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Relationship between childhood secondhand smoke exposure and the occurrence of hyperlipidaemia and coronary heart disease among Chinese non-smoking women: a cross-sectional study

Kewei Wang ,[1,2,3,4] Yuanqi Wang,[1,2,3,4] Ruxing Zhao,[1,2,3,4] Lei Gong,[1,2,3,4] Lingshu Wang,[1,2,3,4] Qin He,[1,2,3,4] Li Chen,[1,2,3,4] Jun Qin[1,2,3,4]

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For numbered affiliations see end of article.

Correspondence to
Professor Li Chen; chenli3@medmail.com.cn and Dr Jun Qin; qinjun@sdu.edu.cn

ABSTRACT

Objective The objective of this study was to evaluate the influence of secondhand smoke (SHS) exposure during childhood on type 2 diabetes mellitus, hypertension, hyperlipidaemia and coronary heart disease among Chinese non-smoking women.

Methods In this cross-sectional study, the SHS exposure data in childhood were obtained using a questionnaire survey. Self-reported childhood SHS exposure was defined as the presence of at least one parent who smoked during childhood.

Results Of the 6522 eligible participants, 2120 Chinese women who had never smoked were assessed. The prevalence of SHS exposure in the entire population was 28.1% (596). SHS exposure during childhood was not significant for the standard risk factors of type 2 diabetes mellitus (p=0.628) and hypertension (p=0.691). However, SHS was positively associated with hyperlipidaemia (p=0.037) after adjusting for age, obesity, education status, physical activity, alcohol consumption, current SHS exposure status, diabetes mellitus and hypertension. In addition, childhood SHS increased the occurrence of coronary heart disease (p=0.045) among non-smokers after further adjusting for hyperlipidaemia.

Conclusion SHS exposure during childhood is associated with prevalent hyperlipidaemia and coronary heart disease in adulthood among non-smoking Chinese women.

Strengths and limitations of this study

- This is the first study to explore the correlation between childhood secondhand smoke exposure and the occurrence of type 2 diabetes mellitus, hypertension, hyperlipidaemia and coronary heart disease in non-smoking women.
- All self-reported diagnoses required the participants to provide a hospital checklist and proof of disease diagnosis to guarantee diagnostic reliability.
- The cross-sectional survey employed herein limited the ability to make casual inferences.
- Specific information on smoking status, such as intensity, amount and duration, was not available in the study.
- The study was restricted to adult women over 40 years old, without including women of other ages and men.

INTRODUCTION

Secondhand smoke (SHS) refers to the mixture of gases and particles that emit from the burning tip of cigarettes and the smoke exhaled by individuals who are active tobacco smokers.[1] Current findings demonstrate that exposure to SHS has been associated with various health problems, such as type 2 diabetes mellitus,[2] hypertension,[3] non-fatty alcohol liver disease,[4] stroke,[5] cardiovascular diseases,[6] lung cancer and even premature death to non-smokers.[7 8] Nowadays, childhood SHS exposure has attracted an increasing amount of social attention. In all age groups, children have less control over SHS exposure in their own environment and are more susceptible to the hazards of SHS.[9 10] Parental smoking has been widely recognised as a major source of SHS exposure for non-smoking children and is detrimental to their physical even mental health.[10] Current studies indicate that childhood SHS exposure could exert contemporaneous and delayed effects on the respiratory health of those exposed.[11 12] The Cardiovascular Risk in the Young Finns Study reported that exposure to parental smoking during childhood was associated with increased subclinical cardiovascular or cerebrovascular disease risk in adulthood.[13 14] Furthermore, it was found

that SHS exposure in childhood could increase adulthood composite carotid artery intima-media thickness.[15]

However, limited data are available to systematically assess the association between childhood SHS exposure and the onset of metabolic diseases, hypertension or coronary heart disease later in life. Therefore, the present study aims to evaluate the association between SHS exposure during childhood and the occurrence of type 2 diabetes, hypertension, hyperlipidaemia and coronary heart disease. This cross-sectional study included a large group of Chinese women who were non-smokers and who responded to a detailed questionnaire on childhood and current exposure to SHS.

