Higher hair nicotine level in children compared to mother living with smoking father in Malaysia

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Abstract

Objectives The objectives of this study are to determine parental and children’s hair nicotine levels, their relationships as well as to investigate the association of smoking status of the fathers with mothers’ and children’s hair nicotine.

Methods A cross-sectional study design was conducted among 124 families who were participants of the Universiti Sains Malaysia Pregnancy Cohort Study. Both parents with their 2 years old children joined this study. A total of 92 hair samples of fathers, 124 hair samples of mothers and 111 hair samples of children were collected and analyzed by gas chromatography–mass spectrometry.

Results Of total, 52.4 % of the fathers reported smoking. None of the mothers were smokers. Hair nicotine levels of fathers were found to be significantly correlated with mothers (r = 0.233, p = 0.026) and children (r = 0.508, p < 0.001). Children living with smoking fathers had significantly higher median hair nicotine level compared to the children of non-smoking fathers (6.08 vs 0.22 ng/mg, p = 0.046). However, this association was not seen in the mothers. Quantile regression showed significant association between fathers’ and children’s hair nicotine.

Conclusion There is a positive relationship between fathers’ hair nicotine with mother’s and children’s hair nicotine. Living with smoking fathers can contribute to higher hair nicotine levels in children but not in mothers.

Keywords Secondhand smoke · Smoking · Hair nicotine · Mothers · Children

Introduction

Secondhand smoke (SHS) refers to the cigarette smoke which is involuntarily breathed in by non-smoker [1]. It is also acknowledged as passive smoke or environmental tobacco smoke (ETS) [1, 2]. As reported by the U. S. Department of Health Human Services [3], non-smoking women were described as exposed to SHS if they are living with husbands who are smokers or exposed to the tobacco smoke at the workplaces. The exposure to SHS among children is usually referred to the children who have one or both parents who are smokers or living with any smokers in the households.

The residual of tobacco smoke contaminants that are left on surfaces and dust after smoking is known as thirdhand smoke (THS) [4]. Examples of THS components include nicotine, formaldehyde, phenol, cresol and naphthalene [5, 6]. SHS and THS are strongly associated and exist together in contaminated places where smoking activity occurs occasionally [4].

There are about 4000 poisonous chemical components in cigarette smoke such as hazardous irritants and systemic toxicants like hydrogen cyanide and sulfur dioxide; mutagens and cancer-causing agents like formaldehyde, bicyclic

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and polycyclic aromatic hydrocarbons (PAH) as well as reproductive toxicants like carbon monoxide and cadmium [3]. Exposure to cigarette smoke can happen anywhere such as at home, work places or public places [7]. Among all places, the main source of SHS is the exposure at home [8].

Numerous studies have reported the detrimental health consequences of SHS exposure on mothers’ and children’s health. The involuntary exposure to tobacco smoke can cause lung cancer, heart diseases, depression, low birth weight, respiratory infections, middle ear disease, cough and asthma attack [9–12]. Non-smokers are not totally protected from SHS even though the smokers are smoking outside, in other rooms or during the non-smokers are away [1]. It has been documented that tobacco smoke can simply diffuse from room to room in an apartment and can move from the outside into a house [13].

SHS exposure can be evaluated using self-reported questionnaire or analysis of biological markers such as thiocyanate, carbon monoxide, PAH, nicotine and cotinine [3, 14]. Thiocyanate, carbon monoxide and PAH are not specific to SHS since they can be influenced by other dietary or environmental sources [15, 16]. Nicotine and its metabolite, cotinine are widely used as specific biomarkers to investigate the exposure to tobacco smoke and they can be measured in hair, urine, blood or saliva [17, 18]. Hair nicotine is an accurate and reliable biomarker for cumulative exposure to SHS besides it is non-invasive and the hair samples are easy to be stored [19, 20]. The hair growth is approximately 1 cm per month so it can be assumed that 1 cm of hair from the scalp signifies 1 month exposure to tobacco smoke [21]. The nicotine in the blood can enter the hair cells through passive diffusion and attach firmly inside the hair shaft until next keratogenesis [20].

