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Paternal smoking and maternal protective behaviors at home on infant's saliva cotinine levels

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Running title: Home smoking exposure in infants

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Abstract

Background

We investigated the association between paternal smoking, avoidance behaviors and maternal protective actions and smoke-free home rules with infant's saliva cotinine in Hong Kong.

Methods

675 non-smoking mothers (mean age 32.6 years) who attended the maternal-child health clinics with their newborns aged ≤18 months completed a questionnaire about paternal smoking and avoidance behaviors, maternal protective actions, smoke-free rules at home and infant's SHS exposure. 389 infants provided saliva sample and its cotinine was tested.

Results

The geometric mean of infant's saliva cotinine was 1.07 ng/ml (95% confidence interval [CI] 0.98, 1.16). Infants living in smoking families with SHS exposure had significantly higher cotinine level than in non-smoking families (adjusted β=0.25, 95%CI 0.16, 0.33). Paternal smoking near infants (within 1.5 meters) was associated with higher cotinine level (adjusted β=0.60, 95%CI 0.22, 0.98), which was not reduced by avoidance behaviors (e.g., smoking in kitchen or balcony). Even fathers smoking ≥ 3 meters away, infants had higher cotinine level than non-smoking families (adjusted β=0.09, 95%CI 0.01, 0.16). Maternal protective actions and smoke-free home rules were not significantly associated with reduced cotinine level.

Conclusion

Paternal smoking avoidance, maternal protective actions and smoke-free policy at home did not reduce infant's saliva cotinine.

Introduction

Secondhand smoke (SHS) in children causes adverse health effects and increases medical costs and the likelihood of smoking initiation (1, 2). Home is the major source of SHS exposure in children. Some smokers are aware of the harms of SHS and make efforts to avoid exposing children to SHS (3). SHS Exposure in young children is best measured through the biomarker saliva cotinine as it is non-invasive and easy to conduct (4). Total smoking ban at home (smoke-free home) predicted more smoking reduction and cessation and was associated with lower cotinine level in children than any partial smoking ban which is defined as allowing smoking at home at specific places or times (5-7). However, adoption of smoke-free home rules is still uncommon. For instance, only less than half (~40%) of the families in the United State and United Kingdom adopts smoke-free home rules (8, 9). One previous study has found that interventions to protect children from SHS at home through encouraging parents to reduce smoking or quit had non-significant effects on reducing cotinine level (10). More in-depth understanding of parent's smoking behaviors at home and the protective strategies for SHS exposure may inform and provide information to design more effective interventions.

Predictors of SHS exposure in children at home have been widely studied, but little is known about the details of parental smoking and avoidance behaviors at home, and non-smoking parent's protective actions on reducing SHS exposure in children (11). Previous studies have differentiated children's SHS exposure by the place where father smokes (indoors or outdoors) (12), or by the degree of smoking ban enacted at home (complete, partial or nil) (13). These studies support the importance of complete smoking ban at home but many people still allow smoking at home without any restrictions or just restrict smoking in specific rooms. In contrast, we cannot identify any studies investigating the association between the places where fathers smoke and infant's SHS exposure at home. A previous study has found that one-third (29.3%) of smoking fathers smoked near the children (<3 meters) at home in Hong Kong while non-smoking mothers attempted to reduce the

harms of SHS in children by enacting protective actions such as “opening the windows”, “asking the father not to smoke near the child”, “moving the child away from the smoke” and “remove the ashtray” (14). However, it remains unclear how these smoking avoidance and SHS protective behaviors at home affect children’s cotinine level.

There is no safe level of SHS exposure, and most guidelines recommended a total smoking ban at home (15). Children living in multi-unit housings are particularly prone to SHS exposure due to shared ventilation system and crowd living environment (16). This is particularly a concern in densely populated cities such as Hong Kong where children live in small and crowded apartments. Comprehensive smoke-free legislation which bans smoking in most public indoor places has been implemented since 2007 with an observed displacement of SHS into the home of young children and an increase in mother’s SHS protective behaviors (14, 17). We investigated the associations between paternal smoking and avoidance behaviors, maternal SHS protective behaviors and smoke-free rules at home with infant’s saliva cotinine level.

Methods

Subjects

From April to September 2012, 771 non-smoking mothers who had new born aged ≤18 months were recruited from 4 major Maternal and Child Health Centers (MCHCs) in Hong Kong (response rate: 66%). All mothers completed a self-administered questionnaire which collected information on family smoking status, paternal smoking and avoidance behaviors, maternal protective actions and smoke-free rules at home that aimed to protect children from SHS exposure. Current analysis only included non-smoking families or smoking families with the father as the only smoker (n=675) with valid cotinine samples (n=389). Family smoking status was categorized into 3 types: non-smoking families (no smokers in the family), smoking families without SHS exposure at home (had a smoking father who did not smoke at home), and smoking families with SHS exposure at home (had a

smoking father who smoked at home). This study gained ethical approval from the Institutional Review Board of the University of Hong Kong/ Hospital Authority Hong Kong West Cluster.

Measurements

Salivary cotinine was used to objectively measure infant's SHS exposure as it correlates well with parent-reported SHS exposure in young children (18) and avoids the limitation of other methods such as parental concerns about invasive procedure for serum sampling (19), limited infant hair growth for hair nicotine (20), and uncontrolled excretion of urine samples for cotinine test (21). Trained research staff used sorbettes (a wand with a small sponge on the end) to collect saliva samples from the infants and stored them in the 2 ml tubes, which would be immediately frozen in ice-pads and transferred to laboratory freezer. The samples were sent for assay by the National University of Singapore using enzyme-linked immunosorbent assay (ELISA). The lower limit of cotinine sensitivity was 0.05 ng/ml (range: 0.06 to 200 ng/ml)(22).