METHODS

Study design and population

This cross-sectional study is part of the REACTION Study and was conducted from January to April 2012 in Shandong province, China.[16] Only women who had been living in their current residence for at least 6 months were allowed to participate. Thus, 6522 women who were ≥40 years old were invited and participated in a health examination. Among the 6522 women, 164 (2.5%) reported current smoking and 66 (1.0%) were former smokers. In addition, 1602 (24.6%) participants completed the questionnaire with a missing smoking status. Furthermore, 1576 (24.2%) women declared childhood exposure status within a mnemonically ambiguous range, and 994 (15.2%) had other missing status (figure 1). Ultimately, data from 2120 individuals, all of whom were non-smokers, were included in this analysis.

Data collection

A validated questionnaire was used to collect information on lifestyle, occurrence of some metabolic and heart diseases, medical history, medication use, and childhood and adulthood SHS exposure status. The participants responded to the validated questionnaire including detailed information on SHS exposure. Body weight and height were measured according to a standard protocol and body mass index (BMI). BMI was used as a measure of obesity and was calculated as weight in kilograms divided by the square of the height in metres. Blood pressure (BP) was measured three times consecutively (OMRON Model HEM-752 FUZZY, Omron Company, Dalian, China) on the left arm after participants had sat for at least 5 min, and the average reading was used for analysis. After at least 10 hours of overnight fasting, venous blood samples were collected between 07:00 and 09:00 for measurement of fasting blood glucose (FBG), total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C) for use in an automatic analyser (ARCHITECT ci16200 Integrated System, Abbott, USA). Postprandial blood glucose was measured after subjects had completed a 75 g oral glucose tolerance test (OGTT).

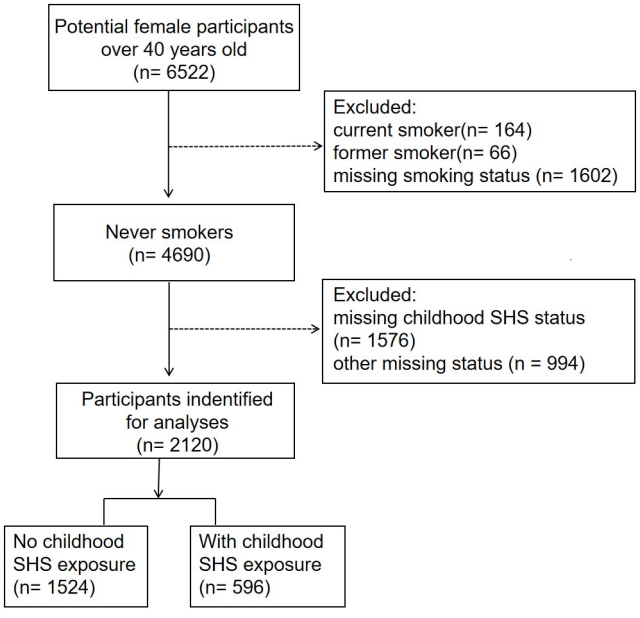

Figure 1 Flow chart for study design showing inclusion of participants in analyses. SHS, secondhand smoke.

Definitions

Childhood SHS exposure was defined based on questionnaire responses regarding the presence of smokers living in the participant's family during childhood. This was truncated at age 18 years, with exposure after this age being included in adulthood exposure. The current SHS exposure was defined as exposure to SHS more than once per week and for longer than 1 year indoors at home or the workplace. Current smoking was defined as having smoked 100 cigarettes in one's lifetime and currently smoking cigarettes. Alcohol consumption status was defined as alcohol intake more than once per month during the past 12 months. Physical activity was assessed using the short form of the International Physical Activity Questionnaire with additional questions on frequency and duration of mild, moderate and vigorous activities.[17] Obesity was defined by the WHO as a BMI of 30.0 or higher.[18] Hypertension was confirmed if participants reported a systolic BP of 140 mm Hg or higher, a diastolic BP of 90 mm Hg or higher, or being on drug therapy for hypertension.[19] According to the 1999 WHO criteria, a diagnosis of type 2 diabetes mellitus was based on an FBG of 7.0 mmol/L (126 mg/dL) or higher and/or a 2-hour OGTT plasma glucose of 11.1 mmol/L (200 mg/dL) or higher, or the current use of antidiabetic agents.[20] Participants were considered to have prior coronary heart disease if they provided a proof from the hospital that could prove they were ever diagnosed with myocardial infarction, acute coronary syndrome or other ischaemic heart diseases. Dyslipidaemia was defined as the presence of at least one of the following: TG of 2.26 mmol/L or higher, TC of 6.22 mmol/L or higher, LDL-C of 4.14 mmol/L or higher, or HDL-C less than 1.04 mmol/L.[21]

