Association Between Self-Reported Tobacco Smoke Exposure and Serum Non-High-Density Lipoprotein Among Adults, Beijing

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Research

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Abstract

**Background:** Cigarette smoking, including secondhand exposure, is a leading cause of several atherosclerotic diseases (AS). Non-high-density lipoprotein cholesterol (non-HDL-C) is considered as a valuable predictor for dyslipidemia and AS. There is a need to identify the association of tobacco smoke exposure (TSE) and the marker of sub-clinical AS.

**Objective:** To examine the association of TSE, including exposed status, cigarette smoking intensity and burden with serum non-HDL-C level.

**Methods:** A cross-sectional study was carried out on 18-79 years old residents in 2017 in Beijing. All participants were interviewed on their tobacco exposed status (unexposed, passively exposed, actively exposed), smoking intensity (number of cigarettes smoked per week) and smoking burden (pack-years of smoking) among current smokers. Non-HDL-C was calculated by subtracting high density lipoprotein cholesterol (HDL-C) from total cholesterol (TC). Covariates were age, education status, alcohol drinking status within 30 days, hypertension, type 2 diabetes, physical inactivity and BMI. We plot graphs to show the tendency between the number of cigarettes smoked per week, passively exposed days per week and non-HDL-C levels, respectively. Multiple regression models were used to assess the association between non-HDL-C level and TSE after the adjustment for covariates.

**Results:** Of the 12,798 participants, mean age was 44.9±15.4 years, 46.8% were male. The prevalence of actively exposed, passively exposed and unexposed to tobacco smoke was 25.7%, 35.7% and 38.6%, respectively. Of the tobacco passively exposed subjects, 21.7% had to exposed to second-hand smoke every day. Among current smokers, 41.2% were smoking more than 120 cigarettes per week, and 56.6% were smoking more over 20 pack-years. With the increasing of the number of cigarettes smoked per week, the serum level of non-HDL-C in male increased gradually (Male: F=2.83, P=0.04; Female: F=1.23, P=0.32; Total: F=3.29, P=0.02). After multivariable adjustment, smokers had a higher non-HDL-C level (OR=1.34, 95%CI: 1.20,1.59, P=0.001) compared with tobacco unexposed subjects. However, passively smokers were not associated with non-HDL-C levels regardless of gender (OR=0.98, 95%CI: 0.78,1.23, P=0.87). Compared with lighter smokers, male smokers who smoked more than 140 cigarettes per week had significantly higher non-HDL-C levels (OR=1.30, 95%CI: 1.00,1.70, P=0.04). Male higher burden smokers (>20 pack-years) had more risk of having higher non-HDL-C level (OR=1.88, 95%CI: 1.46,2.43, P<0.001) compared with other smokers. However, there were no significant differences between non-HDL-C and smoking intensity or burden among female smokers.

**Conclusions:** This study showed that strong associations between TSE and non-HDL-C levels, especially in male smokers. Findings of this work emphasize the importance of encouragement to focus on blood lipid levels among smokers.

Background

Cigarette smoking, including secondhand exposure, is a leading cause of death and disability in the world. Annually, more than 1.5 million Chinese people die from tobacco use. Although the rate of smoking have declined since the 1996s and has decreased from 63.0–52.1% among male in China, which is still much higher than the world average. Dyslipidemia plays an important role in the formation and development of atherosclerosis (AS), and its association with the risk of CVD is indisputable. Therefore, early identifying sensitive bio-markers of dyslipidemia may substantially reduce cardiovascular damage. Non-high-density lipoprotein cholesterol (non-HDL-C), which includes the cholesterol carried by apolipoprotein B-containing particles, has a clear pathophysiologic link to the development of AS and it is a crucial treatment target in primary prevention of coronary heart disease. Nicotine is associated with hemodynamic alterations, dyslipidemia and insulin resistance. Recent findings from NHANES (National Health and Nutrition Examination Survey) in the United States demonstrated that compared to unexposed, participants with active tobacco smoke exposure (TSE) had lower high-density lipoprotein cholesterol (HDL-C) and higher total cholesterol (TC) among adolescents. However, non-HDL-C levels varied in terms of sex, age and ethnic group, and little was known about these associations in Chinese adults. Therefore more research is needed to elucidate the relationship between TSE and lipid profiles in adults.

In the present study, a cross-sectional study was performed to investigate the associations between TSE and non-HDL-C in a Beijing adult population. The hypothesis was that TSE, as assessed by tobacco exposed status (unexposed, passively exposed, actively exposed), smoking intensity (cigarettes per week) and smoking burden (pack-years), is associated with serum non-HDL-C levels.

Materials And Methods

**Study design and population**

This study used data from Beijing Chronic Disease and Risk Factors Surveillance (BCDRFS) in 2017, a cross-sectional study conducted by Beijing Center for Disease Prevention and Control (Beijing CDC). The surveillance covered all 16 districts, 55 township streets, 165 villages or neighborhood committees of Beijing, and 13,240 residents aged 18 to 79 years old were investigated. Among them, 12,798 valid samples were analyzed. The study protocol was approved by the Ethical Review Board of BJCDC (The number is no. 5 of 2017). All participants signed written informed consent.

