Obesity Facts

Research Article

Obes Facts 2022;15:609–620
DOI: 10.1159/000525544

Received: July 2, 2021
Accepted: May 13, 2022
Published online: June 23, 2022

Effect of Exposure to Paternal Smoking on Overweight and Obesity in Children: Findings from the Children Lifeway Cohort in Shenzhen, Southern China

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Keywords
Smoking · Obesity · Overweight · Paternal smoking · Child health

Abstract
Introduction: Paternal smoking associated with childhood overweight and obesity has been a concern, but studies have not investigated smoking exposure and smoking details. We investigated the association of exposures from paternal smoking as well as smoking details on offspring overweight/obesity. ***Methods:*** A total of 4,513 children (aged 7–8 years) in Shenzhen were enrolled. Four different exposures from paternal smoking as well as smoking quantity, duration of smoking, and age of starting smoking details were the exposure variables and demographic characteristics, and circumstances of birth, dietary intake, lifestyle, and nonpaternal-smoking exposure were covariates in the logistic regression analysis to determine the effect of paternal smoking on childhood overweight/obesity, estimating odds ratios (ORs), and 95% confidence intervals (CIs). ***Results:*** Paternal smoking was positively associated with childhood overweight/obesity ($p < 0.05$). Moreover, only preconception exposure, and both pre- and postconception exposure were significantly associated with childhood overweight/obesity (OR 1.54 [95% CI: 1.14–2.08] and OR 1.73 [95% CI: 1.14–2.61], respectively), restricted to boys but not girls. Furthermore, for children with only preconception paternal-smoking exposure, the dose-response relation was positive between smoking quantity, duration of smoking, age at starting, and overweight/obesity for boy offspring (p trend <0.001). We did not find any significant association between only postnatal exposure to paternal smoking and childhood overweight/obesity ($p > 0.05$). ***Conclusions:*** Our findings suggest that paternal smoking is associated with boys' overweight/obesity, and this association may be due to the paternal-smoking exposure before conception rather than the postnatal exposure to paternal smoking. Reducing paternal-smoking exposure before conception might help reduce overweight/obesity in boys.

Introduction

Overweight and obesity are among of the most serious global public health challenges of the 21st century and have reached epidemic proportions in many Asian countries including China [1–3]. These countries also face a grave burden of obesity-related disorders such as diabe-

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tes, hypertension, and cardiovascular diseases, which develop at a younger age than in Western populations [1, 4]. These disorders are also manifested in childhood, and childhood obesity has become a major public concern in these countries [5–7]. Overweight and obesity among children in developing countries are more common in urban than rural areas and is positively correlated with gross national product [8–10] because of the rapid socioeconomic development and urbanization in recent years [11–13]. China has experienced a rapid increase in obesity rates in the past three decades, with more than one-fifth of school-age children being overweight or obese [14]. Shenzhen, a city in southern China, has experienced very rapid economic development since the middle of the 20th century and is now a national economic center, representing one of the most developed regions in China [15]. In Shenzhen, the prevalence of overweight and obesity among school-aged children (mean age 10.3 years) was estimated at 20.2% during 2016–2017 [16].

Previous studies have identified some environmental and lifestyle factors that may be associated with childhood obesity. For example, dietary factors [17], a long time spent in front of a TV [18], little exercise [19], picky eating [20], and eating snacks [21, 22] have been reported as risk factors for increased prevalence of childhood overweight and obesity. However, other evidence suggested that these traditional causes of obesity cannot completely explain the present obesity epidemic [23]. Some epidemiological studies showed that environmental factors may play important roles in childhood obesity, including tobacco exposure, which has become a concern [24–26]. About 1 billion people worldwide smoked daily in 2015, 25% of men and 5.4% of women [27]. China is the largest producer and consumer of cigarettes in the world, with 37.5% of men and 2.2% of women smoking daily [27]. Given that the higher current and future predicted smokers are men [28], we should understand the impact of paternal smoking on the health of offspring.

The association of maternal smoking during pregnancy with offspring overweight in childhood has achieved consensus [29, 30]. Paternal smoking is a concern, but the association is uncertain [31–35]. The controversy is whether the impact of paternal-smoking exposure is mainly from preconception or from pre- or postnatal periods. A few studies had tried to explore this question. In the German Ulm Birth Cohort Study, smoking of both parents at pre- and postnatal periods was positively associated with offspring body mass index (BMI) in 8-year-old children [24]. In a prospective study of a Hong Kong Chinese birth cohort investigating pre-natal or postnatal second-hand smoke (SHS) exposure for children of nonsmoking mothers, paternal smoking was associated with increased offspring BMI in children aged 7 and 11 years old [36]. However, intrauterine tobacco smoke exposure and postnatal SHS exposure could not be distinguished in the study. Moreover, the effect of preconception paternal-smoking exposure was not studied. A few studies have investigated paternal smoking in the period before conception. For example, in the Avon Longitudinal Study of Parents and Children (ALSPAC) cohort, paternal smoking before age 11 was suggested to contribute to obesity in adolescent male offspring, which first highlighted the importance of the developmental timing of the paternal-smoking exposure as well as sex differences in offspring outcomes [37]. However, in the Nord Trøndelag Health Study, the authors did not support a son-specific association of the magnitude reported in the ALSPAC study but could not rule out a weaker association, perhaps common to sons and daughters, which would be consistent with the ALSPAC study [38]. Another study-based screening program in Taiwan determined the effect of longer duration and earlier age of paternal-smoking onset before offspring birth on increased risk of metabolic syndrome in offspring. Unlike the ALSPAC study, the Taiwan study was unable to determine a transgenerational effect of prepubertal paternal smoking on BMI in male offspring [39]. Thus, different sample sources, sample size, and confounders may explain the inconsistent results. Nevertheless, the mechanism of paternal smoking may involve the biological effect of intrauterine tobacco smoke exposure and epigenetic modifications in the germ line before conception, but more evidence is needed.