In the questionnaire, non-smoking mothers reported the places and the distance from children when the father smoked at home, their protective actions and smoke-free home rules. The mothers reported yes or no to 8 of the *places at home where the father smoked*, that included (1) mother's and father's bedroom, (2) living room, (3) dining room, (4) bathroom, (5) kitchen, (6) balcony, (7) at child bedroom and (8) rooftop. The *distance from children when the father smoked at home* was assessed by 2 dichotomous (yes/no) questions of (1) "Does father smoke ≤3 meters from the child at home?" and (2) "Does father smoke ≤1.5 meters from the child at home?" The answers of the 2 questions were then combined and summarized as ">3 meters", "3-1.51 meters" and "≤1.5 meters".

Maternal SHS avoidance actions were measured by asking: "When smokers smoke at home, how often do you: (1) take the child away from the smoke?, (2) open the windows?, (3) post a 'No Smoking' sign at home?, (4) encourage father to reduce smoking/quit?, (5) encourage father not to smoke inside the home?, and (6) encourage father not to smoke near the child?" *Smoke-free home rules* were assessed by the following questions: "if mothers asked smokers to extinguish cigarettes

before entering the home" and "if mothers banned any smoking inside home at the following 7 places: (i) living/dining room, (ii) bathroom, (iii) kitchen, (iv) mother's bedroom, (v) child's bedroom, (vi) balcony and (vii) within 3 meters of the child". The responses for each action/rule were "always," "usually," "rarely," and "never" and were dichotomized as always/usually versus none/rarely. The total number were summed into 0-6 for avoidance action and 0-8 for smokefree rules.

Other collected information included socio-demographic characteristics (infant's gender, the highest parental education, household income, and housing type) and infant's SHS exposure outside the home, which was defined as exposed to SHS at any of the following places: public areas (e.g., bus stops, streets, and parks), outside restaurants, inside the building that they lived (e.g., corridors and garbage room), someone's home and never exposed.

Statistics analysis

Due to the skewed distribution, the salivary cotinine data was log-transformed and geometric mean was used. Cotinine levels between socio-demographic characteristics were compared using independent samples t-test or ANOVA. Generalized linear model (β-coefficient) with the adjustment of children's age and SHS exposure outside the home, parental highest educational attainment, housing types and household income (Model 1). To examine the effects of paternal smoking behaviors and maternal SHS protective actions at home, only households with reported paternal smoking at home (N=90) were included. Children's cotinine level was compared separately between (Model 2) and mutually for (Model 3) paternal smoking behaviors (places and distance from children), maternal SHS avoidance actions and smoke-free rules in smoking households by using generalized linear model (β-coefficient).

Results

There was little difference in the socio-demographic characteristics and smoking status between the families with and without valid infant's cotinine level (Supplementary Table S1). Among the 389

infants who provided valid cotinine samples, 89.7% of their parents had senior secondary or above education; 60.4% had monthly household income ≥HK$20,000 (US$1 = HK$7.8); 42.5% lived in private housings; and 37.0% had a smoking father in which 62.5% were exposed to SHS at home (Table 1).

The overall geometric mean of cotinine levels was 1.07 ng/ml. It was significantly higher in smoking families with SHS exposure (1.63 ng/ml) when compared with the smoking families without SHS exposure (0.90 ng/ml) and the non-smoking families (0.95 ng/ml) ($p < .001$). Fathers smoking at a distance very close to the children (≤ 1.5m) was significantly associated with higher cotinine level in children when compared with smoking 1.5m and 3m away from the children (6.23 versus 1.88 and 1.36 ng/ml, $p = .003$). Infant's cotinine levels were higher if fathers smoked in self-bedroom (19.11 vs. 1.54 ng/ml, $p = 0.001$) and living rooms (4.69 vs. 1.49 ng/ml, $p = 0.007$) (Figure 1). Maternal avoidance actions and places at home with smoking ban were not associated with infant's cotinine level (Figure 2 and 3).

Infants in the families with SHS exposure had significantly higher cotinine level than in non-smoking families (adjusted β= 0.25 95%CI 0.16, 0.33) (Table 2). The number of places where father smoked was associated with increased infant's cotinine levels in the crude model (β=0.16, 95%CI: 0.03, 0.30) and the model adjusting for socio-demographic characteristics (adjusted β=0.15, 95%CI: 0.18, 0.29), but became non-significant after adjusting for other paternal smoking and maternal protective actions. Fathers smoked ≤ 1.5 m around the children was associated with increased infant's cotinine levels in the crude (β=0.66, 95%CI: 0.28,1.04) and all other models adjusted for socio-demographic characteristics (adjusted β=0.67, 95%CI: 0.30, 1.04), plus the number of places at home where fathers smoked (adjusted β=0.62, 95%CI: 0.26, 0.99) and plus maternal protective actions (adjusted β=0.60, 95%CI: 0.22 to 0.98). Infant's cotinine level was not associated with maternal protective actions and the number of smoke-free home rules in the adjusted models.