All investigators and research staff underwent a week-long training session on the use of standardised protocols

and instruments for data collection. Only certified staff were allowed to collect data.

Statistical analysis

Statistical analyses were conducted using IBM SPSS V.24 (IBM Corp) with $p<0.05$ considered to indicate statistical significance. Analysis of variance (for continuous variables) and the X^2 test (for categorical variables) were used to compare baseline characteristics across the childhood SHS exposure groups. Binary logistic regression analysis was used to address the relationship of diseases to SHS exposure categories, while adjusting for the risk factors of diseases including age, obesity, education status, physical activity, alcohol consumption, current SHS exposure status, diabetes mellitus, hypertension and hyperlipidaemia as reported on the background questionnaire. ORs and 95% CIs were calculated by logistic regression analysis. All of the analyses were stratified by exposure status and were restricted to never active smokers.

Patient and public involvement

Patients and/or the public were not involved in the design, conduct, reporting or dissemination plans of this research.

RESULTS

Baseline characteristics of the participants and study variables according to SHS exposure status during childhood are summarised in table 1. Of the 2120 study participants, 596 (28.1%) reported SHS exposure during childhood up until age 18 years, while 1524 (71.9%) claimed no SHS exposure prior to 18 years of age. As is shown in table 1, the mean age of the overall population was 55.52 years (±8.98 years) for the exposed participants and 57.23 years (±9.43 years) for the non-exposed participants. The prevalence of current SHS exposure was higher in the exposed group during childhood than in the non-exposed group (51.2% vs 32.1%). Additionally, the ratio of drinking was higher in the exposed group during childhood than in the non-exposed group (15.8% vs 8.3%). Exercise intensity and degree of education seemed to be significantly different between the childhood exposure group and the no SHS exposure group. However, there seemed to be no relationship between the ratio of obesity and the childhood SHS exposure status ($p>0.05$). Among the 2120 participants, the prevalence of diabetes mellitus, hypertension, hyperlipidaemia and coronary heart disease in the childhood SHS exposure group was 15.77%, 31.38%, 9.9% and 12.42%, respectively.

ORs and 95% CIs for some metabolic diseases and coronary heart diseases, stratified by SHS exposure status, are

Table 1 Baseline characteristics of the female participants stratified by SHS exposure status

Variables	SHS exposure in childhood, N (%)	No SHS exposure in childhood, N (%)	P value
Number of participants	596 (28.1)	1524 (71.9)	
Age, years (mean±SD)	55.52±8.98	57.23±9.43	<0.001
Obesity	68 (11.4)	197 (12.9)	0.342
Current SHS exposure status	305 (51.2)	489 (32.1)	<0.001
Alcohol consumption	94 (15.8)	127 (8.3)	<0.001
Physical activity			<0.001
Never	128 (21.5)	585 (38.4)	
Mild	377 (63.3)	800 (52.5)	
Moderate	52 (8.7)	74 (4.9)	
Vigorous	39 (6.5)	65 (4.3)	
Education status			<0.001
Illiteracy	84 (14.1)	199 (13.1)	
Primary school	100 (16.8)	306 (20.1)	
Junior school	220 (36.9)	638 (41.9)	
Senior school	140 (23.5)	323 (21.2)	
College degree or above	52 (8.7)	58 (3.8)	
Diabetes	94 (15.77)	279 (18.31)	0.168
Hypertension	187 (31.38)	547 (35.89)	0.054
Hyperlipidaemia	59 (9.90)	113 (7.41)	0.060
Coronary heart disease	74 (12.42)	169 (11.09)	0.389

P values were obtained from the X^2 test (for categorical variables) or analysis of variance.
SHS, secondhand smoke.

