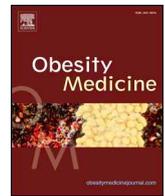




ELSEVIER

Contents lists available at ScienceDirect

## Obesity Medicine

journal homepage: [www.elsevier.com/locate/obmed](http://www.elsevier.com/locate/obmed)

Original research

# Air pollution and child obesity: Assessing the feasibility of measuring personal PM<sub>2.5</sub> exposures and behaviours related to BMI in preschool-aged children in China

Hilary Ong<sup>a,\*</sup>, David Holstius<sup>b</sup>, Yan Li<sup>c</sup>, Edmund Seto<sup>d</sup>, May Wang<sup>e</sup><sup>a</sup> School of Medicine, University of California, San Francisco, 513 Parnassus Ave, San Francisco, CA, 94143, USA<sup>b</sup> Environmental Health Sciences Division, School of Public Health, University of California, Berkeley, 2121 Berkeley Way #5302, Berkeley, CA, 94720, USA<sup>c</sup> Department of Nutritional Sciences, School of Public Health, Kunming Medical University, 1168 West Chunrong West Road, Yuhua Ave, Chengong District, Kunming, 650500, China<sup>d</sup> Department of Environmental and Occupational Health Sciences, University of Washington, Seattle, 1959 NE Pacific St, Campus Box 357234, Seattle, WA, 98195, USA<sup>e</sup> Department of Community Health Sciences, Fielding School of Public Health, University of California, Los Angeles, 640 Charles E Young Dr S, CHS 26-051B, Los Angeles, CA, 90024, USA

## ARTICLE INFO

## Keywords:

Obesity  
Air pollution  
China  
PM<sub>2.5</sub>  
Tobacco smoke

## ABSTRACT

**Background:** Accumulating evidence suggests that air pollutant exposures affect multiple metabolic functions which may place populations at a higher risk for obesity. A pilot study was conducted to assess the feasibility of measuring personal exposures to fine particulate matter (PM<sub>2.5</sub>) and tobacco smoke, to assess the influence of these exposures on the risk of obesity in children in Kunming, a rapidly developing city in China.

**Methods:** We recruited 52 children ages 4–5 years from three preschools in Kunming. Individual-level PM<sub>2.5</sub> exposures were assessed using real-time personal monitors. Tobacco smoke exposures were assessed by a parent-reported questionnaire.

**Results:** Subjects' daily mean PM<sub>2.5</sub> exposure (n = 52) was 32.8 (SD = 13.4) µg m<sup>-3</sup> and daily tobacco exposure was 14.4 (SD = 21.1) cigarettes day<sup>-1</sup>. Our subjects' daily mean exposure to PM<sub>2.5</sub> was 31% higher than the level set by the WHO daily ambient concentration guidelines.

**Conclusion:** We found personal PM<sub>2.5</sub> monitoring to be a feasible method for measuring exposure to PM<sub>2.5</sub> in young children. Future studies with larger sample sizes are warranted to explore the relationship between air pollution exposure and risk of obesity in China.

## 1. Introduction

Child obesity has been rising rapidly in China. In urban areas, obesity rates of Chinese children aged 7–17 years have tripled in 20 years, reaching levels comparable to those in western countries (Chen, 2008). In comparison, child obesity rates in the United States tripled over a longer period of 30 years (Ogden et al., 2010). In China, the greater burden of metabolic-related diseases has been in urban areas, suggesting that physical and social environmental factors associated with urbanization play a role in increasing obesity risk (Withrow and Alter, 2011; Ji and Cheng, 2009).

The etiology of obesity is multifactorial with diet and physical activity being the two established modifiable risk factors for obesity. A

newly identified potential risk factor is air pollutants. Emerging evidence suggests air pollutants contain endocrine disrupting chemicals (EDCs) that can disrupt and alter functions of the endocrine system and increase obesity risk (Hatch et al., 2010; Baillie-Hamilton, 2002; Trasande et al., 2009; Bergman, 2013; Elobeid et al., 2010; Diamanti-Kandarakis et al., 2009; Heindel, 2003; Janesick and Blumberg, 2011; Vrijheid et al., 2012; Bolton et al., 2014).

Air particulate matter less than 2.5 µm in diameter (PM<sub>2.5</sub>) is one of the major contributors to air pollution in mega cities in China. In Beijing, one of China's largest cities, ambient concentrations of PM<sub>2.5</sub> is about ten times higher than the WHO guideline values (Chan and Yao, 2008). From a health standpoint, PM<sub>2.5</sub> or smaller particles are of particular concern because their small size allows them to penetrate

\* Corresponding author.

E-mail address: [Hong@childrensnational.org](mailto:Hong@childrensnational.org) (H. Ong).<sup>1</sup> Present address: Department of Emergency Medicine and Trauma Services, Children's National Medical Center, 111 Michigan Avenue NW, Washington, D.C., 20010, USA.

alveolar gas-exchange regions and be directly absorbed into the systemic circulation (Holtcamp, 2012). Animal data and limited human data suggest PM<sub>2.5</sub> exposure has systemic effects that can influence lipogenesis, lipid metabolism and weight homeostasis (Zou, 2010; Xu et al., 2010; Grun and Blumberg, 2009; Baillie-Hamilton, 2002; McClafferty, 2008; Brook et al., 2004; Heindel, 2003; Fliescher, 2014; Oken and Gillman, 2003; Tappy, 2006; Pei et al., 2013; Chen et al., 2012). Hence, one factor linking rapid urbanization in China and obesity risk may be increasing exposure to air pollutants and EDCs.