To date, the research on the association of fathers’ hair nicotine level with mothers’ and children’s hair nicotine is scarce in Malaysia. Therefore, this study aimed to determine parental and children’s hair nicotine levels as well as to investigate the association of smoking status of the fathers with mothers’ and children’s hair nicotine. We hypothesized that there is a significant association between smoking status of the fathers with hair nicotine of mothers and children.

Materials and methods

Participants

A total of 124 families had participated in this study. The data collection was conducted from April 2013 until December 2013 by visiting the participants’ home in 6 districts of Kelantan, Malaysia. The inclusion criteria were parents aged between 20 and 50 years old, had children aged 2 years old and Malaysian. Mothers who had chronic diseases, pregnant during recruitment time and the children who were delivered less than 37 weeks of gestation were excluded from this study. The ethical approval was obtained from the Universiti Sains Malaysia (USM) Human Research Ethics Committee.

During home visit, the detailed procedure of the study was explained and informed consent was obtained from all individual participants included in the study. Socio-demographics information like age, parents’ education level, monthly household income, smoking status of the fathers and number of children were obtained via interview-administered questionnaire. Parental and children’s hair samples were collected to assess their nicotine levels. Figure 1 presents the flowchart of this study.

Hair nicotine analysis

A total of 30 strands of full-length hair of parents and their children were cut close to the scalp, taped on a white paper, labeled at the hair root and kept in an envelope at room temperature till analysis. Hair sample was not obtained from participants who had hair length less than 3 cm or bald.

Since it had been presumed that 1 cm of hair from the scalp indicated 1 month exposure to tobacco smoke [21], each hair sample was cut into 3 cm from the hair root which represented the exposure to cigarette smoke for the past three months. All hair samples had been washed with deionized water and sonicated twice with dichloromethane (DCM) for 15 min to ensure all external contaminations were completely removed [22]. The samples were dried overnight at 37 °C and minced into 1–2 mm pieces using scissors.

Hair nicotine analysis was determined according to the previous literature using gas chromatography–mass spectrometry (GC–MS) [23]. Preparation of reagents and analysis of hair nicotine were performed by the laboratory technician at the National Poison Centre of Malaysia.
sample of healthy non-smoking and non-SHS exposed woman was chosen for pooled blank hair sample. The concentration of nicotine in hair was calculated by dividing the quantity of nicotine detected in hair (ng) by the mass of hair investigated (mg). The sensitivity of this assay, which was determined by limit of detection (LOD) less than 0.010 ng/mg and limit of quantitation (LOQ), was achieved at 0.040 ng/mg when 5 mg of hair was used. The accuracies and precisions within- and between-assays were less than 11.4 % [23]. The quantifying ions used were m/z 162 for nicotine and m/z 169 for internal standard [16, 24].

Statistical analysis

Data analyses were performed using IBM SPSS Statistics, Version 22.0 (USA) and STATA, Version 12.0 (Texas). The significant differences of number of children, years of education and monthly household income between smokers and non-smokers were tested using independent t test. Data for hair nicotine were obtained when it was above the LOD. The hair samples below the LOD (<0.040 ng/mg) were allocated with a value of quarter of detection limit (i.e., 0.010 ng/mg) for the purpose of statistical analysis. The hair nicotine levels between mothers and children who were living with smoking and non-smoking fathers were compared using Mann–Whitney test. Spearman correlation test was applied to examine the correlation of parental with children’s hair nicotine. Quantile regression analysis with confounder adjustment was used to predict the change in median nicotine levels of the children for every unit change of fathers’ hair nicotine. Quantile regression estimates are more robust against outliers in the response measurements relative to the ordinary least squares regression [25]. Confounders were selected based on previous studies [26–28].