Hence, we used the large population-representative Children Lifeway Cohort of Shenzhen to examine the association of overweight and obesity among children of nonsmoking mothers during different periods of paternal-smoking exposure preconception and postconception separately or overlapping. Moreover, we investigated details about smoking quantity, duration of smoking, and age of starting smoking to detect the dose-response association.

Materials and Methods

Data Collection

The Children Lifeway Cohort was developed to investigate metabolic disease in a prevention and treatment program of occupational groups and adolescents in the Baoan district of Shenzhen, in order to understand the lifestyle and related metabolic disease of children. This cohort recruited first-grade students from

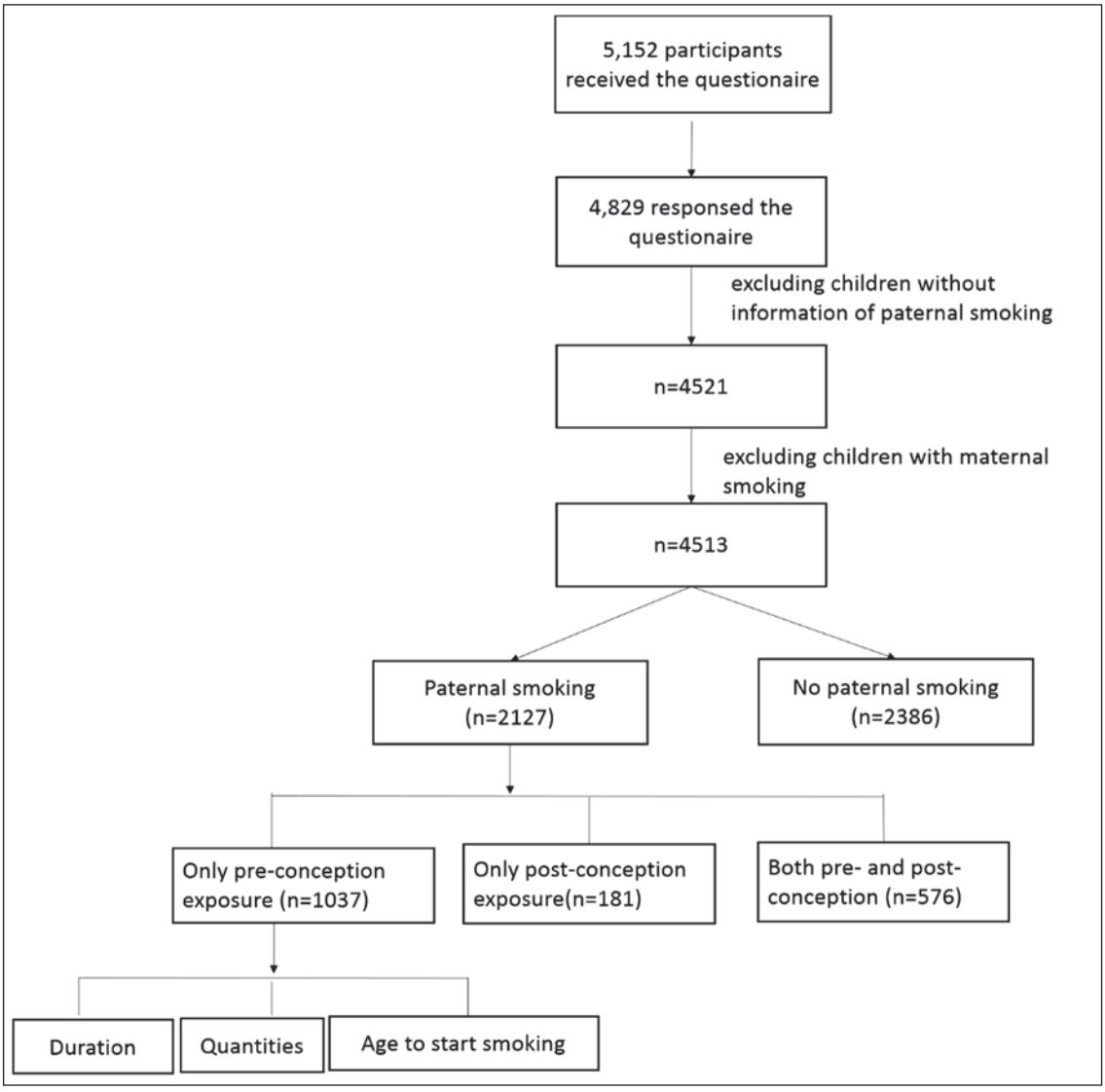

Fig. 1. The flow chart of the study.