Exposure to environmental tobacco smoke may also contribute to obesity risk especially among children. Tobacco smoke exposure (TSE) in children is almost exclusively from second and third hand exposures. Studies have found environmental TSE to play a role in metabolic system dysfunction, to increase BMI and central obesity (Bamia et al., 2004; Carney and Goldberg, 1984; Yang, Decker & Kramer, 2013; Raum et al., 2011; Ino et al., 2011; Kwok et al., 2010). Moreover, maternal smoking is associated with increased occurrence of childhood overweight and obesity (Raum et al., 2011; Toschke et al., 2003; Verhulst et al., 2009; Lisboa et al., 2012; Pei et al., 2013; Yang et al., 2013; Chen et al., 2012). China is the largest producer and consumer of tobacco (Shi, 2009). An estimated 350 million people in China smoke, and an estimated 540 million Chinese are exposed to second hand smoke, many of them children (Shi, 2009). Considering the high rates of smoking in China, the health and cost implications of obesity and other endocrine-related chronic diseases from TSE maybe considerable.

To our knowledge, no previous studies have measured individual PM<sub>2.5</sub> and tobacco smoke exposures in young preschool-aged Chinese children. Furthermore, the relationship between these exposures and other behavioural risk factors such as diet, physical activity and BMI in young Chinese children has not been explored. We conducted a pilot study, with the primary purpose of assessing the feasibility of measuring multiple aspects of obesity risk in preschool-aged children. In particular, the study (i) developed a protocol to measure continuous individual-level ambient PM<sub>2.5</sub> exposures using novel personal monitoring technology, (ii) determined if exposures to PM<sub>2.5</sub> air pollutants are associated with BMI, and (iii) investigated if children's current and past tobacco smoke exposure is associated with their BMI.

## 2. Methods

### 2.1. Study design and sample

Fifty-two preschool children (aged 4–5 years) were recruited from three preschools in Kunming city. Preschools were selected based on likely exposure to various local air pollution sources and willingness to participate in the study. Administrators at various preschools were contacted, and with their permission, fliers with a description of the study and contact information were posted in the schools and distributed to parents and primary caretakers. Children aged 4–5 years who attended the preschool, were in good general health determined by parental self-reports were eligible, and who lived in Kunming city for at least 2 years with their families, were eligible for participation in the study. Children with chronic conditions including congenital or genetic medical problems, diabetes and malignancies were excluded from the study. Because we were limited by the number of portable air quality measurement devices available, we used a rolling recruitment of 15 children over multiple monitoring periods. Institutional Review Board approvals for this study were obtained from University of California, San Francisco, and Kunming Medical University.

### 2.2. Outcome variables

The primary outcome of interest, Body Mass Index (BMI) (kg/m<sup>2</sup>), was calculated from height and weight, and converted to z-score using relevant data from WHO reference growth charts by age and gender. Measurements were recorded to the nearest 0.1 cm (height), and 0.1 kg

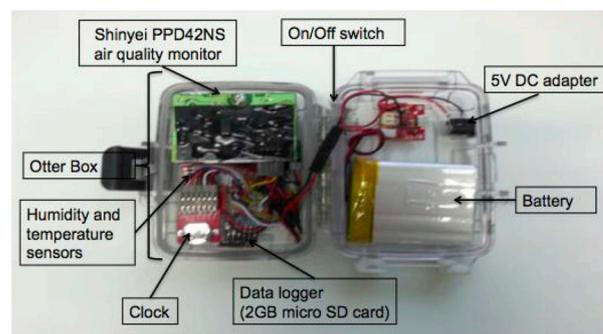


Fig. 1. Portable low-cost personal PM<sub>2.5</sub> air quality monitor.

(weight). Body weight was measured in light clothing and without shoes. Height was measured using a portable stadiometer from Weigh and Measure (<http://www.weighandmeasure.com>, Maryland, USA (ShorrBoard; Weight and Measure, LLC, Olney, MD), and weight was measured using a calibrated digital scale from Tanita (model HD-314, Tanita Corporation of America, Inc, Arlington Heights, IL). Two measurements were obtained for each measure. If measurements had a difference of more than 0.3 kg for weight and 0.2 cm for height, then a third measurement was taken. The average of the two closest measurements was used for analysis. Of note, because children in China often participate in state-mandated anthropometric measurement surveys, our study participants were familiar with measurement protocols and cooperative with data collection.

### 2.3. Exposure assessment

Children's individual-level PM<sub>2.5</sub> exposures were assessed using novel, low-cost portable PM<sub>2.5</sub> air quality monitors, called PANDA (Portable and Affordable Nephelometric Data Acquisition) (Fig. 1). The instrument is described in detail by Holstius et al. (2014). Data collection was conducted between November 2013 and January 2014. Before the monitors were used by participants, they were collocated and calibrated against tapered element oscillating microbalance (TEOM) mass concentrations data at a Kunming PM<sub>2.5</sub> monitoring station for seven days using methods described previously (Holstius et al., 2014). The PANDA monitors measured continuous time-stamped 1-min PM<sub>2.5</sub> personal exposures calibrated to mass concentration units of µg/m<sup>3</sup>. Each subject was provided a PANDA. The child's parent(s) or primary caretaker were instructed to carry the monitors wherever the child went for seven consecutive days. Families were given the following verbal and written instructions: expose monitor to open air to allow it to capture air samples; do not put monitor into any bags or backpacks; place monitor in living areas where child spends most time while at home; every night plug in battery-operated monitor while child sleeps; and keep monitors on 24-h for the assigned seven days.