Discussion

To our best knowledge, only two studies examined the factors that associated with SHS exposure of infants in the East (23, 24). Our results are in line with Baheiraei et al.'s study that infant's SHS exposure is associated with home smoking restrictions (24). Also, our study is the first to comprehensively investigate the associations between paternal smoking and avoidance behaviors, regarding the places of smoking and the distance away from the infants during smoking, maternal SHS protective actions and smoke-free home rules with infant's saliva cotinine level. In this study, more than one-fifth (21.1%) of the infants were exposed to SHS from paternal smoking at home. It is lower than the previous local study in infants (33.4%)(25) while we only included home exposure prevalence. Meanwhile, it is much lower than the preschoolers (63.2%)(26), probably due to stronger maternal protection and unacceptability of exposing infants to SHS by fathers.

SHS exposure at home was significantly associated with substantial increase in saliva cotinine levels in infants ($p<.001$). In this study, the cotinine levels among infants who were exposed to paternal smoking at home were just slightly lower (1.63 ng/ml) than the school-aged children (1.75 ng/ml) who lived with smoking parents without home smoking restriction in the UK (27). Such intense SHS exposure is particularly detrimental to infants, suggesting that effective interventions to reduce the harms are needed. Given that there is no safe level of SHS exposure, the small effect sizes for increased cotinine in related to number of places of father smoked ($\beta=0.13$, $p<0.05$) and distance between father smoked and children ($\beta=0.48$, $p<0.01$) are clinically significant. Although one previous study has reported that preschool-age girls are more exposed to SHS than boys (28), we did not find such difference in infants whose mobility is still limited in our study, no matter in smoking (mean saliva cotinine level in boys: 1.29 ng/ml; in girls: 1.13 ng/ml; $p = .98$) or non-smoking families (mean saliva cotinine level in boys: 0.87 ng/ml; in girls: 1.19 ng/ml; $p = .18$) (not shown in tables).

Similar cotinine levels were observed in infants living in non-smoking families (0.95 ng/ml) and smoking families without SHS exposure at home (0.89 ng/ml). The results suggested the protective effect of the total smoking ban on children's SHS exposure at home. Cotinine levels in infants living in non-smoking families in our studies (0.95 ng/ml) were higher than living with non-smoking parents (0.14 ng/ml) in the UK (27). This might be due to the prevalent and intense SHS exposure outside home in Hong Kong, where smoking is banned in most indoor public areas but common in the streets with very narrow pavements. Small housing units with windows and doors that are close to each other may facilitate SHS diffusion from neighbors as we found in the previous study (29).

We found that 31.3% infants were exposed to father's smoking in 3 meters distance, which was higher than the previous local study (21.3%) in infants in 1997 (25), and also the studies in the US (27.5%) (30) and Japan (14.4%) (31). Smoking near children (<3 meters) was associated with higher odds of hospitalization and significant economic burden (25, 32). Our findings suggested that there would be an increase in disease burden for SHS exposure in infants. In this study, the 6.3% infants who were exposed to smoking within 1.5 meters had significantly increased cotinine levels. Even for fathers who smoked at a distance of 3 meters away, infant's cotinine levels were still significantly higher than those without SHS exposure at home. Smoking near children is abhorred but smoking 3 meters away from children at home is difficult in practice in Hong Kong due to the typical small living flats. Smokers try to avoid exposing children to SHS by smoking in the kitchen, bathroom or balcony. However, we found that smoking in these areas was not associated with reduction in infant's cotinine levels. In this regards, none has similar detail data on places of smoking at home like our study. Western studies found the association between smoking outside the home with doors closed and reduced SHS exposure in infants (≤1-year) (33). However, smoking outside the home in the corridors, especially in publicly subsidized housing estates, is mostly prohibited in Hong Kong.

A previous study suggested the important role of engagement in avoidance actions and endorsement of smoke-free home policies by non-smoking mothers in protecting children from SHS exposure (34), especially those in the smoking family. However, we found relatively independent relationship between mother's specific and the number of SHS protection actions, smoke-free home rules and reduced cotinine level in infants after adjusted for father's smoking behaviors. The findings further support that partial smoking restriction at home was ineffective in reducing SHS in children (35). Recent studies have suggested that third-hand smoke residues that linger on surfaces and in dust after smoking may increase cotinine level in nonsmokers (4). It is uncertain if the ineffective maternal SHS protective actions are specific in Chinese society, where families are generally male dominated with strong Confucianist values, and maternal challenges to paternal smoking behaviors may not be effective. Moreover, smoking avoidance behaviors and SHS protective actions are less feasible in small and crowded living units in Hong Kong and many other cities in Mainland China, Asia and elsewhere.

This study used both self-reported and objective biochemical measurements of SHS exposure (e.g. salivary cotinine). We recruited mother-infant dyads from MCHCs as over 95% of young children were registered for their health care services, mainly for immunization, in Hong Kong (36). However, we only conducted our recruitment in 4 of the 32 centers due to the limitation of resources, which might reduce the representativeness of the sample. The cross-sectional data required caution in inferring causal relationships. The self-reported questionnaire that relied on mother's memory recall is subject to recall bias. For example, mothers may not be able to accurately assess the distance between smoking fathers and children. About half of the respondents refused to provide a saliva sample for cotinine test, due to plausible reasons. Nevertheless, we did not find significant differences in the demographic characteristics and family smoking status between those who provided and those who did not. Cotinine levels in older children higher than 12 ng/ml were regarded as a possible smoker (37) and were commonly excluded from analysis for SHS exposure. However,

as our target group was infants aged 0-18 months, we did not exclude those with high saliva cotinine data given that no procedural and validity errors were identified. The cotinine level could be partially attributed to the third-hand smoke exposure which was not assessed in this study but deserves further investigation in future.