19 of the 134 local primary schools by using cluster sampling in September 2018. In the end, 18 schools were willing to participate. The study administered a questionnaire and physical examination to children. A total of 5,152 children received a paper questionnaire, and the response rate was 93.5% (4,829 children). During the data extraction and analyses, children without information on paternal smoking (n = 308) and whose mother smoked (n = 8) were excluded. Finally, 4,513 students were included in the analyses (Fig. 1). A paper questionnaire was administered to parents to collect information related to children or parents such as demographic characteristics (age, sex, percentage of food expenditure), circumstances of birth (cesarean section, birth weight status, breastfeeding), dietary intake (late-night dinner, vegetables, fruit, snacks, fried/baked food), and lifestyle (physical activity, watching TV, picky eater). The questionnaire showed good reliability and validity with high consistency among measurement items after evaluation (Cronbach's α = 0.776). Data were collected on parents (weight, height, education, and smoking details). Anthropometric measurements of children included weight and height.

Outcome Assessment

Overweight/obesity was defined by using age- and sex-specific BMI cutoff points according to the growth standards of China "Screening for overweight and obesity among school-age children and adolescents (WS/T 586-2018)" [40].

Exposure

Trained interviewers asked parents to recall the smoking habits in their family. The questions were phrased as follows: Did the father or the mother smoke more than 100 cigarettes in his or her

life? If the answer was yes, the father or mother was classified as paternal or maternal smoking [41], then the quantity (1–10, 11–20, >20 cigarettes/day), duration (1–10, 11–20, >20 years), and age of the father/mother when they started smoking (≤20, >20 years old) were asked. If the answer to the question was no, the father or mother were classified as nonpaternal or nonmaternal smoking.

The children were classified according to their exposure to paternal smoking at different periods in the following four categories: no paternal smoking, only preconception exposure (the father smoked only before pregnancy), only postconception exposure (the father smoked only after pregnancy), and both pre- and postconception exposure.

The study investigated whether, besides the mother or father smoking, anyone who lived in the household smoked and the number of smoking household members. It investigated whether, besides the father smoking, anyone else smoked in front of the mother at home or workplace during her pregnancy and whether the mother was exposed to SHS during the pregnancy. The exposure sources from household smoking other than father were analyzed as a covariable.

Other Covariables

The following variables are considered as covariables in the analysis. Parents were asked to report the child's frequency of food intake, namely fried/baked food, late-night dinners, picky eater, and vegetables, fruits, and snacks consumption during the week before the survey. Additionally, the hours of children participated in physical exercise and watched TV per day was investigated.

Data were collected on children's age, sex, birth weight (<2,500, 2,500–4,000, >4000 g), breastfeeding (bottle-feeding, breastfeeding, mixed), cesarean section (yes/no), percentage of income spent on food expenditure (>50%, 30–50%, <30%), and parents' educational level (take the one with higher education).

Statistical Analyses

Multivariate logistic regression was adjusted for dietary and other covariates to evaluate the association between overweight/obesity and four different paternal-smoking exposure periods separately or overlapping, estimating OR and 95% CI. Two multivariable regression models were included in study. The dose-response association between smoking quantity, duration of smoking, age at starting smoking for the father, and risk of childhood overweight and obesity was tested with *P* for linear trend. Analyses were also stratified by child sex to assess any potential sex-specific effect of paternal smoking on childhood overweight/obesity. The level of significance was set at $p < 0.05$. All statistical analyses involved using SPSS 24.0 (IBM Corp., Armonk, NY, USA).

Results

Participant Characteristics

The mean (SD) age of the 4,513 children was 7.10 (0.34) years, and the proportion of boys was slightly more than girls (56.9% vs. 43.1%). A total of 2,127 (47.1%) fathers reported smoking. The proportion of children with overweight/obesity was 16.1% ($n = 728$). Characteristics of children are presented by healthy weight and overweight/obesity in Table 1. Boys were more frequently overweight/obese than girls. Frequency of overweight/obesity was higher for children with than without overweight/obese parents. The proportion of overweight/obesity was higher for children whose parents had higher than lower educational level. Overweight/obesity was more frequent for children with than without cesarean section birth and was more frequent for children with higher than lower birth weight. Frequency of overweight/obesity was lower for children with than without picky eating habits but was higher for children who habitually ate snacks than those who did not. Additionally, overweight/obesity was higher for children with than without fathers who smoked. As compared with children without paternal smoking, those with both pre- and postconception exposure were more frequently overweight/obese ($p = 0.012$) (Table 2).

