or more; 2nd survey), and living with partner (yes or no; at least one "no" or not [yes] from 1st to 5th survey). The characteristics of partner include the followng: partner's smoking behavior change from baseline to 5th survey ("no smoking" to "no smoking" [maintained non-smoker], "smoking" to "no smoking" [quitter], "no smoking" to "smoking" [relapse/initiation], or "smoking" to "smoking" [maintained current smoker]) and partner's age group and partner's education. The characteristics of household were based on equivalent household income (quartiles; 1st survey). The characteristics of child include the following: number of siblings (0 or > = 1; 1st survey); birth of new sibling (yes or no; at least once during follow-up period); exclusive breast feeding for the child (yes or no; 1st survey); and child's visit to physician for asthma, otitis media, or external otitis and child's hospitalization for asthma, respiratory illness, including pneumonia and infection, otitis media, or external otitis, and all causes, including all diseases and injuries (yes or no; at least once during follow-up period).

To examine whether parental smoking cessation was associated with SHS-related children's diseases in another way, combined SHS-related children's diseases were defined if children suffered from at least one of asthma (physician visit and hospitalization), respiratory illness (hospitalization), or "otitis media or external otitis" (physician visit and hospitalization) during the follow-up period. Because respiratory illness included "common cold" in the questionnaire, we used only hospitalization for respiratory illness (not physician visit) as usually pediatricians do not hospitalize children for a common cold. "Otitis media" was considered as an SHS-related children's disease, while "external otitis" was not (Eriksen et al., 2012; USDHHS, 2006). However, because a combined category of "otitis media and external otitis" was used in the questionnaire, we included this category as an SHS-related disease. Missing data were used as a dummy category in the analyses. Cutoffs of 10 for mothers and 20 for fathers were applied to the category "number of cigarettes smoked per day" on the basis of the smoking distribution. Maternal and paternal current smokers who smoked indoors at home (i.e., indoor smoker) were categorized as indoor smoking "yes."

Statistical analyses

Mothers and fathers were analyzed separately. Chi-squared tests were used to compare the difference in smoking cessation rates according to each characteristic variable. Log-binomial regression models were used to calculate the prevalence ratios (PRs) and 95% confidence intervals (CIs) for smoking cessation because the outcome was more than 10% (McNutt et al., 2003; Spiegelman and Hertzmark, 2005). In some instances, the models did not converge, and we therefore used log-Poisson models, which provide consistent but not fully efficient estimates of the PRs (i.e., the confidence intervals are slightly wider) (McNutt et al., 2003; Zou, 2004). Univariable and multivariable analyses were used to document the crude and adjusted relationship between independent variables and smoking cessation. The characteristics of subjects who participated in the study significantly differed from those who did not respond at follow-up survey (Table S1). To account for non-response, weighted estimating equations or multiple-imputation models are recommended rather than complete-case analysis or simple imputation methods (Little et al., 2012). We therefore constructed logistic regression models to explain the non-response using all listed covariates in Table S1. Next, we generated inverse probability weight by the logistic model, after confirming no evidence of poor fit of the model. We then used the results with inverse probability weight, which are shown as main results (weighted results). The complete-case analysis is shown as supplemental information (Tables S2 and S3). Details of covariates and non-response analysis are available in supplemental information.

Probability values for statistical tests were two-tailed, and $P < 0.05$ was considered statistically significant. All analyses were performed using SAS version 9.2 (SAS Institute, Cary, NC, USA).

Results

Characteristics and cessation rates among smoking mothers are shown in Table 1. At 4-year follow-up, 16.7% of smoking mothers had stopped. In the univariable model, child's hospitalization for respiratory illness, ear diseases, all causes, and SHS-related disease showed positive association with maternal smoking cessation. However, after multivariable adjustments, these hospitalizations were no longer significantly associated. Other factors which were significantly associated with smoking cessation were fewer cigarettes smoked per day (PR [95% CI] = 0.40 [0.34, 0.46] for "≥11 cigarettes" compared with "1–9 cigarettes"), not being a teenage mother (PR [95% CI] = 0.55 [0.37, 0.83] for "16–19 years old" compared with "20–29 years old"), higher educational attainment (PR [95% CI] = 1.17 [1.03, 1.33] for "technical school or junior college" compared with "high school or less"), partner's smoking status (PR [95% CI] = 3.27 [2.86, 3.75] for "quitters" and 1.50 [1.22, 1.84] for "maintained non-smokers" compared with "maintained current smokers"), born as subsequent child (PR [95% CI] = 0.84 [0.74, 0.96] for "≥1" compared with "0"), and having a new sibling (PR [95% CI] = 1.41 [1.25, 1.59] for "yes" compared with "no").