Results

Socio-demographic characteristics of the participants are presented in Table 1. Of total, 52.4 % of the fathers were smokers. None of the mothers reported smoking. The smokers had significantly lesser years of education \((p = 0.007)\) and monthly household income \((p = 0.025)\) compared to non-smokers. Also, the mothers who were living with smoking husbands had significantly less years of education \((p = 0.013)\) than those who were living with non-smoking husbands.

Among currently reported non-smokers, 86.4 % never smoked and 13.6 % stopped smoking more than 6 months. Most of the smokers smoked more than 10 cigarettes daily (61.4 %). The mean number of cigarettes smoked daily was 11 cigarettes and the average years of smoking were 15.11 (SD 7.11) years.

A total of 92 hair samples were obtained from the fathers, 124 hair samples from mothers and 111 hair samples from the children. As shown in Table 2, the majority of the fathers (77.2 %) and children (64.9 %) had nicotine detected in their hair, which is above the detection limit. However, only 29 (23.4 %) of the mother’s hair had detected with nicotine.

The concentrations of nicotine in parents’ and children’s hair are summarized in Table 3. The median of hair nicotine in fathers’, mothers’ and children’s hair was 6.68 (IQR 0.01–125.97) ng/mg, 0.01 (IQR 0.01–0.01) ng/mg and 3.24 (IQR 0.01–25.23) ng/mg, respectively.

Fathers’ hair nicotine was positively correlated with mother’s \((r = 0.233; p = 0.026)\) and children’s \((r = 0.508; p < 0.001)\) hair nicotine levels (Table 4). However, mother’s hair nicotine level was not significantly correlated with children’s \((r = 0.162; p = 0.087)\).

Table 5 displays the comparison of mothers’ and children’s hair nicotine living with smoking and non-smoking fathers. The median hair nicotine of mothers who were living with smoking husbands was not significantly different from mothers who were living with non-smoking husbands \((p = 0.494)\). In contrast, the median hair nicotine of children living with smoking fathers \((6.08 \text{ ng/mg})\) was significantly higher \((p = 0.046)\) than the children living with non-smoking fathers \((2.22 \text{ ng/mg})\).

The association of the children’s hair nicotine by the fathers’ hair nicotine is depicted in Table 6. In the unadjusted model (Model 1), for every unit increase of fathers’ hair nicotine levels, the change in median nicotine levels of children was 0.10 ng/mg (95 % confidence interval 0.08, 0.11). This association remained consistent after adjusting for family income, parity, father’s education and mothers’ nicotine levels. Adjustment for potential confounders did not attenuate this association considerably.

Discussions

This current study found that fathers’ hair nicotine level has been linked with mothers’ and children’s hair nicotine. Also, the smoking status of the fathers was significantly associated with hair nicotine level of the children. However, no significant association was noted in mothers’ hair nicotine.

None of the woman in this study reported smoking. This is probably because cigarette smoking among women is unacceptable by many cultures in Malaysia. Compared to mothers, the majority of children (64.9 %) had nicotine levels detected in their hairs. Only 23.4 % of the mothers’ hairs were detected with nicotine. It had been suggested
that someone with undetectable nicotine in hair was regarded as unexposed to SHS smoke [2]. Perhaps, the majority of mothers with undetected hair nicotine in this study were most likely aware of the health outcomes of SHS and had avoided themselves from the passive tobacco smoke.