Table 2 Association between reported childhood SHS exposure and diseases adjusted for some variables

	Model 1		Model 2		Model 3	
	OR_1 (95% CI)	P value	OR_2 (95% CI)	P value	OR_3 (95% CI)	P value
Diabetes mellitus	0.94 (0.71 to 1.23)	0.628	/	/	/	/
Hypertension	0.96 (0.76 to 1.20)	0.691	/	/	/	/
Hyperlipidaemia	1.41 (0.99 to 2.00)	0.056	1.47 (1.02 to 2.11)	0.037	/	/
Coronary heart disease	1.44 (1.05 to 2.00)	0.026	1.50 (1.08 to 2.10)	0.017	1.41 (1.01 to 1.98)	0.045

Model 1: adjusted for age, obesity, education status, physical activity, alcohol consumption and current SHS exposure status.
Model 2: adjusted for age, obesity, education status, physical activity, alcohol consumption, current SHS exposure status, diabetes mellitus and hypertension.
Model 3: adjusted for age, obesity, education status, physical activity, alcohol consumption, current SHS exposure status, diabetes mellitus, hypertension and hyperlipidaemia.
SHS, secondhand smoke.

shown in table 2. As it is shown, model 1 was adjusted for age, obesity, education status, physical activity, alcohol consumption and current SHS exposure status. After adjusting for these variables, there was no correlation between the childhood SHS exposure status and the onset of diabetes mellitus (p=0.628). Meanwhile, the SHS exposure status during childhood was not associated with the onset of hypertension (p=0.691). In model 2, after further adjustments for age, obesity, education status, physical activity, alcohol consumption, current SHS exposure status, diabetes mellitus and hypertension, SHS exposure during childhood was associated with the occurrence of hyperlipidaemia (adjusted OR: 1.47; 95% CI: 1.02 to 2.11; p=0.037). Furthermore, there was a statistical difference between childhood SHS exposure and coronary heart disease in model 1 after adjusting for age, obesity, education status, physical activity, alcohol consumption and current SHS exposure status (p=0.026) as shown in table 2. At the same time, by adjusting age, obesity, education status, physical activity, alcohol consumption, current SHS exposure status, diabetes mellitus and hypertension, a significant relationship still exists between them (p=0.017). Considering hyperlipidaemia as the risk factor for coronary heart diseases,[10] model 3 was adjusted for age, obesity, education status, physical activity, alcohol consumption, current SHS exposure status, diabetes mellitus, hypertension and hyperlipidaemia. The SHS exposure status during childhood was associated with a higher risk of prevalent coronary heart disease (adjusted OR: 1.41; 95% CI: 1.01 to 1.98; p=0.045).

DISCUSSION

In this study, it was observed that SHS exposure was associated with hyperlipidaemia and coronary heart disease in a large, community-based population in China. To our knowledge, this is the first clinical study to systematically document a significant relationship between childhood SHS exposure and the occurrence of hyperlipidaemia and coronary heart disease in non-smoking women.

As shown in table 1, there was no statistical difference between childhood SHS exposure and these four diseases by X^2 test. The average age of people with childhood SHS exposure was significantly lower than that of people without childhood exposure. The occurrence of diabetes mellitus and hypertension is associated with age.[22 23] Thus, it is hypothesised that the age of the participants may have an impact on the results of the X^2 test, and in the case of similar ages, the risk difference of diabetes and hypertension between different groups may be more significant. Furthermore, the X^2 test indicated no association between hyperlipidaemia, coronary heart disease and childhood SHS exposure, which may also be confounded by age. Although the mean age of the childhood SHS exposure group was lower than that of the control group, the exposure group had slightly higher rates of hyperlipidaemia and coronary heart disease than in the control group. Therefore, it could be inferred that after adjustment of age, the risk of diseases in the exposed group would be higher than that in the current participant population.