Conclusions

Infant cotinine level was significantly higher in smoking families with SHS exposure. Paternal smoking avoidance behaviors, maternal SHS protective actions, and smoke-free home rules were not effective in reducing infant cotinine. To protect children from SHS, the home should be completely smoke-free, and smokers should quit.

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Declaration of interests

None

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Figure Legend

Figure 1: Places where fathers smoked at home and infant's saliva cotinine level.

Figure 2: Maternal avoidance actions and infant's saliva cotinine level.

Figure 3: Smoke-free rules at home and infant's saliva cotinine level.

Paternal smoking and maternal protective behaviors at home on infant's saliva cotinine levels

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Abstract

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Conclusion

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Introduction

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Predictors of SHS exposure in children at home have been widely studied, but little is known about the details of parental smoking and avoidance behaviors at home, and non-smoking parent's protective actions on reducing SHS exposure in children (11). Previous studies have differentiated children's SHS exposure by the place where father smokes (indoors or outdoors) (12), or by the degree of smoking ban enacted at home (complete, partial or nil) (13). These studies support the importance of complete smoking ban at home but many people still allow smoking at home without any restrictions or just restrict smoking in specific rooms. In contrast, we cannot identify any studies investigating the association between the places where fathers smoke and infant's SHS exposure at home. A previous study has found that one-third (29.3%) of smoking fathers smoked near the children (<3 meters) at home in Hong Kong while non-smoking mothers attempted to reduce the

harms of SHS in children by enacting protective actions such as “opening the windows”, “asking the father not to smoke near the child”, “moving the child away from the smoke” and “remove the ashtray” (14). However, it remains unclear how these smoking avoidance and SHS protective behaviors at home affect children’s cotinine level.

There is no safe level of SHS exposure, and most guidelines recommended a total smoking ban at home (15). Children living in multi-unit housings are particularly prone to SHS exposure due to shared ventilation system and crowd living environment (16). This is particularly a concern in densely populated cities such as Hong Kong where children live in small and crowded apartments. Comprehensive smoke-free legislation which bans smoking in most public indoor places has been implemented since 2007 with an observed displacement of SHS into the home of young children and an increase in mother’s SHS protective behaviors (14, 17). We investigated the associations between paternal smoking and avoidance behaviors, maternal SHS protective behaviors and smoke-free rules at home with infant’s saliva cotinine level.

Methods

Subjects

From April to September 2012, 771 non-smoking mothers who had new born aged ≤18 months were recruited from 4 major Maternal and Child Health Centers (MCHCs) in Hong Kong (response rate: 66%). All mothers completed a self-administered questionnaire which collected information on family smoking status, paternal smoking and avoidance behaviors, maternal protective actions and smoke-free rules at home that aimed to protect children from SHS exposure. Current analysis only included non-smoking families or smoking families with the father as the only smoker (n=675) with valid cotinine samples (n=389). Family smoking status was categorized into 3 types: non-smoking families (no smokers in the family), smoking families without SHS exposure at home (had a smoking father who did not smoke at home), and smoking families with SHS exposure at home (had a

smoking father who smoked at home). This study gained ethical approval from the Institutional Review Board of the University of Hong Kong/ Hospital Authority Hong Kong West Cluster.

Measurements

Salivary cotinine was used to objectively measure infant's SHS exposure as it correlates well with parent-reported SHS exposure in young children (18) and avoids the limitation of other methods such as parental concerns about invasive procedure for serum sampling (19), limited infant hair growth for hair nicotine (20), and uncontrolled excretion of urine samples for cotinine test (21). Trained research staff used sorbettes (a wand with a small sponge on the end) to collect saliva samples from the infants and stored them in the 2 ml tubes, which would be immediately frozen in ice-pads and transferred to laboratory freezer. The samples were sent for assay by the National University of Singapore using enzyme-linked immunosorbent assay (ELISA). The lower limit of cotinine sensitivity was 0.05 ng/ml (range: 0.06 to 200 ng/ml)(22).

In the questionnaire, non-smoking mothers reported the places and the distance from children when the father smoked at home, their protective actions and smoke-free home rules. The mothers reported yes or no to 8 of the *places at home where the father smoked*, that included (1) mother's and father's bedroom, (2) living room, (3) dining room, (4) bathroom, (5) kitchen, (6) balcony, (7) at child bedroom and (8) rooftop. The *distance from children when the father smoked at home* was assessed by 2 dichotomous (yes/no) questions of (1) "Does father smoke ≤3 meters from the child at home?" and (2) "Does father smoke ≤1.5 meters from the child at home?" The answers of the 2 questions were then combined and summarized as ">3 meters", "3-1.51 meters" and "≤1.5 meters".

Maternal SHS avoidance actions were measured by asking: "When smokers smoke at home, how often do you: (1) take the child away from the smoke?, (2) open the windows?, (3) post a 'No Smoking' sign at home?, (4) encourage father to reduce smoking/quit?, (5) encourage father not to smoke inside the home?, and (6) encourage father not to smoke near the child?" *Smoke-free home rules* were assessed by the following questions: "if mothers asked smokers to extinguish cigarettes

before entering the home" and "if mothers banned any smoking inside home at the following 7 places: (i) living/dining room, (ii) bathroom, (iii) kitchen, (iv) mother's bedroom, (v) child's bedroom, (vi) balcony and (vii) within 3 meters of the child". The responses for each action/rule were "always," "usually," "rarely," and "never" and were dichotomized as always/usually versus none/rarely. The total number were summed into 0-6 for avoidance action and 0-8 for smokefree rules.