There are many other factors that can influence the metabolism of nicotine in the body. The primary female sex hormone which is estrogen can affect nicotine metabolism in the body [30]. The researchers had found that the elimination of nicotine is greater in women compared to men and the use of oral contraceptive can also increase

| Table 1 Characteristics of the participants (n = 124)a | Fathers’ smoking status |  |  |
|------------------------------------------------------|-------------------------|-----------------|-----------------|
| Characteristics                                      | Smokers (n = 65)        | Non-smokers (n = 59) |
| Number of childrenb                                  | 2.65 (1.41)             | 2.75 (1.47)     | 0.701 |
| 1                                                    | 13 (20.0)               | 10 (16.9)       |     |
| 2                                                    | 23 (35.4)               | 23 (39.0)       |     |
| 3 and above                                          | 29 (44.6)               | 26 (44.1)       |     |
| Fathers’ education (years)b                          | 13.55 (2.89)            | 14.98 (2.87)    | 0.007* |
| Primary/secondary school                             | 31 (47.7)               | 14 (23.7)       |     |
| Diploma degree                                       | 25 (38.5)               | 23 (39.0)       |     |
| Bachelor degree/Masters/PhD                         | 9 (13.8)                | 22 (37.3)       |     |
| Mothers’ education (years)b                          | 14.41 (2.42)            | 15.86 (3.79)    | 0.013* |
| Primary/secondary school                             | 22 (33.8)               | 12 (20.3)       |     |
| Diploma degree                                       | 33 (50.8)               | 22 (37.3)       |     |
| Bachelor degree/Masters/PhD                         | 10 (15.4)               | 25 (42.4)       |     |
| Mothers’ employment status                           |                         |                 |     |
| Employed                                             | 51 (78.5)               | 54 (91.5)       |     |
| Housewife                                            | 14 (21.5)               | 5 (8.5)         |     |
| Monthly household incomeb,c                          | 3477.69 (2171.44)       | 4820.34 (4033.26) | 0.025* |
| <RM 2300                                             | 22 (33.8)               | 13 (22.0)       |     |
| RM 2300–RM 5599                                      | 35 (53.9)               | 30 (50.9)       |     |
| ≥RM 5600                                             | 8 (12.3)                | 16 (27.1)       |     |
| Smoking status                                       |                         |                 |     |
| Current cigarette smokers                            | 65 (100.00)             | –               | –    |
| Never smokers                                        | –                       | 51 (86.4)       |     |
| Former cigarette smokers                             | –                       | 8 (13.6)        |     |
| Numbers of cigarettes/day (n = 57)b                  | 10.55 (6.74)            | –               | –    |
| 1–10                                                 | 22 (38.6)               | –               |     |
| 11 and more                                          | 35 (61.4)               | –               |     |

* PhD Doctor of Philosophy, RM Ringgit Malaysia

a Data were presented as frequency (percentage), unless otherwise indicated
b Data were presented as mean (standard deviation)

| Table 2 Percentage of nicotine detected in parental and children’s hair |  |
|---------------------------------------------------------------|-------------------|
| Characteristic                                               | Fathers’ hair nicotine (n = 92) | Mothers’ hair nicotine (n = 124) | Children’s hair nicotine (n = 111) |
|---------------------------------------------------------------|-------------------------|-----------------------------|-----------------------------|
| Detected                                                     | 71 (77.2)              | 29 (23.4)                   | 72 (64.9)                   |
| <LOD                                                         | 21 (22.8)              | 95 (76.6)                   | 39 (35.1)                   |

Data were presented in frequency (percentage)

LOD limit of detection
nicotine metabolism [30]. Other than that, the application of chemical hair treatment such as coloring, bleaching and perming can also decrease the concentration of nicotine in hair by 9% [31, 32].

Higher median hair nicotine level was observed in children who were living with smoking fathers (6.08 ng/mg) compared to the children living with non-smoking fathers (0.22 ng/mg). The finding in this study is consistent with previous studies [33, 34]. In contrast with adults, young children usually unable to keep away themselves from the SHS [35]. Other than that, the nicotine in the hair shaft of young children can accumulate faster while the elimination process is slower [36]. Children are also at greater risk to be exposed to THS because they spend more time playing, crawling, touching contaminated surface and tend to put in non-food item in their mouth [13]. They also have higher metabolic rates and breathe in a greater amount of air per kilogram body weight, which result in more air pollutants entering their body [37].