Children's tobacco smoke exposure during pregnancy and current exposure were assessed by a self-administered questionnaire completed by the parent or primary caretaker. If parent or primary caretaker indicated in the questionnaire that there were household members who smoke, he/she was then asked to record the number of household members who smoke, his/her relation to the child, number of cigarettes smoked a day, and if there are restrictions on cigarette smoking inside the home.

### 2.4. Other variables of interest

Data on household socio-demographics, parents' BMI, feeding practices during infancy (breast- and/or formula-fed), and child's physical activity and sleep habits were gathered from the parent or caretaker by self-administered questionnaires, which were pretested with five local Kunming families.

Parent(s) or primary caretaker were asked to record the amount to child's vigorous physical activity in a day excluding physical activity in preschool (recorded as minutes per day). Vigorous physical activity was defined as activities such as games or exercise that makes your child breathe fast. As daily physical activity may differ between weekdays and weekends, parent(s) or primary caretaker were asked to record activity level for both weekdays and weekends. To our knowledge there is no validated questionnaire or measure of physical activity for Chinese preschool children. A study by Jiang et al. used parent-reported questionnaire to estimate children's physical activity level (Jiang et al., 2006).

In addition, parent(s) or primary caretaker were asked to maintain a diary of all food and beverages their child consumed outside of preschool for three consecutive days (two weekdays and one weekend day specified by investigator). Consecutive three-day food diaries is an often used food diary in China to survey children's food intake and nutritional status (Hui and Nelson, 2006; Sun et al., 2013). Subjects were provided with a food portion photograph book, food diary recording sheets, and written instructions on how to complete the food record. Parent(s) or primary caretaker were shown examples of a food diary and given verbal instructions on how to complete the food diary. Parent(s) or primary caretaker were asked to use the food portion photograph book estimate to the best of their ability, weights of foods and volume of beverages that their child had eaten and drank. The importance of maintaining normal diets was emphasized. Our research staff clarified missing or questionable data the day when parent(s) or primary caretaker returned the food diary and other questionnaires. Food and beverages consumed in preschool were recorded by two trained research staff. At the three participating preschools, students ate breakfast, lunch and dinner at preschool. For every meal, each child was given a standardized portion of food determined by the school. Over two consecutive days, our research staff weighed and recorded food items in two standard meal portions for every meal, and weights of each food were averaged. The number of standard portions consumed by participating students was recorded. If there was food waste, our staff measured the waste, and the weight was subtracted from the portion the student consumed.

At the end of the study, participating children and their parent(s) or primary caretaker were asked to comment on the study including appropriateness and clarity of the use of the air quality device, food diary, and self-administered questionnaires.

### 2.5. Data management

Continuous PM2.5 measurements over the seven-day monitoring period were averaged over time to assess each child's average daily exposure. BMI was converted to BMI z-score using WHO child growth standards (Stata igrowup package, <http://www.who.int/childgrowth/software/en/>). Bivariate and box plots, t-tests, and Pearson correlation coefficient (Stata version 12, Stata Corporation, Inc., College Station, TX) were some of the analytical methods used to explore relationships between exposure and outcome. Socio-economic status (SES) was determined from family monthly income and parental education, which were operationalized as ordinal variables with income given a score ranging from 1 (lowest) to 7 (highest) and education given a score ranging from 1 (lowest) to 5 (highest). These two scores were added to provide a measure of SES. The combined scores were divided into tertiles to reflect high, middle and low socio-economic groups. The staff and parental-recorded dietary intakes were analysed using FoodWorks nutrient analysis software (version 15, 2013: The Nutrition Company Ltd., Long Valley, NJ) to generate daily caloric intake (kCal/day) for each subject. When the food recorded could not be matched exactly, the best possible match was found in FoodWorks and used as a substitute. Recipes provided by subjects and schools were also entered into FoodWorks.

Physical activity level for each participant, measured as minutes per day for weekday and weekend days were totalled, and a 1-week average was calculated.

**Table 1**  
Baseline characteristics of study participants (N = 52)

Study Participants (N = 52)	
Age, months (mean (SD))	55 (4.2)
Sex, male	26 (50%)
Socio-Economic Status (SES)	
High	17 (33%)
Middle	30 (58%)
Low	5 (10%)

**Table 2**  
Descriptive statistics of variables (N = 52).

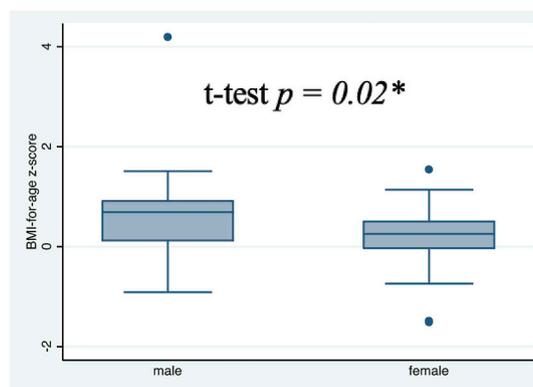
	Mean	SD	Range	Reference Values
BMI-Z	0.41	0.85	-1.51 - 4.19	-
Caloric Intake (Kcal/day)	1608	416	917-2599	1200-1400
Physical Activity (mins/day)	43.3	58.8	3.6-343	≥60
Sleep (hrs)	9.9	0.5	8.6-11.5	11-13
Exclusively Breastfed (months) (N = 42)	6.8	2.6	6-12	6
Parental BMI	22.9	2.3	18.3-28.2	18.5-24.9