Other collected information included socio-demographic characteristics (infant's gender, the highest parental education, household income, and housing type) and infant's SHS exposure outside the home, which was defined as exposed to SHS at any of the following places: public areas (e.g., bus stops, streets, and parks), outside restaurants, inside the building that they lived (e.g., corridors and garbage room), someone's home and never exposed.

Statistics analysis

Due to the skewed distribution, the salivary cotinine data was log-transformed and geometric mean was used. Cotinine levels between socio-demographic characteristics were compared using independent samples t-test or ANOVA. Generalized linear model (β-coefficient) with the adjustment of children's age and SHS exposure outside the home, parental highest educational attainment, housing types and household income (Model 1). To examine the effects of paternal smoking behaviors and maternal SHS protective actions at home, only households with reported paternal smoking at home (N=90) were included. Children's cotinine level was compared separately between (Model 2) and mutually for (Model 3) paternal smoking behaviors (places and distance from children), maternal SHS avoidance actions and smoke-free rules in smoking households by using generalized linear model (β-coefficient).

Results

There was little difference in the socio-demographic characteristics and smoking status between the families with and without valid infant's cotinine level (Supplementary Table S1). Among the 389

infants who provided valid cotinine samples, 89.7% of their parents had senior secondary or above education; 60.4% had monthly household income ≥HK$20,000 (US$1 = HK$7.8); 42.5% lived in private housings; and 37.0% had a smoking father in which 62.5% were exposed to SHS at home (Table 1).

The overall geometric mean of cotinine levels was 1.07 ng/ml. It was significantly higher in smoking families with SHS exposure (1.63 ng/ml) when compared with the smoking families without SHS exposure (0.90 ng/ml) and the non-smoking families (0.95 ng/ml) ($p < .001$). Fathers smoking at a distance very close to the children (≤ 1.5m) was significantly associated with higher cotinine level in children when compared with smoking 1.5m and 3m away from the children (6.23 versus 1.88 and 1.36 ng/ml, $p = .003$). Infant's cotinine levels were higher if fathers smoked in self-bedroom (19.11 vs. 1.54 ng/ml, $p = 0.001$) and living rooms (4.69 vs. 1.49 ng/ml, $p = 0.007$) (Figure 1). Maternal avoidance actions and places at home with smoking ban were not associated with infant's cotinine level (Figure 2 and 3).

Infants in the families with SHS exposure had significantly higher cotinine level than in non-smoking families (adjusted β= 0.25 95%CI 0.16, 0.33) (Table 2). The number of places where father smoked was associated with increased infant's cotinine levels in the crude model (β=0.16, 95%CI: 0.03, 0.30) and the model adjusting for socio-demographic characteristics (adjusted β=0.15, 95%CI: 0.18, 0.29), but became non-significant after adjusting for other paternal smoking and maternal protective actions. Fathers smoked ≤ 1.5 m around the children was associated with increased infant's cotinine levels in the crude (β=0.66, 95%CI: 0.28,1.04) and all other models adjusted for socio-demographic characteristics (adjusted β=0.67, 95%CI: 0.30, 1.04), plus the number of places at home where fathers smoked (adjusted β=0.62, 95%CI: 0.26, 0.99) and plus maternal protective actions (adjusted β=0.60, 95%CI: 0.22 to 0.98). Infant's cotinine level was not associated with maternal protective actions and the number of smoke-free home rules in the adjusted models.

Discussion

To our best knowledge, only two studies examined the factors that associated with SHS exposure of infants in the East (23, 24). Our results are in line with Baheiraei et al.'s study that infant's SHS exposure is associated with home smoking restrictions (24). Also, our study is the first to comprehensively investigate the associations between paternal smoking and avoidance behaviors, regarding the places of smoking and the distance away from the infants during smoking, maternal SHS protective actions and smoke-free home rules with infant's saliva cotinine level. In this study, more than one-fifth (21.1%) of the infants were exposed to SHS from paternal smoking at home. It is lower than the previous local study in infants (33.4%)(25) while we only included home exposure prevalence. Meanwhile, it is much lower than the preschoolers (63.2%)(26), probably due to stronger maternal protection and unacceptability of exposing infants to SHS by fathers.

SHS exposure at home was significantly associated with substantial increase in saliva cotinine levels in infants ($p<.001$). In this study, the cotinine levels among infants who were exposed to paternal smoking at home were just slightly lower (1.63 ng/ml) than the school-aged children (1.75 ng/ml) who lived with smoking parents without home smoking restriction in the UK (27). Such intense SHS exposure is particularly detrimental to infants, suggesting that effective interventions to reduce the harms are needed. Given that there is no safe level of SHS exposure, the small effect sizes for increased cotinine in related to number of places of father smoked ($\beta=0.13$, $p<0.05$) and distance between father smoked and children ($\beta=0.48$, $p<0.01$) are clinically significant. Although one previous study has reported that preschool-age girls are more exposed to SHS than boys (28), we did not find such difference in infants whose mobility is still limited in our study, no matter in smoking (mean saliva cotinine level in boys: 1.29 ng/ml; in girls: 1.13 ng/ml; $p = .98$) or non-smoking families (mean saliva cotinine level in boys: 0.87 ng/ml; in girls: 1.19 ng/ml; $p = .18$) (not shown in tables).