Association of Different Paternal-Smoking Exposure Periods and Overweight/Obesity in Children

Association of Only Preconception Exposure and Overweight/Obesity

Table 3 shows two multivariable regression models for the effect of different paternal-smoking exposure periods on risk of overweight and obesity in children. In Model I (adjustment for only basic and birth characteristics), as compared with children without paternal smoking, for those with only preconception exposure, the risk of childhood overweight and obesity was increased (OR 1.41 [95% CI: 1.17–1.85]). In Model II, after further adjustment for lifestyle and dietary factors, this effect remained statistically significant (OR 1.54 [95% CI: 1.14–2.08]). When stratified by sex, the effect of only preconception exposure on childhood overweight and obesity was statistically significant for only boys ($p < 0.05$).

Association of Both Pre- and Postconception and Probability of Overweight/Obesity

Probability of overweight/obesity was increased with both pre- and postconception exposure from paternal smoking (Model I: OR 1.55 [95% CI: 1.02–2.61]; Model II: OR 1.73 [95% CI: 1.11–2.75]). On subgroup analysis by sex, the effect of both pre- and postconception exposure on overweight/obesity existed for only boys ($p < 0.05$). We found no association between only postconception exposure and childhood overweight and obesity ($p > 0.05$).

Table 1. Characteristics of all children and those with healthy weight and overweight/obesity (n = 4,513)

	All children, n (%)	Healthy weight (N = 3,785)	Overweight/ obesity (N = 728)	p value
Age	7.10±0.34	7.10±0.34	7.10±0.32	0.975
Sex				
Girls	1,946 (43.1)	1,715 (88.1)	231 (11.9)	**<0.001**
Boys	2,567 (56.9)	2,070 (80.6)	497 (19.4)	
Percentage of food expenditure according to income				
>50%	995 (22.0)	775 (83.2)	156 (16.8)	
30–50%	2,451 (56.3)	1,996 (82.8)	415 (17.2)	0.950
<30%	977 (21.6)	757 (82.8)	157 (17.2)	
Father overweight/obese				
No	2,428 (58.7)	1,445 (87.3)	211 (12.7)	<**0.001**
Yes	1,706 (41.3)	898 (75.4)	293 (24.6)	
Mother overweight/obese				
No	2,491 (86.3)	2,047 (83.8)	396 (16.2)	<**0.001**
Yes	395 (13.7)	278 (72.0)	108 (28.0)	
Parents' educational level				
Junior middle and below	838 (18.6)	660 (82.5)	140 (17.5)	
Junior high/junior college	2,588 (57.3)	2,048 (84.2)	390 (16.0)	**0.046**
Bachelor degree or above	1,087 (24.1)	820 (80.6)	198 (19.4)	
Breastfeeding				
Bottle-feeding	479 (11.5)	394 (82.4)	84 (17.6)	
Breastfeeding	3,097 (74.5)	2,527 (83.0)	516 (17.0)	0.598
Mixed	579 (13.9)	466 (81.3)	107 (18.7)	
Cesarean section				
No	2,434 (59.8)	2,021 (84.5)	372 (15.5)	<**0.001**
Yes	1,637 (40.2)	1,294 (79.9)	325 (20.1)	
Birth weight, g				
<2,500	197 (4.7)	161 (86.1)	26 (13.9)	
2,500–4,000	3,706 (89.1)	2,911 (83.2)	588 (16.8)	<**0.001**
>4,000	257 (6.2)	176 (71.5)	70 (28.5)	
Physical exercise, hr/day				
<1	1,090 (26.4)	897 (83.9)	172 (16.1)	
1–2	1,726 (41.8)	1,402 (82.3)	302 (17.7)	0.539
>2	1,311 (31.8)	1,072 (82.9)	221 (17.1)	
Watching TV, hr/day				
<1	2,385 (61.6)	1,948 (82.8)	406 (17.2)	
1–2	1,065 (27.5)	875 (83.5)	173 (16.5)	0.838
>2	424 (10.9)	343 (82.5)	73 (17.5)	
Picky eaters				
No	1,757 (42.3)	1,307 (75.5)	423 (24.5)	<**0.001**
Yes	2,397 (57.7)	2,084 (88.2)	280 (11.8)	
Fried/baked food, times/week				
<1	3,499 (86.4)	2,867 (81.3)	582 (18.7)	
1–3	397 (9.8)	317 (81.5)	72 (18.5)	0.629
>3	153 (3.8)	124 (81.0)	29 (19.0)	
Late-night dinner, times/week				
<1	3,051 (73.5)	50 (82.0)	11 (18.0)	
1–3	1,040 (25.0)	858 (83.4)	171 (16.6)	0.852
>3	61 (1.5)	2,482 (82.7)	521 (17.3)	
Fruit, times/week				
<3	1,344 (32.3)	1,100 (83.4)	219 (16.6)	
4–6	1,286 (30.9)	1,057 (83.0)	217 (17.0)	0.623
≥7	1,528 (36.7)	1,234 (82.0)	270 (18.0)	
Vegetable, times/week				
<3	1,125 (27.0)	917 (82.6)	193 (17.4)	
4–6	698 (16.8)	571 (82.9)	118 (17.1)	0.989
≥7	2,340 (56.2)	1,906 (82.8)	397 (17.2)	