### 3. Results

Half of our child study participants were male, with a mean age of 55 months (SD 4.2). Most families were in the middle socio-demographic group (Table 1). Table 2 presents descriptive statistics of health behaviours. Of health behaviours surveyed, participants had a high mean daily caloric intake (1608 kCal/day) and low mean physical activity level (43 min/day), compared to recommendations (1200-1400 kCal/day and > 60 min/day respectively). Mean BMI z-score was 0.41, which is within normal limits. Even in our small sample we found significantly higher BMI z-score for boys compared to girls (Fig. 2).

Our subjects' daily mean PM2.5 exposures by preschool ranged from 29.5 to 35.1 µg/m3. Among all preschools the total mean PM2.5 exposure was 31.8 µg/m3, a level that is 27% higher than the WHO daily ambient concentration recommendation (24-h daily mean exposure to be less than 25 µg/m3 (Table 3).

Tobacco smoke exposure (TSE) (exposure to household members who smoke) was separated into in-utero exposure during pregnancy and current exposure (Table 4). Current second-hand tobacco smoke exposure was high amongst all children (15 cigarettes/day), and essentially no different from in-utero exposure (14.4 cigarettes/day), with 31% of children having some second-hand exposure. In some cases, current exposures were higher than in-utero (e.g., one child had a mother who currently smoked, and about two times as many



\*significance at  $p < 0.05$

**Fig. 2.** BMI z-score and Gender.

\*significance at  $p < 0.05$ .

**Table 3**  
Daily mean PM2.5 exposure among study children by preschool.

Preschool	N	Daily PM2.5 Exposure*	
		Mean	SD
A1	11	29.5	0.9
A2	10	29.7	1.4
B	8	35.1	0.8
C	10	33.5	1.9
ALL	39	31.8	1.3

**Table 4**  
In-utero and current second-hand tobacco exposure (cigarettes/day) among study children (N = 52).

Source of Tobacco Smoke	In-Utero				Current			
	N		Cigarette/Day		N		Cigarette/Day	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Mother	0	0	0	0	1	2	5.0	0
Father	23	45	11.5	5.6	23	45	12.3	5.8
Grandparents	8	15	14.0	9.1	15	28	11.3	6.6
Total	31	60	14.4	9.2	31	60	15.0	8.1

% represents percent of total study participants (N = 52) who were exposed to tobacco smoke in-utero and at time of study.

grandparents were current smokers compared to when the child was in-utero). While the feasibility study had insufficient power to detect significant associations between TSE and the children's BMI, there was some indication that current TSE may be slightly higher for boys than girls (Fig. 3). No participants indicated that mothers smoked during pregnancy. In fact, almost all in-utero smoke exposure was from males of the family (father and grandfather). Similar results were reported for participants' current TSE.

Thus far, preliminary analyses of data from this limited pilot study have not revealed a relationship between PM2.5 exposure and BMI z-score (Table 5). However, a small positive correlation was found between BMI z-score and caloric intake, physical activity, and SES.

**4. Discussion**

Our pilot study found novel low-cost personal PM2.5 monitoring to be feasible for young children with the involvement of their parents. Parents were able to provide an assessment of their children's eating and

**Table 5**  
Correlation coefficients for exposure, outcomes and covariates (N = 36).

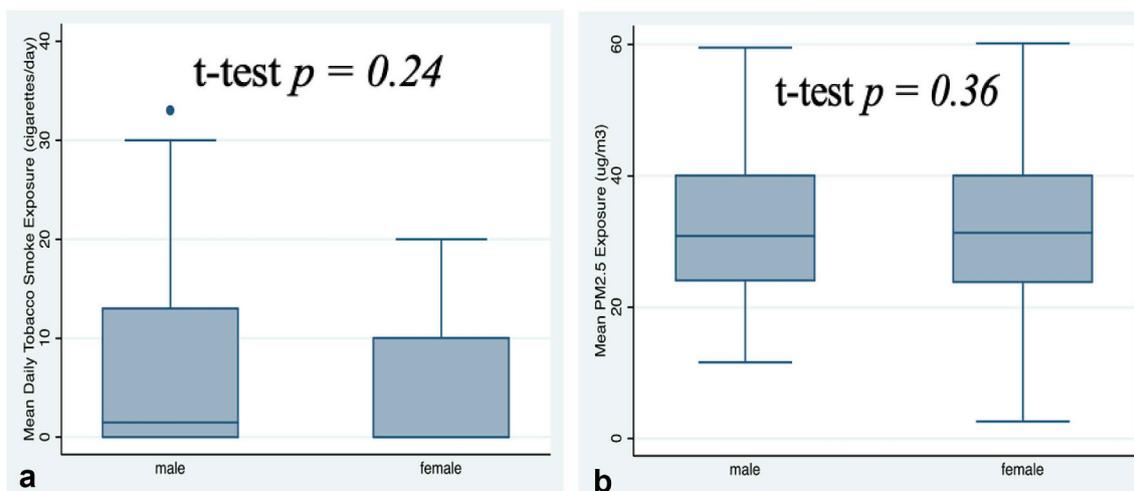
	BMI-Z	PM2.5	TSE	Calories	PA	SES
BMI-Z	1.00					
PM2.5	-0.07	1.00				
TSE*	-0.04	0.07	1.00			
Calories	0.07	-0.08	-0.10	1.00		
PA**	0.15	-0.05	-0.18	-0.10	1.00	
SES	0.15	0.12	-0.12	0.16	-0.06	1.00

\* TSE = Tobacco Smoke Exposure.