Similar cotinine levels were observed in infants living in non-smoking families (0.95 ng/ml) and smoking families without SHS exposure at home (0.89 ng/ml). The results suggested the protective effect of the total smoking ban on children's SHS exposure at home. Cotinine levels in infants living in non-smoking families in our studies (0.95 ng/ml) were higher than living with non-smoking parents (0.14 ng/ml) in the UK (27). This might be due to the prevalent and intense SHS exposure outside home in Hong Kong, where smoking is banned in most indoor public areas but common in the streets with very narrow pavements. Small housing units with windows and doors that are close to each other may facilitate SHS diffusion from neighbors as we found in the previous study (29).

We found that 31.3% infants were exposed to father's smoking in 3 meters distance, which was higher than the previous local study (21.3%) in infants in 1997 (25), and also the studies in the US (27.5%) (30) and Japan (14.4%) (31). Smoking near children (<3 meters) was associated with higher odds of hospitalization and significant economic burden (25, 32). Our findings suggested that there would be an increase in disease burden for SHS exposure in infants. In this study, the 6.3% infants who were exposed to smoking within 1.5 meters had significantly increased cotinine levels. Even for fathers who smoked at a distance of 3 meters away, infant's cotinine levels were still significantly higher than those without SHS exposure at home. Smoking near children is abhorred but smoking 3 meters away from children at home is difficult in practice in Hong Kong due to the typical small living flats. Smokers try to avoid exposing children to SHS by smoking in the kitchen, bathroom or balcony. However, we found that smoking in these areas was not associated with reduction in infant's cotinine levels. In this regards, none has similar detail data on places of smoking at home like our study. Western studies found the association between smoking outside the home with doors closed and reduced SHS exposure in infants (≤1-year) (33). However, smoking outside the home in the corridors, especially in publicly subsidized housing estates, is mostly prohibited in Hong Kong.

A previous study suggested the important role of engagement in avoidance actions and endorsement of smoke-free home policies by non-smoking mothers in protecting children from SHS exposure (34), especially those in the smoking family. However, we found relatively independent relationship between mother's specific and the number of SHS protection actions, smoke-free home rules and reduced cotinine level in infants after adjusted for father's smoking behaviors. The findings further support that partial smoking restriction at home was ineffective in reducing SHS in children (35). Recent studies have suggested that third-hand smoke residues that linger on surfaces and in dust after smoking may increase cotinine level in nonsmokers (4). It is uncertain if the ineffective maternal SHS protective actions are specific in Chinese society, where families are generally male dominated with strong Confucianist values, and maternal challenges to paternal smoking behaviors may not be effective. Moreover, smoking avoidance behaviors and SHS protective actions are less feasible in small and crowded living units in Hong Kong and many other cities in Mainland China, Asia and elsewhere.

This study used both self-reported and objective biochemical measurements of SHS exposure (e.g. salivary cotinine). We recruited mother-infant dyads from MCHCs as over 95% of young children were registered for their health care services, mainly for immunization, in Hong Kong (36). However, we only conducted our recruitment in 4 of the 32 centers due to the limitation of resources, which might reduce the representativeness of the sample. The cross-sectional data required caution in inferring causal relationships. The self-reported questionnaire that relied on mother's memory recall is subject to recall bias. For example, mothers may not be able to accurately assess the distance between smoking fathers and children. About half of the respondents refused to provide a saliva sample for cotinine test, due to plausible reasons. Nevertheless, we did not find significant differences in the demographic characteristics and family smoking status between those who provided and those who did not. Cotinine levels in older children higher than 12 ng/ml were regarded as a possible smoker (37) and were commonly excluded from analysis for SHS exposure. However,

as our target group was infants aged 0-18 months, we did not exclude those with high saliva cotinine data given that no procedural and validity errors were identified. The cotinine level could be partially attributed to the third-hand smoke exposure which was not assessed in this study but deserves further investigation in future.

Conclusions

Infant cotinine level was significantly higher in smoking families with SHS exposure. Paternal smoking avoidance behaviors, maternal SHS protective actions, and smoke-free home rules were not effective in reducing infant cotinine. To protect children from SHS, the home should be completely smoke-free, and smokers should quit.

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Declaration of interests

None

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Table 1. Infant's saliva cotinine levels by different characteristics (N=379).

	N (%)	Cotinine level Geometric Mean (ng/ml) (95% CI)	p
Total	389 (100.0)	1.07 (0.98, 1.16)	
Sex			.25
Male	204 (52.4)	1.02 (0.91, 1.14)	
Female	185 (47.6)	1.12 (0.98, 1.28)	
Parental highest education			.16
Junior secondary	40 (10.3)	0.90 (0.73, 1.09)	
Senior secondary	186 (47.8)	1.15 (1.00, 1.33)	
Post-secondary	163 (41.9)	1.02 (0.90, 1.14)	
Family income (HKD/month, USD 1=HKD 7.8)			.75
<10,000	40 (10.3)	1.13 (0.76, 1.69)	
10,000-19,999	114 (29.3)	1.13 (0.96, 1.33)	
20,000-29,999	84 (21.6)	1.01 (0.86, 1.18)	
≥30,000	151 (38.8)	1.04 (0.91, 1.18)	
Housing type			.053
Public rental housing	138 (35.5)	1.19 (1.00, 1.41)	
Subsidized sale flats	65 (16.7)	0.92 (0.75, 1.13)	
Private permanent housing	161 (41.4)	0.99 (0.88, 1.10)	
Others	25 (6.4)	1.40 (1.03, 1.92)	
Had outdoor SHS exposure in past 7 days			.10
Yes	212 (54.5)	1.00 (0.90, 1.11)	
No	177 (45.5)	1.15 (1.00, 1.33)	
Family smoking and SHS exposure at home			< .001
Non-smoking family	245 (63.0)	0.95 (0.87, 1.04)	
Smoking family without SHS	54 (13.9)	0.90 (0.72, 1.11)	
Smoking family with SHS	90 (23.1)	1.63 (1.30, 2.04)	
Paternal smoking distance to children at home (meter) [a]			.003
>3	62 (68.9)	1.36 (1.04, 1.77)	
3-1.51	22 (24.4)	1.88 (1.30, 2.73)	
≤1.5	6 (6.7)	6.23 (1.36, 28.50)	

[a] Among smoking families with SHS (N=90).