Table 1 (continued)

	All children, *n* (%)	Healthy weight (*N* = 3,785)	Overweight/ obesity (*N* = 728)	*p* value
Snacks, times/week				
1–3	2,383 (57.9)	1,453 (61.0)	257 (38.9)	
4–6	1,012 (24.6)	1,698 (82.1)	370 (17.9)	**0.007**
≥7	719 (17.5)	215 (77.3)	63 (22.7)	
Other household members smoke (number)				
0	2,786 (67.5)	2,278 (82.9)	470 (17.1)	
1	1,007 (24.4)	825 (83.1)	168 (16.9)	0.784
2	336 (8.1)	268 (81.5)	61 (18.5)	
Mother exposed to SHS during pregnancy				
No	2,826 (76.7)	2,213 (83.1)	450 (16.9)	0.091
Yes	860 (23.3)	662 (80.5)	160 (19.5)	
Paternal smoking				
No	2,386 (52.9)	2,028 (85.0)	358 (15.0)	**0.032**
Yes	2,127 (47.1)	1,757 (82.6)	370 (17.4)	

Data are *n* (%) or mean (SD). SHS, second-hand smoke. [a] Children with sex–age-specific BMI ≥85 percentile and BMI ≥95 percentile were classified as overweight and obese; all other children were classified as healthy weight.

Table 2. Univariate analysis of effect of different paternal-smoking exposure periods on overweight and obesity in children (*n* = 4,513)

Exposure from paternal smoking	All children, *n* (%)	Healthy weight (*N* = 3,528)	Overweight/obesity (*N* = 728)	*p* value
No paternal smoking (ref)	2,386 (57.6)	2,028 (85.0)	358 (15.0)	–
Only preconception exposure	1,037 (24.5)	863 (83.2)	174 (16.8)	0.188
Only postconception exposure	181 (4.3)	153 (84.5)	28 (15.5)	0.866
Both pre- and postconception exposure	576 (13.6)	465 (80.7)	111 (19.3)	**0.012**

Association of Smoking Quantity, Duration of Smoking, Age of Starting Smoking, and Probability of Overweight/Obesity

We found a dose-dependent association of age of starting smoking and probability of childhood overweight and obesity for children with only preconception exposure, from a 1.27-fold increased probability for starting over age 20 years to a 1.64-fold increased probability for starting under age 20 years, with no paternal-smoking exposure as the reference (p_{trend} = 0.047) (Table 4). Moreover, a longer duration of paternal smoking before conception was associated with increased probability of childhood overweight and obesity. For fathers smoking 1–10 years, 11–20 years, or >20 years, the probability of overweight and obesity in offspring with pre-fatherhood exposure was increased (OR 1.30, OR 1.43, and OR 1.57, respectively) as compared with offspring with no exposure (P_{trend} = 0.020) (Table 4). In addition, we found a dose-response effect of cigarette number smoked by fathers, increasing from 1.55-fold to 1.39-fold–1.85-fold with fathers consuming 1–10, 10–20, and >20 cigarettes per day (p_{trend} = 0.001). Furthermore, after stratification by sex, those relationships were found for only boys (p_{trend} <0.001).

Discussion

Our present study clarified the association between four different periods of exposure to paternal smoking and risk of overweight/obesity among school-aged children in Shenzhen. The results showed a significant paternal-smoking effect on the child's overweight/obesity status, from only preconception exposure and both pre- and postconception exposure. The effects related to child-sex differences restricted to boys but not girls. Moreover, we

found a positive dose-response association between smoking quantity and duration, age of starting smoking, and overweight/obesity among boys with only preconception exposure.

Generally speaking, the causes of overweight and obesity in children are multifactorial [42]. For example, lifestyle is closely related to overweight/obesity [6]. Lindsay-et al. [6] conducted a systematic review of the evidence on the associations between parental feeding practices, child eating behaviors, and risk of overweight and obesity in Southeast Asian children 2–12 years old. The authors found nonresponsive parental feeding practices and unhealthy child eating behaviors associated with risk of child overweight and obesity in several Southeast Asian countries including Thailand, Vietnam, Singapore, the Philippines, and Malaysia [6]. Taveras et al. [43] found high consumption of fried food associated with high BMI among children. A study from China reported the likelihood of overweight/obese as 1.5-fold increased for children who had permission to purchase snacks with pocket money [22]. Our study showed that children with overweight/obesity ate more snacks and were less frequently picky eaters than those with healthy weight. Rapid economic developments in Asia have caused transitions in nutrition and physical activity behavior, which has contributed to a quick spread of the obesity epidemic over the past decades [3, 44]. Besides the effect of dietary and lifestyle factors, overweight and obesity of children were also linked to circumstances of birth in several studies, such as a cesarean section birth and high birth weight. Our study also showed the strong association between weight status of the father or mother and their children, which suggests an underlying genetic predisposition, so parental BMI might confound this association due to its influence on fetal development and thus birth weight. Overweight and obesity have also been found prevalent in populations belonging to different economic classes in several Western studies [45]. For Brazilian students belonging to families from the highest economic class, the risk of overweight was 2-fold increased and the risk of obesity 3-fold increased as compared with classmates belonging to families from the lowest economic class [46]. However, the Hong Kong 1997 cohort [36] as well as our study showed a less clearly socially patterned prevalence of Chinese childhood overweight and obesity.