\*\*PA = Physical Activity.

physical activity behaviors. This finding has practical implications for the design of future studies of preschool-aged children in China, as young children cannot reliably report their own exposures and behaviors. Personal monitoring allows objective measurements of individual-specific exposures to ambient and indoor air pollution. No study to-date has used personal monitoring to measure individual-level PM2.5 exposures in young children in China. The new PANDA personal air quality monitor has an advantage over typical air pump and filter methods for PM2.5 exposure assessment which are too heavy and bulky to be used to monitor young children. The instruments (PM2.5 and tobacco exposure, and behavior assessments) used in our feasibility study may be useful for larger epidemiological studies that aim to explore associations between environmental pollution and risk of obesity. Additionally, the same instruments may support future research to develop, implement, evaluate and disseminate community-based family interventions that will address child obesity and reduce exposure to air pollution. Despite our small sample size, there were statistically significant gender differences in BMI z-score, suggesting the importance of closely examining sex-specific differences in future research. We did not see a significant association between PM2.5 exposure and TSE with BMI z-score. However, our exposure assessments were able to document the high levels of PM2.5 exposures as well as second-hand tobacco smoke exposures that may be occurring in young preschool-aged children in this region of China.

The feasibility of our study methods has important relevance for future obesity research in China that has cities with high levels of air pollution. In animal studies, after controlling for diet and physical activity, PM2.5 increased insulin resistance, adipogenesis and adipocyte hypertrophy via potentiating inflammatory responses (Zou, 2010; Xu et al., 2010). In a study by Xu et al., young mice exposed to PM2.5 had a higher predilection for visceral abdominal fat accumulation, versus subcutaneous fat (Xu et al., 2010). In assessing for obesity risk, it is important to consider fat distribution because abdominal or truncal adiposity is known to be associated with higher risks of developing



**Fig. 3.** a and b. PM2.5, tobacco smoke exposure and gender.

chronic non-communicable diseases, including type 2 diabetes, dyslipidemia, hypertension and some forms of cancer (Heindel, 2003). Research is ongoing to better understand these interactions in children, but mice models show that exposure to PM2.5 at an early age increases risk of obesity (Xu et al., 2010).

In our study, the PANDA monitors captured objective measurements of individual PM2.5 exposures that included TSE. The additional data from the TSE questionnaire helped to quantify exposures among our subjects. The high second-hand TSE we observed among young children in our study is alarming, but not surprising given the high prevalence of smoking within China. We expected an association between PM2.5 and TSE exposures from our data, as it is known that tobacco smoke contains PM2.5. However, we did not find an association between the self-reported current TSE and PM2.5 exposures as measured by PANDA monitors. This may indicate that the two instruments are capturing different aspects of TSE. For future studies, perhaps both instruments are needed to measure TSE – a questionnaire to capture details of TSE (identifying the person(s) who are involved in child's care who smokes, and also identifying amount and frequency), and also objective measurements from air quality monitors like the PANDA.

TSE exposures in childhood have been associated with increase adiposity, BMI and obesity (Bamia et al., 2004; Carney and Goldberg, 1984; Yang et al., 2013; Raum et al., 2011; Ino et al., 2011; Kwok et al., 2010; Toschke et al., 2003; Verhulst et al., 2009; Lisboa et al., 2012; Pei et al., 2013; Yang et al., 2013; Chen et al., 2012). Pathophysiologically, research suggest that tobacco smoke include many EDCs that may have an antiestrogenic effect that produces an imbalance in androgens and estrogen, and influences distribution, uptake and storage of body fat mass (Xie et al., 2010). There is also accumulating evidence that show children exposed to environmental tobacco have higher serum leptin, C-reactive protein, fibrinogen, and interleukin-6 levels; all implicated in the pathogenesis of obesity (Nagel et al., 2009; Santos-Silva et al., 2013; Lisboa et al., 2012; Ino et al., 2011). Behavioural differences between smoking and non-smoking mothers and family environments also plays a role in influencing breastfeeding practices, child's physical activity and diet, that could alter risk for obesity (Toschke et al., 2003; Lisboa et al., 2012; Santos-Silva et al., 2013). Nonetheless, links between tobacco smoke and child obesity involve complex biomolecular pathways.

Our study also found significantly high second-hand TSE during pregnancy. This is important because TSE in-utero can alter metabolic programming through changes in epigenetics and levels of cortisol (Lisbao, 2012). Infants who were exposed to tobacco smoke in-utero may be born with already a high risk of obesity. In animal models, adult offspring from mothers who were exposed to nicotine during gestation and/or lactation had higher risk of developing obesity and other metabolic and endocrine disorders (Bruin et al., 2007; Somm et al., 2008; Somm et al., 2009; Santos-Silva et al., 2013; Raum et al., 2011). One study also found that adolescents who were prenatally exposed to maternal smoking had higher intra-abdominal fat (La Merrill and Birnbaum, 2011). Other studies have further suggested that tobacco smoke exposure in utero may have gender specific differences on body weight (Somm et al., 2008; Ng et al., 2009), which suggest a predilection for obesity.