Table 2 shows characteristics and cessation rate among smoking fathers. At 4-year follow-up, 14.5% of smoking fathers had stopped. In the univariable model, interestingly, child's hospitalization for asthma showed a negative association with smoking cessation. In the multivariable model, no children's disease status was associated with paternal smoking cessation. On the other hand, the following characteristics were significantly associated with smoking cessation in the multivariable model: fewer cigarettes smoked per day (PR [95% CI] = 0.56 [0.51, 0.62] for "≥21 cigarettes" compared with "1–19 cigarettes"), indoor smoking (PR [95% CI] = 0.80 [0.75, 0.85] for "yes" compared with "no"), older age (PR [95% CI] = 1.51 [1.05, 2.19] for "50–71 years old" compared with "20–29 years old"), higher educational attainment (PR [95% CI] = 1.30 [1.21, 1.40] for "university (4 years) or more" compared with "high school or less"), living with partner (PR [95% CI] = 1.56 [1.14, 2.13] for "no" compared with "yes"), and partner's smoking status (PR [95% CI] = 4.04 [3.50, 4.66] for "quitters" and 2.19 [1.95, 2.46] for "maintained non-smokers" compared with "maintained current smokers").

Discussion

Overall, 16.7% of maternal smokers and 14.5% of paternal smokers stopped smoking while their children were growing up (0.5 to 4.5 years old). The maternal smoking cessation rate during the child-rearing period observed in this study was similar to that of a previous Australian cohort study(Rattan et al., 2013), which found that 16% of women who continued smoking in pregnancy had stopped at 5-year follow-up.

Surprisingly, the onset of SHS-related children's diseases was not associated with either maternal or paternal smoking cessation at follow-up in the multivariable models. This finding may be explained by the following: (i) Because smoking cessation is difficult even among patients with their "own" smoking-related diseases such as lung cancer or stroke (Cooley et al., 2009; Ives et al., 2008), it may be natural that they could not stop smoking due to the onset of their child's disease ("not their own"). Furthermore, as women who are pregnant or child rearing are busy and often stressed, they may not be able to stop smoking (Halterman et al., 2007). (ii) In some cases, the SHS-related disease may not be associated with parental smoking (i.e., other cause of disease), or parents may believe that their child's SHS exposure is small, even when it is large: for example, when parents smoke near the kitchen fan (Johansson et al., 2004). Therefore, parents may not know or not believe that the child's disease is related to SHS. (iii) If parents quit soon after the first smoking status survey and have still stopped at 4-year follow-up, the onset of any child's SHS-related disease during follow-up might be prevented. This leads to underestimation of the association between child's SHS-related disease onset and parental smoking cessation. However, this third hypothesis was rejected as we found that there is no trend of reduction in child's SHS-related diseases (either physician visit or hospitalization) among parental quitters during the 4-year follow-up (compared with the trend among

Table 1
Prevalence ratios (95% CI) for smoking cessation according to characteristics, among smoking mothers; Japan, 2001–2005.