Our studies focused on the relation between paternal smoking and obesity. In considering the numerous confounding factors mentioned above, we built Model I by adjusting for confounders such as sex, actual age, overweight of father and mother, percentage of food expendi-

Table 3. Multivariate regression analysis of the association of different paternal-smoking exposure periods and overweight and obesity in children and by sex (n = 4,513)

	Total				Boys				Girls			
	Model I[a]		Model II[b]		Model I[a]		Model II[b]		Model I[a]		Model II[b]	
	OR (95% CI)	p value	OR (95% CI)	p value	OR (95% CI)	p value	OR (95% CI)	p value	OR (95% CI)	p value	OR (95% CI)	p value
No paternal smoking (ref)	1.00		1.00		1.00		1.00		1.00		1.00	
Only preconception exposure	1.41 (1.07–1.85)	**0.015**	1.54 (1.14–2.08)	**0.004**	1.78 (1.27–2.49)	**0.001**	2.11 (1.45–3.08)	**<0.001**	0.88 (0.55–1.43)	0.612	0.91 (0.54–1.53)	0.709
Only postconception exposure	0.73 (0.30–1.82)	0.502	0.78 (0.28–2.15)	0.624	0.67 (0.22–2.04)	0.483	0.52 (0.14–1.99)	0.345	0.97 (0.20–4.61)	0.968	1.36 (0.27–6.86)	0.713
Both pre- and postconception exposure	1.55 (1.02–2.14)	**0.041**	1.73 (1.14–2.61)	**0.009**	1.75 (1.11–2.75)	**0.016**	2.22 (1.34–3.67)	**0.002**	1.04 (0.52–2.05)	0.908	1.08 (0.50–2.31)	0.846

OR, odds ratio; 95% CI, 95% confidence interval; SHS, second-hand smoke. [a] Model I: adjusting for sex, actual age, father overweight, mother overweight, percentage of food expenditure, educational level of parents, cesarean section birth, birth weight, breastfeeding, other household smoking, and mother exposed to SHS during pregnancy. [b] Model II: model I plus picky eaters, TV watching time, physical exercise, frequency of eating fried/baked food, late-night dinners, and vegetables, fruit, and snacks consumption.

Obes Facts 2022;15:609–620
DOI: 10.1159/000525544

Table 4. Dose-response relationships between the smoking quantities, duration of smoking, age to start smoking, and childhood overweight/obesity among children with only preconception exposure from paternal smoking (n = 1,037)

		Total				Boy				Girl			
		Model I[a]		Model II[b]		Model I[a]		Model II[b]		Model I[a]		Model II[b]	
		OR (95% CI)	*p* value	OR (95% CI)	*p* value	OR (95% CI)	*p* value	OR (95% CI)	*p* value	OR (95% CI)	*p* value	OR (95% CI)	*p* value
Age for father starting smoking (years old)	0	1.00		1.00		1.00		1.00		1.00		1.00	
	≤20	**1.52 (1.11–2.08)**	**0.009**	**1.64 (1.16–2.31)**	**0.005**	**1.88 (1.27–2.77)**	**0.002**	**2.26 (1.45–3.53)**	**<0.001**	1.03 (0.60–1.77)	0.920	1.07 (0.59–1.93)	0.834
	>20	1.14 (0.76–1.72)	0.524	1.27 (0.82–1.99)	0.282	1.48 (0.91–2.43)	0.118	1.79 (1.03–3.11)	0.039	0.63 (0.29–1.40)	0.258	0.65 (2.76–1.53)	0.324
p for trend			0.120		**0.047**		**0.012**		**0.002**	0.368		0.463	
Duration of paternal smoking (years)	0		1.00		1.00		1.00		1.00	1.00		1.00	
	1–10	1.30 (0.76–2.21)	0.347	1.19 (0.64–2.21)	0.587	1.43 (0.74–2.74)	0.288	1.53 (0.71–3.29)	0.283	1.13 (0.43–2.93)	0.808	0.81 (2.62–2.51)	0.715
	11–20	**1.43 (1.02–2.01)**	**0.039**	**1.69 (1.17–2.44)**	**0.005**	**1.84 (1.22–2.79)**	**0.004**	**2.33 (1.47–3.70)**	**<0.001**	0.81 (0.42–1.54)	0.519	0.93 (0.47–1.81)	0.820
	>20	1.57 (0.79–3.10)	0.199	1.39 (0.66–2.94)	0.390	**2.35 (1.04–5.31)**	**0.041**	1.95 (0.79–4.86)	0.150	0.65 (0.14–3.01)	0.578	0.80 (0.17–3.89)	0.787
p for trend			**0.020**		**0.008**		**0.001**		**<0.001**		0.442		0.726
Cigarettes (numbers)	0		1.00		1.00		1.00		1.00		1.00		1.00
	1–10	1.39 (0.97–2.01)	0.073	1.55 (1.04–2.31)	0.032	1.77 (1.14–2.75)	0.012	2.11 (1.27–3.51)	0.004	0.89 (0.46–1.73)	0.739	1.05 (0.52–2.14)	0.889
	11–20	1.34 (0.92–1.95)	0.123	1.39 (0.92–2.10)	0.123	1.55 (0.97–2.48)	0.067	1.73 (1.01–2.94)	0.045	1.04 (0.55–1.96)	0.901	0.98 (0.48–1.98)	0.952
	>20	1.57 (0.89–2.75)	0.118	**1.85 (1.03–3.34)**	**0.041**	**2.17 (1.11–4.23)**	**0.023**	**3.05 (1.49–6.24)**	**0.002**	0.62 (0.18–2.17)	0.450	0.65 (0.18–2.33)	0.505
p for trend			**0.026**		**0.010**		**0.003**		**<0.001**		0.670		0.671