Table 2. Associations of family smoking status and secondhand smoke exposure at home, father smoking behaviors, and maternal SHS avoidance actions and smokefree home rules with infant's cotinine levels.

	Log_{10}-transformed saliva cotinine level (ng/ml) β-coefficient (95% CI)			
	Crude	**Model 1 [a]**	**Model 2 [b]**	**Model 3 [c]**
Among all families which provided saliva (n = 389)				
Family smoking status and SHS exposure at home				
Non-smoking families	Ref.	Ref.	--	--
Smoking families without SHS	-0.03 (-0.12, 0.07)	-0.02 (-0.09, 0.12)	--	--
Smoking families with SHS	0.24 (0.15, 0.32)***	0.25 (0.16, 0.33)***	--	--
Distance that fathers smoked around the children				
>3	0.16 (0.06, 0.25)**	0.09 (0.01, 0.16)*	--	--
3-1.51	0.30 (0.16, 0.44)***	0.30 (0.15, 0.45)***	--	--
≤1.5	0.82 (0.55, 1.08)***	0.68 (0.42, 0.94)***	--	--
Among smoking families with SHS (n = 90)				
Paternal smoking behaviors				
Number of places where fathers smoked (0-6)	0.16 (0.03, 0.30)*	0.15 (0.18, 0.29)*	0.11 (-0.03, 0.24)	0.11 (-0.03, 0.25)
Distance that fathers smoked around the children				
> 3meters	Ref.	Ref.	Ref.	Ref.
3-1.51 meters	0.14 (-0.08, 0.36)	0.21 (-0.001, 0.42)	0.17 (-0.05, 0.39)	0.15 (-0.07, 0.37)
≤ 1.5 meter	0.66 (0.28, 1.04)***	0.67 (0.30, 1.04)***	0.62 (0.26,0.99)***	0.60 (0.22, 0.98)**
Maternal protective behaviors				
Number of smokefree rules at home (0-8)	-0.02 (-0.06, 0.02)	-0.01 (-0.05, 0.02)	-0.02 (-0.06, 0.02)	-0.001 (-0.04, 0.04)
Number of maternal SHS avoidance actions (0-6)	0.07 (-0.05, 0.18)	0.06 (-0.05, 0.17)	0.08 (-0.03, 0.20)	0.04 (-0.08, 0.15)

Note.

[a] Model 1: Adjusting for infant's age, household income, parental highest education attainment, type of housing and outdoor SHS exposure.

[b] Model 2: All adjusted for Model 1 variables; for paternal smoking behaviors mutually adjusted for the number of smoking places and distance; and for mother protective behaviors mutually adjusted for numbers of smokefree home rules and avoidance actions.

[c] Model 3: Adjusting for Model 1 variables and mutually adjusted for all variables in the table.

***p-value <0.001, **p-value <0.01 and *p-value<0.05

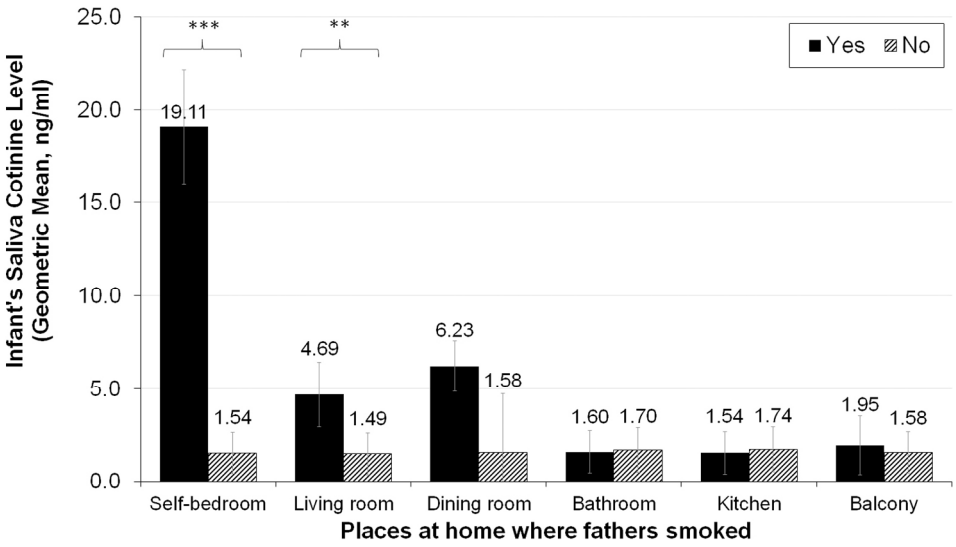

Figure 1: Places where fathers smoked at home and infant's saliva cotinine level.