Characteristics			Quit at 5th survey (when their child was 4.5 years old)				
	Weighted no.	%	Weighted no.	%	*P* value[a]	Univariable PRs (95% CI)	Multivariable PRs[b] (95% CI)
Total smoking mothers at baseline	7860	100.0	1311	16.7		NA	NA
Characteristics of mother							
Number of cigarettes smoked per day					<0.0001		
1–9	2224	28.3	609	27.4		1.0 (reference)	1.0 (reference)
10	2767	35.2	439	15.9		**0.58 (0.52, 0.65)**	**0.64 (0.57, 0.73)**
≥11	2829	36.0	254	9.0		**0.33 (0.29, 0.38)**	**0.40 (0.34, 0.46)**
Missing	41	0.5	9	21.7		0.79 (0.44, 1.43)	0.76 (0.39, 1.48)
Smoking indoors					<0.0001		
No	2232	28.4	443	19.8		1.0 (reference)	1.0 (reference)
Yes	5496	69.9	830	15.1		**0.73 (0.65, 0.81)**	0.95 (0.85, 1.07)
Missing	132	1.7	38	28.7		**0.85 (0.72, 0.99)**	1.35 (0.96, 1.89)
Mother's age					0.0003		
16–19 years old	299	3.8	29	9.8		**0.55 (0.39, 0.78)**	**0.55 (0.37, 0.83)**
20–29 years old	4965	63.2	881	17.7		1.0 (reference)	1.0 (reference)
30–39 years old	2525	32.1	394	15.6		**0.88 (0.79, 0.98)**	0.97 (0.84, 1.13)
40–49 years old	70	0.9	7	9.7		0.55 (0.27, 1.12)	0.85 (0.39, 1.83)
Mother's education					<0.0001		
High school or less	4720	60.1	693	14.7		1.0 (reference)	1.0 (reference)
Technical school or junior college	1871	23.8	404	21.6		**1.47 (1.32, 1.64)**	**1.17 (1.03, 1.33)**
University (4 years) or more	222	2.8	65	29.2		**1.99 (1.60, 2.47)**	1.27 (0.97, 1.66)
Missing	1046	13.3	149	14.3		0.97 (0.82, 1.14)	0.81 (0.52, 1.26)
Living with partner (father)					<0.0001		
Yes	6591	83.9	1170	17.8		1.0 (reference)	1.0 (reference)
No (at least once)	1269	16.1	141	11.1		**0.62 (0.53, 0.74)**	0.92 (0.67, 1.29)
Characteristics of partner							
Smoking status change from baseline to 5th survey					<0.0001		
"No smoking" to "no smoking" (maintained non-smokers)	460	5.9	109	23.6		**1.69 (1.42, 2.02)**	**1.50 (1.22, 1.84)**
"Smoking" to "no smoking" (quitters)	622	7.9	302	48.5		**3.48 (3.13, 3.86)**	**3.27 (2.86, 3.75)**
"No smoking" to "smoking" (relapse)	68	0.9	7	9.6		0.68 (0.33, 1.43)	0.70 (0.32, 1.52)
"Smoking" to "smoking" (maintained current smokers)	5635	71.7	786	14.0		1.0 (reference)	1.0 (reference)
Missing	1075	13.7	107	10.0		**0.72 (0.59, 0.87)**	1.00 (0.68, 1.47)
Partner's (father's) age					<0.0001		
18–19 years old	136	1.7	9	6.7		**0.37 (0.20, 0.69)**	0.51 (0.25, 1.04)
20–29 years old	4016	51.1	726	18.1		1.0 (reference)	1.0 (reference)
30–39 years old	2976	37.9	493	16.6		0.92 (0.83, 1.02)	0.98 (0.85, 1.12)
40–49 years old	413	5.3	55	13.3		**0.74 (0.57, 0.95)**	0.84 (0.62, 1.13)
50–71 years old	59	0.8	6	10.4		0.58 (0.27, 1.22)	0.65 (0.29, 1.44)
Missing	259	3.3	22	8.3		**0.46 (0.31, 0.69)**	0.81 (0.47, 1.40)
Partner's (father's) education					<0.0001		
High school or less	4800	61.1	731	15.2		1.0 (reference)	1.0 (reference)
Technical school or junior college	911	11.6	187	20.6		**1.35 (1.17, 1.56)**	1.13 (0.96, 1.34)
University (4 years) or more	905	11.5	221	24.4		**1.60 (1.41, 1.83)**	1.15 (0.97, 1.35)
Missing	1244	15.8	172	13.8		0.91 (0.78, 1.06)	1.41 (0.92, 2.17)
Characteristics of household							
Equivalent household income					<0.0001		
1st quartile (Lowest)	1918	24.4	305	15.9		**0.74 (0.65, 0.84)**	1.03 (0.87, 1.21)
2nd quartile	1898	24.2	255	13.4		**0.62 (0.54, 0.72)**	0.87 (0.73, 1.03)
3rd quartile	1878	23.9	324	17.3		**0.80 (0.70, 0.91)**	0.96 (0.82, 1.12)
4th quartile (highest)	1865	23.7	402	21.6		1.0 (reference)	1.0 (reference)
Missing	302	3.8	25	8.1		**0.38 (0.26, 0.56)**	0.75 (0.44, 1.28)
Characteristics of child							
Number of siblings					<0.0001		
0	3883	49.4	770	19.8		1.0 (reference)	1.0 (reference)
≥1	3977	50.6	541	13.6		**0.69 (0.62, 0.76)**	**0.84 (0.74, 0.96)**
Birth of new sibling during follow-up					<0.0001		
No	5124	65.2	696	13.6		1.0 (reference)	1.0 (reference)
Yes (at least once)	2735	34.8	615	22.5		**1.66 (1.50, 1.83)**	**1.41 (1.25, 1.59)**
Low birthweight							
No	7149	91.0	1205	16.9	0.1812	1.0 (reference)	1.0 (reference)
Yes	711	9.1	106	14.9		0.88 (0.74, 1.06)	0.97 (0.79, 1.18)
Breastfeeding exclusive							
Yes	797	10.1	151	19.0		1.0 (reference)	1.0 (reference)
No	6982	88.8	1144	16.4	0.0596	0.86 (0.74, 1.00)	0.97 (0.82, 1.16)
Missing	81	1.0	16	19.6		1.03 (0.65, 1.64)	1.07 (0.63, 1.81)
Physician visits for asthma during follow-up							
No	6727	85.6	1141	17.0	0.0935	1.0 (reference)	1.0 (reference)
Yes (at least once)	1133	14.4	169	15.0		0.88 (0.76, 1.02)	0.90 (0.74, 1.08)
Hospitalization for asthma during follow-up							
No	7570	96.3	1265	16.7	0.6449	1.0 (reference)	1.0 (reference)
Yes (at least once)	290	3.7	46	15.7		0.94 (0.72, 1.23)	1.10 (0.77, 1.57)
Hospitalization for respiratory illness during follow-up							
No	6724	85.6	1097	16.3	0.0359	1.0 (reference)	1.0 (reference)
Yes (at least once)	1136	14.5	214	18.8		**1.15 (1.01, 1.32)**	1.02 (0.83, 1.24)
Physician visits for ear diseases during follow-up							