OR, odds ratio; 95% CI, 95% confidence interval; SHS, second-hand smoke. [a] Model I: adjusting for sex, actual age, father overweight, mother overweight, percentage of food expenditure, educational level of parents, caesarean section birth, birth weight, breastfeeding, other household smoking, and mother exposed to SHS during pregnancy. [b] Model II: model I plus picky eaters, TV watching time, physical exercise, frequency of eating fried/baked food, late-night dinners, and vegetables, fruit, and snacks consumption.

ture, educational level of parents, cesarean section birth, birth weight, breastfeeding, other household smoking, and mother exposed to SHS during pregnancy. Furthermore, Model II included risk factors characterizing the current lifestyle of children such as physical activity, duration of TV viewing and the child's frequency of fried/baked food, late-night dinners, picky eater, and vegetables, fruits, and snacks consumption.

Probability of overweight/obesity was greater for children exposed than not exposed to paternal smoking. This finding was consistent with previous studies in Europe or China. For example, a cross-sectional study in 2005 in Bavaria of 5,899 children (mean age 5.8 years) found increased probability of overweight/obesity with paternal smoking (adjusted OR 1.5/1.9) [47]. The Hong Kong 1997 cohort study also showed greater BMI at age 7 and 11 years in children with than without smoking fathers [36].

The most important finding of this study was that paternal smoking at preconception was significantly associated with childhood overweight/obesity. We divided paternal smoking into four different exposure periods and found a significant relation with only preconception exposure, and both pre- and postconception exposure, which robustly suggested that preconception exposure is a key exposure. Moreover, we found dose-dependent associations of greater probability of childhood overweight/obesity with younger age of father starting smoking, longer duration of smoking, and more cigarettes smoked by fathers. This finding was consistent with several other studies. One study from Taiwan suggested that the effect of paternal smoking on the offspring's risk of metabolic syndrome was significant with exposure starting before but not after the proband offspring's birth (OR 1.27 [95% CI: 1.11–1.45] versus OR 0.9 [95% CI: 0.78–1.14]) [38]. Two other epidemiological studies found that paternal smoking during preadolescence (<11 years old) was associated with increased BMI in offspring [38, 48].

In addition, we found a positive dose-response association between smoking quantity, duration of smoking, and age of starting smoking with boys' overweight/obesity, which suggests a potential sex-specific relevance. Similar results were found in several other studies. For example, Mejia et al. [49] showed that the association between paternal smoking and offspring overweight/obesity was most marked for boys (p = 0.032). In another earlier study [48], using the ALSPAC data, only sons showed intergenerational associations with paternal mid-childhood smoking onset, which indicates a sex-specific transgenerational response system in humans. In this study, the authors hypothesized that the son-specific segregation fits with transmission of information via the sex chromosomes, particularly the Y chromosome. Several experimental and human studies showed a positive association between the Y chromosome and obesity. An animal experiment showed that a second sex chromosome, either Y or X, causes similar increases in body weight, adiposity, and lean mass of mice, relative to mice with a single X chromosome. Under some conditions, the effect of the second sex chromosome to increase body weight is greater if that chromosome is Y rather than X [50]. In a case-control study including 180 males, Y chromosome microdeletions were more common in obese than normal-weight men [51]. These studies, including ours, support the hypothesis that tobacco-related chemical exposure of the father before conception may cause potential damage or modifications on father male-germ cells, which might be expressed over generations [52, 53]. The mechanism of this phenomenon may explain the new field of epigenomic paternal transmission, which suggests that paternal exposure to environmental challenges plays a critical role in the offspring's future health and the transmission of acquired traits through generations [54]. Several studies showed that smoking alters DNA methylation patterns and gene transcription levels in human spermatozoa [55, 56]. Paternal exposure to cigarette smoke leads to increased global methylation of sperm DNA and alterations to the differentially methylated regions of the *DLK1* gene in offspring, which in turn leads to elevated liver fat accumulation and may perturb long-term metabolic function in offspring [57, 58]. In addition, Barbara Hammer et al. [59] found that paternal cigarette smoke exposure at preconception regulated spermatozoal miRNAs and possibly influences the body weight of offspring in early life.