While participants in our study generally complied with our study procedures and provided positive feedback on study instruments, understandably a few participants thought that the PANDA monitors were inconvenient to carry and to charge every night. Also due to technical and mechanical issues, air pollution exposure data from thirteen participants (25% of total participants) were not recorded on some of the PANDA monitors. The sample in the present study was small (n = 52), and consequently it was not possible to find an association between air pollution exposure and BMI. All questionnaires were parental-reported and self-administered, and answers maybe over- or under-estimated. Despite these limitations, this study is an important first step in investigating air pollution exposure and child obesity risk in China.

Our assessment of PM2.5 and second-hand tobacco smoke exposures in young children only scratches the surface of the many environmental

exposures that need feasible children's assessment methods. Because interventions to reduce child obesity have only been modestly successful, more comprehensive understanding of environmental toxins and EDCs such as air pollution and tobacco smoke, is important in combating the obesity epidemic.

## 5. Conclusion

China faces large public health costs caused by environmental pollution. The World Health Organization has recently singled out air pollution to be the world's largest health risk, and China has one of the worst air pollution in the world. This study presented novel and feasible methods to measure air pollution (PM2.5 and tobacco smoke) exposure and obesity risk in young children in China that will allow future studies to conduct more detailed analysis on environmental exposures and child health. Future large epidemiologic studies are needed to fully explore the relationship between air pollution exposure and obesity risk. With continued economic development and urbanization, if environmental pollution is related to obesity and metabolic disease, and these are not improved, the economic productivity, costs, and health burdens on Chinese society will be staggering.

## Funding

This work was supported by Dean's Research Fellowship from University of California, San Francisco (UCSF), School of Medicine. Support was also provided by the UCSF Clinical and Translational Research Fellowship Program (CTRFP) and UCSF Clinical and Translational Science Institute (CTSI).

## Declaration of competing interest

No conflicts of interests are declared.

## Acknowledgements

We thank Meiling Gao for assistance with co-location and calibration of the PANDAs with the TEOMs, and students and staff at the Kunming Medical University who assisted this study.

## References

- Baillie-Hamilton, P.F., 2002. Chemical toxins: a hypothesis to explain the global obesity epidemic. *J. Altern. Complement. Med.* 8 (2), 185–192. <https://doi.org/10.1089/10755302317371479>.
- Bamia, C., Trichopoulos, A., Lenas, D., Trichopoulos, D., 2004. Tobacco smoking in relation to body fat mass and distribution in a general population sample. *Int. J. Obes.* 28 (8), 1091–1096. <https://doi.org/10.1038/sj.ijo.0802697>.
- Bergman, Å., Heindel, J.J., Jobling, S., Kidd, K., Zoeller, T.R., World Health Organization, 2013. *State of the Science of Endocrine Disrupting Chemicals 2012*. World Health Organization.
- Bolton, J.L., Auten, R.L., Bilbo, S.D., 2014. Prenatal air pollution exposure induces sexually dimorphic fetal programming of metabolic and neuroinflammatory outcomes in adult offspring. *Brain Behav. Immun.* 37, 30–44. <https://doi.org/10.1016/j.bbi.2013.10.029>.
- Brook, R.D., Franklin, B., Cascio, W., Hong, Y., Howard, G., Lipsett, M., ... Expert Panel on Population and Prevention Science of the American Heart Association, 2004. Air pollution and cardiovascular disease: a statement for healthcare professionals from the expert panel on population and prevention science of the american heart association. *Circulation* 109 (21), 2655–2671. <https://doi.org/10.1161/01.CIR.0000128587.30041.C8>.
- Bruin, J.E., Kellenberger, L.D., Gerstein, H.C., Morrison, K.M., Holloway, A.C., 2007. Fetal and neonatal nicotine exposure and postnatal glucose homeostasis: identifying critical windows of exposure. *J. Endocrinol.* 194 (1), 171–178. <https://doi.org/10.1677/JOE-07-0050>.
- Carney, R.M., Goldberg, A.P., 1984. Weight gain after cessation of cigarette smoking: a possible role for adipose-tissue lipoprotein lipase. *N. Engl. J. Med.* 310 (10), 614–616. <https://doi.org/10.1056/NEJM198403083101002>.
- Chan, C.K., Yao, X., 2008. Air pollution in mega cities in China. *Atmos. Environ.* 42 (1), 1–42.
- Chen, C., 2008. Overview of obesity in mainland China. *Obes. Rev.* 9 (s1), 14–21.
- Chen, Y., Chen, P., Hsieh, W., Portnov, B.A., Chen, Y., Lee, Y.L., 2012. Environmental factors associated with overweight and obesity in Taiwanese children. *Paediatr.*