In addition, this current study also found that nicotine was also detected in the hairs of children who were living with non-smoking fathers. This is probably due to SHS

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### Table 3 Concentration of parents’ and children’s hair nicotine

|                        | Hair nicotine concentration (ng/mg) |
|------------------------|-------------------------------------|
|                        | Fathers (n = 92) | Mothers (n = 124) | Children (n = 111) |
| Maximum level          | 495.13            | 82.76             | 78.07             |
| GM                     | 1.44              | 0.04              | 0.32              |
| Median                 | 6.68              | 0.01              | 3.24              |
| 25th percentile        | 0.01              | 0.01              | 0.01              |
| 75th percentile        | 125.97            | 0.01              | 25.23             |

*GM geometric mean*

### Table 4 Correlation of parents’ and children’s hair nicotine

|                        | Mothers’ hair nicotine | Children’s hair nicotine |
|------------------------|------------------------|-------------------------|
|                        | r  p value            | r  p value              |
| Fathers’ hair nicotine | 0.233 0.026*          | 0.508 <0.001*           |
| Mothers’ hair nicotine | – –                   | 0.162 0.087             |

* Statistical significant of the association

* p value based on Spearman correlation

### Table 5 Comparison of mothers’ and children’s hair nicotine levels between smoking and non-smoking fathers

|                        | Hair nicotine (ng/mg) | Smoking fathers | Non-smoking fathers | p value |
|------------------------|-----------------------|-----------------|---------------------|---------|
|                        | n  Median (IQR)       | n  Median (IQR) |                     |         |
| Mothers                | 65 0.01 (0.01–0.04)   | 59 0.01 (0.01–0.01) | 0.494               |
| Children               | 61 6.08 (0.01–26.52)  | 50 0.22 (0.01–16.24) | 0.046*              |

* IQR interquartile range

* Statistical significant of the association

* p value based on Mann–Whitney test

### Table 6 Association between fathers’ and children’s hair nicotine levels

|                        | Fathers’ hair nicotine | Change in children’s hair nicotine | 95 % CI | p value |
|------------------------|------------------------|------------------------------------|---------|---------|
| Model 1                | 0.10                   | 0.08, 0.11                         | <0.001  |
| Model 2                | 0.09                   | 0.06, 0.12                         | <0.001  |
| Model 3                | 0.07                   | 0.04, 0.10                         | <0.001  |

*Model 1: unadjusted association-only fathers’ nicotine level was considered as the exposure variable in the model

Model 2: Model 1 plus adjusted for family income, parity and fathers’ education

Model 3: Model 2 plus adjusted for mothers’ nicotine levels
exposure in public places where smoking restriction was not applied or due to exposure to cigarette smoke from smoking family members in the household. In Malaysia, strict policies on smoking are not applied and this can cause abundant exposure to SHS [26].

The main strength of this study was the measurement of hair nicotine biomarker using GC–MS to determine SHS exposure rather than merely relied on self-reported information using questionnaire, which tends to cause bias or obtaining incorrect information from the participants.

However, few limitations were acknowledged. The total number of smokers in each household was not identified and other family members who were smokers were not recorded. Also, the smoking fathers were not being asked whether they smoked in the presence of their children or not. The history of oral contraceptive pill consumption and the application of chemical treatment to hair were also not recorded. These could be considered by future researchers.

**Conclusion**

This is one of the few studies in the developing country settings to investigate the nicotine level of SHS-exposed women and children. We found that fathers’ hair nicotine level has positive relationship with mothers’ and children’s hair nicotine. Living with smoking fathers can lead to higher hair nicotine levels in children but not in mothers. The findings of this study can be used as a reference by policy makers to create new regulation regarding smoking in the presence of a child. It also can elevate awareness, especially among Malaysians population about the possible negative impact of smoking on their children. Knowledge on health effects of SHS exposure must be improved to prevent tobacco-related diseases.

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**Compliance with ethical standards**

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

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