One unanticipated result was that there was no statistical significance between diabetes mellitus and childhood smoking exposure. A prospective analysis suggested that SHS exposure in childhood was associated with a higher rate of type 2 diabetes.[24] In addition, a meta-analysis study including seven studies indicated that non-smokers with SHS exposure showed a 22% increased prevalence of type 2 diabetes mellitus compared with those who reported no exposure.[25] However, Houston *et al* found that there was no correlation between SHS exposure and diabetes incidence among non-smokers.[26] The relationship between these factors remains unknown and controversial. Therefore, further research is required to investigate the exact correlation and the potential intrinsic mechanism.

As shown above, there was no statistically significant correlation observed between childhood exposure and the onset of hypertension. A previous study in China observed no group differences between SHS exposure at one to three times per week and the risk of hypertension. However, SHS exposure at higher exposure rates was associated with a higher risk of hypertension.[27] Another study, involving Bulgarian former smokers and non-smokers,

found no significant association between SHS exposure and hypertension.[28] The mechanism and correlation linking SHS exposure to hypertension is not well elucidated and remains controversial,[3] which also requires further investigation in the future.

An ex vivo study performed in healthy non-smokers exposed to SHS indicated that exposure could result in lipid peroxidation, LDL-C modification and an accumulation of LDL-C in macrophages.[29] A meta-analysis study suggested that younger people who get exposed to SHS may be more susceptible to lipid metabolic disorder.[30] Another study reported that compared with control subjects, apolipoprotein B was lower in SHS-exposed children and youth.[31] In this investigation, we observed that the possibility of hyperlipidaemia in the childhood SHS exposure group was 1.47-fold compared with the unexposed group ($p<0.05$) after adjustment for possible confounders.

As shown in this study, the possibility of coronary heart disease in the childhood SHS exposure group was 1.41-fold compared with the unexposed group ($p<0.05$). A previous study found that exposure to SHS significantly increased the risk of coronary heart disease.[32] The risk associated with SHS exposure was large in China while the risk was only modest in the USA.[33] In addition, a previous study indicated that non-smokers exposed to SHS had a significantly increased risk of coronary heart disease by 22% compared with those without exposure.[34] The biological mechanisms that directly explain the association between childhood SHS exposure and coronary heart disease are still controversial and unclear. Although childhood exposure and adulthood exposure differ in the manner of exposure, the underlying mechanisms linking childhood SHS exposure to coronary heart disease may be similar to those implicated in SHS exposure. Previous studies have shown that the putative mechanisms by which SHS exposure is linked to coronary heart disease include impaired arterial structure, arterial dysfunction[10] and atherosclerosis formation.[6 35] When it comes to childhood exposure, a previous study indicated that SHS exposure in children and teenagers had a deleterious effect on their cardiovascular health and those outcomes as a consequence of SHS exposure may persist into their adult life.[10] It is hypothesised that this relationship may be explained by the fact that childhood exposure could activate platelet activation,[36] promote thrombus formation,[37] damage arterial endothelial cells and affect lipid metabolism,[33] all of which promote the progression of atherosclerosis, and ultimately result in cardiovascular consequences.[10]

Some countries have implemented comprehensive smoke-free public space legislation to protect non-smokers from SHS exposure, whereas a large proportion of the world's population is still confronted with SHS exposure, especially in the low-income and middle-income countries.[38 39] A 2019 national adolescent tobacco survey conducted by China's Center for Disease Control and Prevention revealed that SHS exposure in children and adolescents declined from 72.9% to 63.2% at home and in public places between 2014 and 2019; despite the decline, the probability of SHS exposure in children is still high.[40] In addition, children who have smoking parents are significantly more likely to be exposed to SHS and are more likely to smoke later in life.[41] This phenomenon may be related to plenty of inter-related factors, including SHS exposure itself, parental modelling or a physical tendency to SHS exposure. In the future, the control and prevention of SHS exposure deserves more attention.