- Perinat. Epidemiol. 26 (6), 561–571.
- Diamanti-Kandarakis, E., Bourguignon, J., Giudice, L.C., Hauser, R., Prins, G.S., Soto, A.M., ... Gore, A.C., 2009. Endocrine-disrupting chemicals: an endocrine society scientific statement. *Endocr. Rev.* 30 (4), 293–342. <https://doi.org/10.1111/pe.12001>.
- Eloheid, M.A., Padilla, M.A., Brock, D.W., Ruden, D.M., Allison, D.B., 2010. Endocrine disruptors and obesity: an examination of selected persistent organic pollutants in the NHANES 1999–2002 data. *Int. J. Environ. Res. Public Health* 7 (7), 2988–3005. <https://doi.org/10.3390/ijerph7072988>.
- Fleischer, N.L., Meriardi, M., van Donkelaar, A., Vadillo-Ortega, F., Martin, R.V., Betran, A.P., ... O'Neill, M.S., 2014. Outdoor air pollution, preterm birth, and low birth weight: analysis of the world health organization global survey on maternal and perinatal health. *Environ. Health Perspect.* 122 (4), 397–403. <https://doi.org/10.1289/ehp.1306837>.
- Grün, F., Blumberg, B., 2009. Endocrine disrupters as obesogens. *Mol. Cell. Endocrinol.* 304 (1), 19–29. <https://doi.org/10.1016/j.mce.2009.02.018>.
- Hatch, E.E., Nelson, J.W., Stahlhut, R.W., Webster, T.F., 2010. Association of endocrine disruptors and obesity: perspectives from epidemiological studies. *Int. J. Androl.* 33 (2), 324–332. <https://doi.org/10.1111/j.1365-2605.2009.01035.x>.
- Heindel, J.J., 2003. Endocrine disruptors and the obesity epidemic. *Toxicol. Sci. : Off. J. Soc. Toxicol.* 76 (2), 247–249.
- Holstius, D., Pillarisetti, A., Smith, K., Seto, E., 2014. Field calibrations of a low-cost aerosol sensor at a regulatory monitoring site in California. *Atmos. Meas. Tech. Discuss* 7 (1), 605–632.
- Holtcamp, W., 2012. Obesogens: an environmental link to obesity. *Environ. Health Perspect.* 120 (2), a62–a68. <https://doi.org/10.1289/ehp.120-a62>.
- Hui, L.L., Nelson, E.A.S., 2006. Meal glycaemic load of normal-weight and overweight Hong Kong children. *Eur. J. Clin. Nutr.* 60 (2), 220–227. <https://doi.org/10.1038/sj.ejcn.1602305>.
- Ino, T., Shibuya, T., Saito, K., Ohtani, T., 2011. Effects of maternal smoking during pregnancy on body composition in offspring. *Pediatr. Int.* 53 (6), 851–857. <https://doi.org/10.1111/j.1442-200X.2011.03383.x>.
- Janesick, A., Blumberg, B., 2011. Endocrine disrupting chemicals and the developmental programming of adipogenesis and obesity. *Birth Defects Res. Part C Embryo Today - Rev.* 93 (1), 34–50. <https://doi.org/10.1002/bdrc.20197>.
- Ji, C.Y., Cheng, T.O., 2009. Epidemic increase in overweight and obesity in Chinese children from 1985 to 2005. *Int. J. Cardiol.* 132 (1), 1–10. <https://doi.org/10.1016/j.ijcard.2008.07.003>.
- Jiang, J., Rosenqvist, U., Wang, H., Greiner, T., Ma, Y., Toschke, A.M., 2006. Risk factors for overweight in 2- to 6-year-old children in Beijing, China. *Int. J. Pediatr. Obes.* 1 (2), 103–108.
- Kwok, M.K., Schooling, C.M., Lam, T.H., Leung, G.M., 2010. Paternal smoking and childhood overweight: evidence from the Hong Kong “Children of 1997”. *Pediatrics* 126 (1), e46–e56. <https://doi.org/10.1542/peds.2009-2642>.
- La Merrill, M., Birnbaum, L.S., 2011. Childhood obesity and environmental chemicals. *Mt. Sinai J. Med.* 78 (1), 22–48.
- Lisboa, P.C., Oliveira, E., Moura, E.G., 2012. Obesity and endocrine dysfunction programmed by maternal smoking in pregnancy and lactation. *Front. Physiol.* 3, 437.
- McClafferty, H., 2008. Interactions between environmental health and pediatric obesity. *Explore J. Sci. Heal.* 4 (5), 328–332. <https://doi.org/10.1016/j.explore.2008.07.004>.
- Nagel, G., Arnold, F.J., Wilhelm, M., Link, B., Zoellner, I., Koenig, W., 2009. Environmental tobacco smoke and cardiometabolic risk in young children: results from a survey in south-west Germany. *Eur. Heart J.* 30 (15), 1885–1893. <https://doi.org/10.1093/eurheartj/ehp180>.
- Ng, S.P., Conklin, D.J., Bhatnagar, A., Bolanowski, D.D., Lyon, J., Zelikoff, J.T., 2009. Prenatal exposure to cigarette smoke induces diet- and sex-dependent dyslipidemia and weight gain in adult murine offspring. *Environ. Health Perspect.* 117 (7), 1042–1048. <https://doi.org/10.1289/ehp.0800193>.