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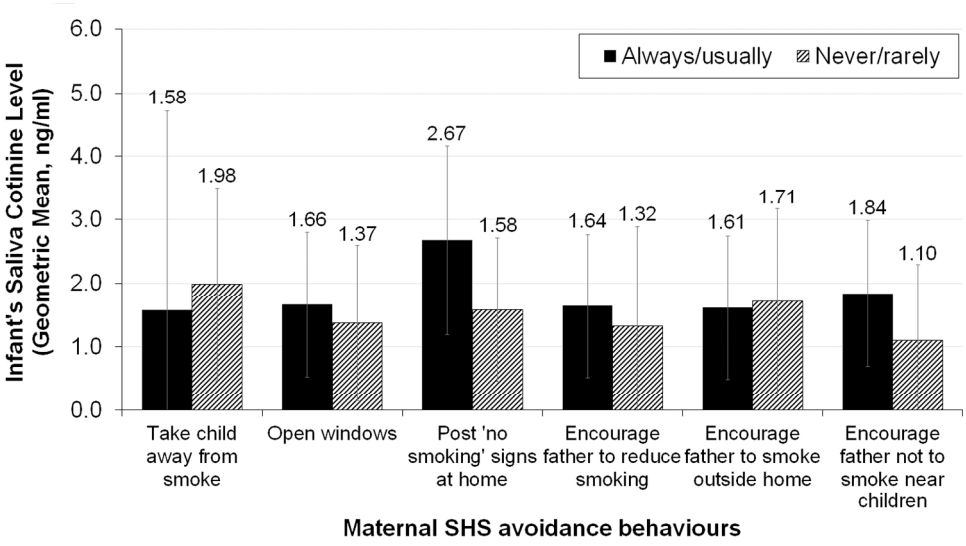

Maternal avoidance actions and infant's saliva cotinine level.

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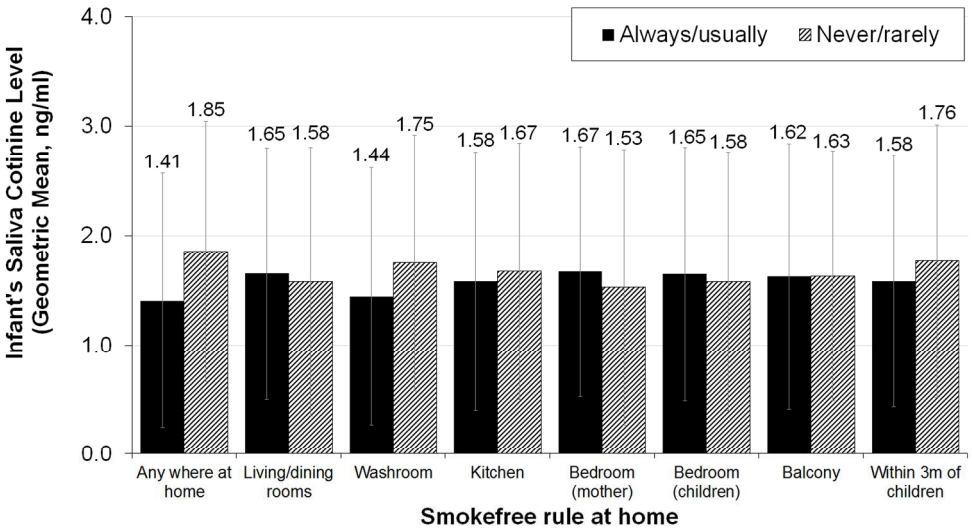

Smoke-free rules at home and infant's saliva cotinine level.

199x116mm (300 x 300 DPI)

Supplementary Table S1
Children characteristics, SHS exposure and family characteristics between those who provided and did not provide salivary cotinine. [a]

	Total		Without cotinine		With cotinine		Effect size
N (%)	675 (100.0)		286 (42.4)		389 (57.6)		
Mother's age (years), mean(SD)	32.6 (4.5)		32.4 (4.6)		32.8 (4.5)		0.095
Infant's age (months), mean(SD)	6.5 (5.7)		5.8 (6.0)		7.0 (5.4)		0.214
	n	%	n	%	n	%	
Sex							0.020
Male	348	51.6	144	50.5	204	52.4	
Female	326	48.4	141	49.5	185	47.6	
Parental education							0.032
Junior secondary	80	11.9	37	13.0	43	11.1	
Senior secondary	297	44.1	122	42.8	175	45.0	
Post-secondary	297	44.1	126	44.2	171	44.0	
Family income (HKD/month, USD 1=HKD 7.8)							0.040
<10,000	68	10.1	28	9.9	40	10.3	
10,000-19,999	200	29.8	86	30.5	114	29.3	
20,000-29,999	152	22.7	68	24.1	84	21.6	
≥30,000	250	37.4	100	35.5	151	38.8	
Housing type							0.042
Public rental housing	235	34.9	97	34.0	138	35.6	
Subsidized sale flats	115	17.1	50	17.5	65	16.8	
Private permanent housing	286	42.5	125	43.9	161	41.5	
Others	37	5.5	13	4.6	24	6.2	
Had outdoor SHS exposure in past 7 days							0.017
Yes	363	53.8	151	52.8	212	54.5	
No	312	46.2	135	47.2	177	45.5	
Family smoking & SHS exposure at home							0.040
Non-smoking family	415	61.5	170	59.4	245	63.0	
Smoking family without SHS	101	15.0	47	16.4	54	13.9	
Smoking family with SHS	159	23.6	69	24.1	90	23.1	
Paternal smoking distance to children at home (meter) [a]							0.112
>3	111	69.8	49	71.0	62	68.9	
3-1.51	34	21.4	12	17.4	22	24.4	
≤1.5	14	8.8	8	11.6	6	6.7	

[a] Missing data were excluded from analysis.