This study was the first to elucidate the correlation between childhood SHS exposure and the occurrence of type 2 diabetes mellitus, hypertension, hyperlipidaemia and coronary heart disease in non-smoking women. An important advantage of this present study was the ability to determine the association between exposure to SHS and the occurrence of diabetes mellitus, hypertension, hyperlipidaemia and coronary heart disease. However, several limitations should be noted. First of all, SHS exposure data were collected using self-reported measures, without obtaining biomarker data such as polycyclic aromatic hydrocarbon or cotinine levels. Thus, recall and reporting biases may exist which can influence the accuracy of the outcomes to some extent. Another limitation was a lack of specific information on smoking status, such as intensity, amount, and daily or cumulative duration. Hence, the influence of extent and amount of SHS exposure on the onset of diseases could not be analysed. Besides, there were no specific categories and classifications of adulthood SHS smoking. People may have current SHS exposure not only at home or work, but also in other public or private places. The same people may be exposed at several different places, therefore, the amount and type of exposure cannot be determined clearly. Thus, we did not analyse the association between adulthood exposure and diseases, but treated this risk factor as an adjusted factor. Another limitation of this analysis was that this study used BMI as an evaluation criteria for obesity, without further measuring for body composition (ie, body fat percentage) and regional fat deposition by means of dual energy X-ray absorptiometry. Additionally, participants who were ever diagnosed with myocardial infarction, acute coronary syndrome or other ischaemic heart diseases could be diagnosed with coronary heart disease. We did not count the detailed number of each type of these diseases. Therefore, the association between these heart diseases and SHS exposure could not be analysed. In addition, the results were restricted to adult women over 40 years old, without including women of other ages and men. Finally, the cross-sectional survey employed herein does not allow for conclusions to be drawn concerning a possible causal influence of SHS exposure on hypertension and diabetes mellitus. Therefore, longitudinal designs are required in future investigations.

CONCLUSIONS

It was demonstrated in this study that childhood SHS exposure had a significant influence on hyperlipidaemia and coronary heart disease in a female non-smoking population, suggesting that SHS exposure in children could be an important risk factor for hyperlipidaemia and coronary heart disease development in their adult life. These findings suggested that SHS exposure in women represents an urgent public health event, especially with respect to SHS exposure during childhood. Thus, the Chinese government should take measures to increase awareness of the health dangers on SHS exposure and limit SHS exposure by providing tobacco-free environments and improve compliance with relevant policy.

Author affiliations
[1]Department of Endocrinology, Shandong University Qilu Hospital, Jinan, Shandong, China
[2]Institute of Endocrine and Metabolic Diseases of Shandong University, Jinan, China
[3]Key Laboratory of Endocrine and Metabolic Diseases, Shandong Province medicine & health, Jinan, China
[4]Jinan Clinical Research Center for Endocrine and Metabolic Diseases, Jinan, China

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Contributors KW was the principal investigator of the study, responsible for and main contributor to all phases of the study: the study design, quality assessment and the manuscript drafting. YW, RZ and LG collected the data. LW and QH analysed the data. LC and JQ participated in reviewing and revising the manuscript. All authors approved the final manuscript for publication.

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Patient consent for publication Not required.

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ORCID iD
Kewei Wang http://orcid.org/0000-0001-6504-5750