- Ogden, C.L., Carroll, M.D., Curtin, L.R., Lamb, M.M., Flegal, K.M., 2010. Prevalence of high body mass index in US children and adolescents, 2007–2008. *Jama* 303 (3), 242–249. <https://doi.org/10.1001/jama.2009.2012>.
- Oken, E., Gillman, M.W., 2003. Fetal origins of obesity. *Obes. Res.* 11 (4), 496–506.
- Pei, Z., Flexeder, C., Fuertes, E., Thiering, E., Koletzko, B., Cramer, C., ... Heinrich, J., 2013. Early life risk factors of being overweight at 10 years of age: results of the German birth cohorts GINIplus and LISAplus. *Eur. J. Clin. Nutr.* 67 (8), 855–862. <https://doi.org/10.1038/ejcn.2013.80>.
- Raum, E., Küpper-Nybelen, J., Lamerz, A., Hebebrand, J., Herpertz-Dahlmann, B., Brenner, H., 2011. Tobacco smoke exposure before, during, and after pregnancy and risk of overweight at age 6. *Obesity* 19 (12), 2411–2417. <https://doi.org/10.1038/oby.2011.129>.
- Santos-Silva, A.P., Oliveira, E., Pinheiro, C.R., Santana, A.C., Nascimento-Saba, C.C., Abreu-Villaca, Y., ... Lisboa, P.C., 2013. Endocrine effects of tobacco smoke exposure during lactation in weaned and adult male offspring. *J. Endocrinol.* 218 (1), 13–24. <https://doi.org/10.1530/JOE-13-0003>.
- Somm, E., Schwitzgebel, V.M., Vauthay, D.M., Aubert, M.L., Hüppi, P.S., 2009. Prenatal nicotine exposure and the programming of metabolic and cardiovascular disorders. *Mol. Cell. Endocrinol.* 304 (1), 69–77. <https://doi.org/10.1016/j.mce.2009.02.026>.
- Somm, E., Schwitzgebel, V.M., Vauthay, D.M., Camm, E.J., Chen, C.Y., Giacobino, J., ... Hüppi, P.S., 2008. Prenatal nicotine exposure alters early pancreatic islet and adipose tissue development with consequences on the control of body weight and glucose metabolism later in life. *Endocrinology* 149 (12), 6289–6299. <https://doi.org/10.1210/en.2008-0361>.
- Sun, C., Xia, W., Zhao, Y., Li, N., Zhao, D., Wu, L., 2013. Nutritional status survey of children with autism and typically developing children aged 4–6 years in Heilongjiang Province, China. *J. Nutr. Sci.* 2, e16. <https://doi.org/10.1017/jns.2013.9>.
- Tappy, L., 2006. Adiposity in children born small for gestational age. *Int. J. Obes.* 30, S36–S40.
- Toschke, A.M., Montgomery, S.M., Pfeiffer, U., von Kries, R., 2003. Early intrauterine exposure to tobacco-inhaled products and obesity. *Am. J. Epidemiol.* 158 (11), 1068–1074. <https://doi.org/10.1093/aje/kwg258>.
- Trasande, L., Cronk, C., Durkin, M., Weiss, M., Schoeller, D.A., Gall, E.A., ... Gillman, M.W., 2009. Environment and obesity in the national children's study. *Environ. Health Perspect.* 117 (2), 159–166. <https://doi.org/10.1289/ehp.11839>; [10.1289/ehp.11839](https://doi.org/10.1289/ehp.11839).
- Verhulst, S.L., Nelen, V., Hond, E.D., Koppen, G., Beunckens, C., Vael, C., ... Desager, K., 2009. Intrauterine exposure to environmental pollutants and body mass index during the first 3 years of life. *Environ. Health Perspect.* 117 (1), 122–126. <https://doi.org/10.1289/ehp.0800003>.
- Vrijheid, M., Casas, M., Bergstrom, A., Carmichael, A., Cordier, S., Eggesbo, M., ... Nieuwenhuijsen, M., 2012. European birth cohorts for environmental health research. *Environ. Health Perspect.* 120 (1), 29–37. <https://doi.org/10.1289/ehp.1103823>; [10.1289/ehp.1103823](https://doi.org/10.1289/ehp.1103823).
- Withrow, D., Alter, D., 2011. The economic burden of obesity worldwide: a systematic review of the direct costs of obesity. *Obes. Rev.* 12 (2), 131–141.
- Xie, B., Palmer, P.H., Pang, Z., Sun, P., Duan, H., Johnson, C.A., 2010. Environmental tobacco use and indicators of metabolic syndrome in Chinese adults. *Nicotine Tob. Res. : Off. J. Soc. Res. Nicotine Tob.* 12 (3), 198–206. <https://doi.org/10.1093/ntr/ntp194>.
- Xu, X., Yavar, Z., Verdin, M., Ying, Z., Mihai, G., Kampfrath, T., ... Sun, Q., 2010. Effect of early particulate air pollution exposure on obesity in mice: role of p47phox. *Arterioscler. Thromb. Vasc. Biol.* 30 (12), 2518–2527. <https://doi.org/10.1161/ATVBAHA.110.215350>.
- Yang, S., Decker, A., Kramer, M.S., 2013. Exposure to parental smoking and child growth and development: a cohort study. *BMC Pediatr.* 13 (1), 104. <https://doi.org/10.1186/1471-2431-13-104>.
- Zou, M.H., 2010. Is NAD(P)H oxidase a missing link for air pollution-enhanced obesity? *Arterioscler. Thromb. Vasc. Biol.* 30 (12), 2323–2324. <https://doi.org/10.1161/ATVBAHA.110.216648>.