Table 1 (*continued*)

Characteristics			Quit at 5th survey (when their child was 4.5 years old)				
	Weighted no.	%	Weighted no.	%	*P* value[a]	Univariable PRs (95% CI)	Multivariable PRs[b] (95% CI)
No	5091	64.8	820	16.1	0.0679	1.0 (reference)	1.0 (reference)
Yes (at least once)	2769	35.2	491	17.7		1.10 (0.99, 1.22)	1.07 (0.95, 1.20)
Hospitalization for ear diseases during follow-up							
No	7774	98.9	1286	16.6	0.0028	1.0 (reference)	1.0 (reference)
Yes (at least once)	85	1.1	25	28.7		**1.73 (1.24, 2.43)**	1.18 (0.77, 1.79)
Hospitalization for all-causes during follow-up							
No	5772	73.4	922	16.0	0.0055	1.0 (reference)	1.0 (reference)
Yes (at least once)	2088	26.6	389	18.6		**1.17 (1.05, 1.30)**	1.15 (0.97, 1.36)
Physician visits for SHS-related diseases during follow-up							
No	4382	55.8	707	16.1	0.1438	1.0 (reference)	1.0 (reference)
Yes (at least once)	3478	44.3	604	17.4		1.08 (0.98, 1.19)	1.08 (0.96, 1.20)[c]
Hospitalization for SHS-related diseases during follow-up							
No	6545	83.3	1059	16.2	0.0092	1.0 (reference)	1.0 (reference)
Yes (at least once)	1314	16.7	251	19.1		**1.18 (1.04, 1.34)**	1.06 (0.86, 1.31)[c]

Abbreviations: No, numbers; PR, prevalence ratio; CI, confidence interval; NA, not applicable; SHS, secondhand smoke.
Bold = statistical significance of $p < 0.05$.
[a] Chi-square tests.
[b] Adjusted for listed all variables except for combined SHS-related children's diseases (both physician visit and hospitalization).
[c] Adjusted for listed all variables except for asthma, respiratory illness and ear diseases (both physician visit and hospitalization).

continuing smokers) using time intervals of child-age $0.5 < -1.5$, $1.5 < -2.5$, $2.5 < -3.5$ and $3.5 < -4.5$ years old (data not shown).