REFERENCES

1 Rashiden I, Ahmad Tajuddin Nur Amani @ Natasha bt, Yee A, *et al*. The efficacy of smoking ban policy at the workplace on secondhand smoking: systematic review and meta-analysis. *Environ Sci Pollut Res* 2020;27:29856–66.
2 Jeon J, Jung KJ, Kimm H, *et al*. Changes in secondhand smoke exposure levels and risk of type 2 diabetes in middle age: the Korean genome and epidemiology study (KoGES). *BMJ Open Diabetes Res Care* 2019;7:e000859.
3 Skipina TM, Soliman EZ, Upadhya B. Association between secondhand smoke exposure and hypertension: nearly as large as smoking. *J Hypertens* 2020;38:1899–908.
4 Liu Y, Dai M, Bi Y, *et al*. Active smoking, passive smoking, and risk of nonalcoholic fatty liver disease (NAFLD): a population-based study in China. *J Epidemiol* 2013;23:115–21.
5 Parekh TM, Wu C, McClure LA, *et al*. Determinants of cigarette smoking status in a national cohort of black and white adult ever smokers in the USA: a cross-sectional analysis of the REGARDS study. *BMJ Open* 2019;9:e027175.
6 Yankelevitz DF, Cham MD, Hecht H, *et al*. The association of Secondhand tobacco smoke and CT Angiography-Verified coronary atherosclerosis. *JACC Cardiovasc Imaging* 2017;10:652–9.
7 Hackshaw AK, Law MR, Wald NJ. The accumulated evidence on lung cancer and environmental tobacco smoke. *BMJ* 1997;315:980–8.
8 Dai H, Hao J. The prevalence of exposure to workplace Secondhand smoke in the United States: 2010 to 2015. *Nicotine Tob Res* 2017;19:1300–7.
9 Treyster Z, Gitterman B. Second hand smoke exposure in children: environmental factors, physiological effects, and interventions within pediatrics. *Rev Environ Health* 2011;26:187–95.
10 Raghuveer G, White DA, Hayman LL, *et al*. Cardiovascular consequences of childhood Secondhand tobacco smoke exposure: prevailing evidence, burden, and racial and socioeconomic disparities: a scientific statement from the American heart association. *Circulation* 2016;134:e336–59.
11 Cook DG, Strachan DP. Health effects of passive smoking. 3. parental smoking and prevalence of respiratory symptoms and asthma in school age children. *Thorax* 1997;52:1081–94.
12 Gerald LB, Gerald JK, Gibson L, *et al*. Changes in environmental tobacco smoke exposure and asthma morbidity among urban school children. *Chest* 2009;135:911–6.
13 Gall S, Huynh QL, Magnussen CG, *et al*. Exposure to parental smoking in childhood or adolescence is associated with increased carotid intima-media thickness in young adults: evidence from the cardiovascular risk in young Finns study and the childhood determinants of adult health study. *Eur Heart J* 2014;35:2484–91.
14 West HW, Juonala M, Gall SL, *et al*. Exposure to parental smoking in childhood is associated with increased risk of carotid atherosclerotic plaque in adulthood: the cardiovascular risk in young Finns study. *Circulation* 2015;131:1239–46.
15 Chen W, Yun M, Fernandez C, *et al*. Secondhand smoke exposure is associated with increased carotid artery intima-media thickness: the Bogalusa heart study. *Atherosclerosis* 2015;240:374–9.
16 Ning G, Reaction Study Group. Risk evaluation of cAncers in Chinese diabeTic individuals: a lONgitudinal (reaction) study. *J Diabetes* 2012;4:172–3.
17 Craig CL, Marshall AL, Sjöström M, *et al*. International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc* 2003;35:1381–95.
18 World Health Organisation. Obesity: preventing and managing the global epidemic. Report of a who consultation. *World Health Organ Tech Rep Ser* 2000;894:i–253.
19 Wang Z, Chen Z, Zhang L, *et al*. Status of hypertension in China: results from the China hypertension survey, 2012-2015. *Circulation* 2018;137:2344–56.
20 Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a who consultation. *Diabet Med* 1998;15:539–53.
21 Li Y, Zhao SP, Ye P, *et al*. [Status of cholesterol goal attainment for the primary and secondary prevention of atherosclerotic cardiovascular disease in dyslipidemia patients receiving lipid-lowering therapy: DYSIS-China subgroup analysis]. *Zhonghua Xin Xue Guan Bing Za Zhi* 2016;44:665–70.
22 Akhtar S, Nasir JA, Sarwar A, *et al*. Prevalence of diabetes and pre-diabetes in Bangladesh: a systematic review and meta-analysis. *BMJ Open* 2020;10:e036086.
23 Wang C, Yuan Y, Zheng M, *et al*. Association of Age of Onset of Hypertension With Cardiovascular Diseases and Mortality. *J Am Coll Cardiol* 2020;75:2921–30.
24 Lajous M, Tondeur L, Fagherazzi G, *et al*. Childhood and adult secondhand smoke and type 2 diabetes in women. *Diabetes Care* 2013;36:2720–5.
25 Pan A, Wang Y, Talaei M, *et al*. Relation of active, passive, and quitting smoking with incident type 2 diabetes: a systematic review and meta-analysis. *Lancet Diabetes Endocrinol* 2015;3:958–67.