Our results did not confirm the association between childhood overweight/obesity and postconception exposure, which includes intrauterine SHS exposure and postnatal SHS exposure. Previous studies had assessed the association between intrauterine SHS exposure and child weight status, but the results were inconsistent. Dior et al. [60] showed that exposure to paternal smoking during pregnancy was positively and independently associated with BMI at age 17 years. However, Braun et al. [61] reported that the associations between self-reported prenatal SHS exposure and BMI were close to the null value. Oken et al. [62] did not find increased BMI among 3-year-old children born to women with SHS exposure during pregnancy. The inconsistency in results may be due to the differences in the definition of intrauterine SHS expo-

sure, the duration of exposure, or the age at follow-up. Most of the studies focused on the postnatal period, yielding different results on SHS exposure from mothers or fathers. Oliver et al. [63] confirmed no significant association between child postnatal SHS exposure (not just from the father) and weight status up to age 4 years. Oller et al. [64] took advantage of the large sample size in a Danish birth cohort study to stratify children by SHS exposure prenatally only, postnatally only, or both periods, with results indicating that SHS exposure postnatally only was not statistically significant with childhood overweight at age 7 years. In contrast, some studies assessed the effect of postnatal SHS exposure and found a positive association with child BMI. In a longitudinal cohort study, McConnell et al. [65] estimated the effect of SHS exposure on children BMI at age 10 years: SHS exposure was positively associated with BMI growth. The reason for this discrepancy may be that the association between postnatal exposure and children overweight/obesity may only become apparent at later ages. Many studies have reported stronger effects of postnatal SHS exposure as children become older [66].

Strengths and Limitations

The strengths of the study include the large population-based sample for separating the effect of each exposure period and classifying children into the four different categories. Moreover, details about smoking quantity, duration of smoking, and age of starting smoking were collected to detect the dose-response association. In addition, we administered a relatively comprehensive questionnaire, which took into account a wide range of potential confounders, such as dietary factors. Furthermore because of the small number of female or maternal smokers, we were able to isolate the confounder (maternal active smoking) when we discussed the association between paternal smoking and overweight/obesity among children.

Several limitations should be considered. First, because of the observational nature of this study, we could not determine the causal relationship between paternal smoking and children's overweight/obesity. Second, we assessed childhood adiposity with the proxy measure of BMI, which cannot specify whether the obesity is central, peripheral, or in the organ at risk. Third, the classification of smoking exposure status of children may not be precise enough because of parental self-reporting rather than detecting a biological marker of smoking exposure such as cotinine, etc.

Conclusions

In this cross-sectional study based on data from a large sample survey among students in primary school in Shenzhen, we observed a high association between paternal smoking before conception and childhood overweight/obesity, with child sex-related differences restricted to boys. Furthermore, we found positive dose-response associations between the father's smoking quantity, duration of smoking, and age of starting smoking and overweight/obesity for boys. The present study supports that tobacco exposure of the father before conception may play a critical role in the offspring's future health and the transmission of acquired traits through generations. However, a large longitudinal cohort study and more evidence of the biological mechanism are needed to confirm these results. Even so, these findings suggest the need to promote smoking cessation or no smoking among young boys to help reduce the risk of obesity among potential offspring.

Acknowledgments

We are grateful to all the doctors and nurses in the department of laboratory medicine, Baoan Central Hospital of Shenzhen. We also thank all the school doctors and teachers in Baoan District who participated in the survey.

Statement of Ethics

The study was approved by the Ethics Committee of Baoan Central Hospital of Shenzhen (protocol code IRB-PJ-2018-002, 2018-09) and was performed according to the Declaration of Helsinki. Participation was voluntary and written informed consent was obtained from all parents of the participants prior to enrolment.

Conflict of Interest Statement

The authors have no conflict of interests to declare.

Funding Sources

This research received no external funding.

Author Contributors

Each author substantially contributed to conduction of this research and drafting of this paper. Yingbin You, Hui Liu, and Yanxiang Qiao were in charge of conducting the survey and physical

examination for children. Rong Wu, Boya Li, and Rongqing Lin were involved in questionnaire design and participated in the survey. Zan Ding completed information collection and preprocessed data. Ruiguo Liu conducted the literature search, cleaned data, performed statistical analyses, and drafted the manuscript. Hua Zhou, Pi Guo, and Qingying Zhang conceived the study, were involved in questionnaire design, and provided significant guidance for drafting and editing the manuscript. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

Data Availability Statement

The data presented in this study are available on request from the corresponding author. The data are not publicly available due to privacy restrictions.

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