We found several determinants for parental smoking cessation after multivariable adjustments. (I) Partner's smoking change was the strongest determinant for parental smoking cessation, confirming previous evidence (Nafstad et al., 1996; Schneider et al., 2010). Although there is no time information on which is earlier or later for maternal or paternal smoking behavioral change, maternal smoking was strongly correlated with paternal smoking, regardless of the cessation sequence. Compared with maintained current smoking partners ("smoking to smoking"), maintained non-smoking partners ("no smoking to no smoking") was a significant determinant of parental smoking cessation in both mothers and fathers. In order to increase smoking cessation in parents effectively, mutual partner support may be a key intervention (Park et al., 2012). (II) Parents who smoked fewer cigarettes were more likely to quit smoking. This is possibly because of nicotine dependence and is consistent with previous studies among the general public and pregnant women (Hagimoto et al., 2010; Schneider et al., 2010; Vangeli et al., 2011). (III) Some socioeconomic characteristics, such as partner's education and household income, were not significantly associated with smoking cessation at follow-up, while "own" educational attainments were associated among both mothers and fathers. This may be because education levels represent a complex set of "health literacy" skills, which relate to health behavior change including smoking cessation (Stewart et al., 2013). (IV) New sibling and sibling number were associated with maternal smoking cessation at follow-up, while these were not associated with paternal cessation. This is probably because pregnancy is an event that may make the mother quit smoking (Chamberlain et al., 2013; Schneider et al., 2010). The different result between mother and father is reasonable because pregnancy occurs only in women and some pregnant women live apart from their partner during the perinatal period in Japan (Ohga et al., 2005). To date, maternal smoking has been well researched, whereas paternal smoking has received little attention (Blackburn et al., 2005). Therefore, little is known about the determinants of paternal smoking cessation and our findings may contribute to filling an information gap on paternal smoking determinants.

Limitations and strength of the study

There are several limitations to this study. First, we used data from a child (born in 2001)-based family-cohort to access parental smoking behavior. Lack of detailed information on other children and family members may lead to misclassification of children's diseases and missing of confounding factors. However, results limited to first-child cases did not largely differ (data not shown), and important confounding factors of parental "own" status such as cigarettes smoked per day (a proxy for nicotine dependence) and education attainment could be included in the analyses (Vangeli et al., 2011). Second, smoking variables were self-reported without biomarker validation, but the reliability of self-reported smoking behavior was generally high (Caraballo et al., 2001). Third, smoking information before and during pregnancy was not available. Approximately half the women who stopped smoking during their pregnancies relapsed after childbirth (Tong et al., 2009; Yasuda et al., 2013). We speculated on, but could not investigate, whether women who stopped smoking in pregnancy and relapsed when their child was 6 months old were more likely to quit smoking again at follow-up than those who smoked in pregnancy. In a previous study, 45% of women who quit in pregnancy relapsed at 6 months follow-up, and 44% of women who quit in pregnancy currently smoked at 5-year follow-up (Rattan et al., 2013). Finding almost the same smoking prevalence at 6-months and 5-year follow-up among women who quit in pregnancy (45% and 44%, respectively) may imply a small impact of the factor "women who quit in pregnancy." Fourth, we could not include the effect of any tobacco control measures such as workplace smoking restrictions. However, this effect may be small and not specific because we found no significant tobacco control measures between 2001 and 2005 in Japan, except for small a price increase in 2003 (Katanoda et al., 2014).

Despite these limitations, this study has the strengths of a large representative sample with possible generalizability for the whole country and a prospective design that allows the examination of a detailed picture for parental smoking cessation with multiple covariate adjustments.

Conclusions

We found that less than 20% of smoking parents stopped smoking while their children were aged 0.5 to 4.5 years old. SHS-related children's diseases were not associated with parental smoking cessation. It may be necessary to focus support on parental smoking cessation within the child's medical care setting (Stein et al., 2000).

Conflict of interest

Authors have no conflicts of interest to disclose.