26 Houston TK, Person SD, Pletcher MJ, *et al*. Active and passive smoking and development of glucose intolerance among young adults in a prospective cohort: cardia study. *BMJ* 2006;332:1064–9.
27 Li N, Li Z, Chen S, *et al*. Effects of passive smoking on hypertension in rural Chinese nonsmoking women. *J Hypertens* 2015;33:2210–4.
28 Sipetić Grujičić S, Terzić Supić Z, Nikolić Željka, *et al*. Risk factors for the development of arterial hypertension. *Med Glas* 2014;11:19–25.
29 Valkonen M, Kuusi T. Passive smoking induces atherogenic changes in low-density lipoprotein. *Circulation* 1998;97:2012–6.
30 Chen H-J, Li G-L, Sun A, *et al*. Age differences in the relationship between Secondhand smoke exposure and risk of metabolic syndrome: a meta-analysis. *Int J Environ Res Public Health* 2019;16:1409.
31 Yang B, Li M, Chen B, *et al*. Deterioration of endothelial function and carotid intima-media thickness in Tibetan male adolescents exposed to second-hand smoke. *J Renin Angiotensin Aldosterone Syst* 2012;13:413–9.
32 Asfar T, Koru-Sengul T, Ruano-Herreria EC, *et al*. Secondhand smoke exposure among high-risk patients in the United States (NHANES 2001-2012): implications for clinical practice. *Nicotine Tob Res* 2019;21:551–6.
33 Lv X, Sun J, Bi Y, *et al*. Risk of all-cause mortality and cardiovascular disease associated with secondhand smoke exposure: a systematic review and meta-analysis. *Int J Cardiol* 2015;199:106–15.
34 Zhang D, Liu Y, Cheng C, *et al*. Dose-related effect of secondhand smoke on cardiovascular disease in nonsmokers: systematic review and meta-analysis. *Int J Hyg Environ Health* 2020;228:113546.
35 Barnoya J, Glantz SA. Cardiovascular effects of secondhand smoke: nearly as large as smoking. *Circulation* 2005;111:2684–98.
36 Enstrom JE, Kabat GC. Environmental tobacco smoke and coronary heart disease mortality in the United States--a meta-analysis and critique. *Inhal Toxicol* 2006;18:199–210.
37 Dunbar A, Gotsis W, Frishman W. Second-hand tobacco smoke and cardiovascular disease risk: an epidemiological review. *Cardiol Rev* 2013;21:94–100.
38 Yao T, Sung H-Y, Mao Z, *et al*. The healthcare costs of secondhand smoke exposure in rural China. *Tob Control* 2015;24:e221–6.
39 Sansone G, Fong GT, Yan M, *et al*. Secondhand smoke exposure and support for smoke-free policies in cities and rural areas of China from 2009 to 2015: a population-based cohort study (the ITC China survey). *BMJ Open* 2019;9:e031891.
40 Chinese Center for Disease Control and Prevention. Chinese center for disease control and prevention released 2019 China adolescent tobacco survey report, 2015. Available: http://www.chinacdc.cn/jkzt/sthd_3844/slhd_4156/202005/t20200531_216942.html [Accessed 30 May 2019].
41 Thomson B, Rojas NA, Lacey B, *et al*. Association of childhood smoking and adult mortality: prospective study of 120 000 Cuban adults. *Lancet Glob Health* 2020;8:e850–7.