The contents of the surveillance included face-to-face questionnaire interviews, physical measurements and laboratory tests. The questionnaire collected information on demography, status of having chronic non-communicable diseases (NCD) and risk factors, including tobacco use, alcohol consumption, physical activity, food consumption, etc.
Height, weight, waist circumference, and blood pressure were measured. Height was measured in the standing position to the nearest 0.1 cm without shoes. Weight was measured to the nearest 0.1 kg while the subjects were minimally clothed without shoes. Body mass index (BMI) was calculated as weight (kg) divided by square of height (m$^2$). Overweight and obesity were defined based on BMI of 24 and 28 Kg/m$^2$ as cutoff points recommended by the Working Group on Obesity in China\textsuperscript{13}. Resting blood pressure (BP) and heart rate were measured 3 times with 1-minute intervals in the sitting position after 15 min of rest on the left arm by standard methods using electronic sphygmomanometer (HBP-1300, OMRON). Hypertension was defined as a systolic blood pressure (SBP) $\geq$ 140 mmHg, and/or diastolic BP (DBP) $\geq$ 90 mmHg, or previous medical diagnosis and the use of antihypertensive medications in the last two weeks\textsuperscript{14}. Diabetes mellitus (DM) was defined as either previous medical diagnosis of DM, or fulfillment of the diagnostic criteria for diagnosis based on fasting $\geq$ 126 mg/dl (7.0 mmol/l) in this survey\textsuperscript{15}. Fasting blood samples were obtained from participants after a 10 hours’ overnight fast. TC and HDL-C were measured by an enzymatic method using Hitachi auto-analyzer (Tokyo, Japan). We calculated non-HDL-C by subtracting HDL-C from TC.

### Ascertainment Of Tse

The question on tobacco use included frequency, amount of smoking currently and in the past, and smoking cessation. Smoker was defined as a documented lifetime use of more than 100 cigarettes\textsuperscript{16,17}. The primary TSE variables were tobacco exposure status (unexposed, passively exposed, actively exposed), smoking intensity (number of cigarettes smoked per week among current smokers) and smoking burden (pack-years of smoking).

Based on the self-reported smoking patterns, tobacco exposure status was divided into three subgroups: tobacco unexposed (non-smoking and no exposure to secondhand smoke), passively exposed (non-current smokers but exposed to secondhand smoke), actively exposed (current smokers).

Smoking intensity and burden were two measures of tobacco exposure levels among current smokers, who were defined as smoking within the previous 30 days. Smoking intensity could reflect short-term exposure levels and it was divided into three categories: current light smoker (< 70 cigarettes per week), moderate smoker ($\geq$ 70 and < 140 cigarettes per week) and heavy smoker ($\geq$ 140 cigarettes per week)\textsuperscript{18}. Smoking burden which was a comprehensive indicator of smoking amount and duration, was divided into two categories according to the median level: lower burden (< 20 pack-years) and higher burden ($\geq$ 20 pack-years)\textsuperscript{19}. Pack-years of smoking was defined as the product of the average number of packs of cigarette smoked per day multiplied by the average days of smoking in one year.

### Covariates

We selected covariates into the analysis, which were age, gender, education status, alcohol drinking within 30 days, hypertension, type 2 diabetes, physical inactivity and overweight and obesity. Education status was self-reported as primary school and below, secondary school, or university degree and above. According to whether they had consumed alcohol within 30 days, participants were divided into two groups as current drinkers or not. Physical inactivity was defined as less than 150 minutes of moderate intensity activity or equivalent quantity per week according to WHO classification criteria\textsuperscript{20}.

### Statistical analysis

Statistical analyses were performed in complex sampling module which considering sampling weight and post-weight using SPSS software (ver. 21.0 for Windows; SPSS, Chicago, IL, USA) for all analyses. All tests were two-sided, and $P < 0.05$ was considered statistically significant.

Differences between covariates (i.e., age, education status, current drinking, physical inactivity, hypertension, diabetes, BMI, non-HDL-C) and tobacco exposed status (i.e., actively exposed, passively exposed, unexposed) were examined by performing logistic regression models adjusting age and sex (Table 1). Moreover, subgroup analysis was performed according to sex to explore the distribution of population characteristics among different genders.
Table 1: Age-adjusted demographic characteristics by tobacco exposure status for Beijing participants, aged 18–79.

| Variable                        | Total subjects | Overall (N = 12798) | Men (N = 5994) | Actively exposed (n = 3286) | Passively exposed (n = 4573) | Unexposed (n = 4939) | Actively exposed (n = 3068) | Passively exposed (n = 1414) | Unexposed (n = 1512) | P   |
|--------------------------------|----------------|---------------------|----------------|----------------------------|-----------------------------|----------------------|----------------------------|----------------------------|----------------------|-----|
| Age (X ± SD, y)\(^a\)         | 47.8 ± 0.6     | 46.3 ± 0.5          | 45.5 ± 0.6     | 51.2 ± 0.9                 | < 0.01                     | 46.2 ± 0.5           | 45.1 ± 0.7                 | 52.5 ± 1.2                 | < 0.01               |     |
| Education status (%)\(^b\)    |                |                     |                |                            |                             |                      |                            |                            |                      |     |
| Primary school                 | 9.2            | 9.3                 | 7.6            | 10.5                       | < 0.01                     | 9.0                  | 6.3                       | 10.0                      | < 0.01               |     |
| Secondary school               | 64.7           | 71.0                | 64.1           | 59.4                       |                            | 71.1                 | 67.0                      | 60.7                      |                     |     |
| College degree                 | 26.0           | 19.7                | 28.3           | 30.2                       |                            | 19.8                 | 26.7                      | 29.3                      |                     |     |
| Current drinking within 30d (%)\(^b\) | 37.3          | 57.4                | 33.3           | 21.7                       | < 0.01                     | 58.3                 | 49.8                      | 32.4                      | < 0.01               |     |
| Physical inactivity (%)\(^b\) | 54.3           | 50.9                | 60.0           | 52.7                       | < 0.01                     | 50.4                 | 54.3                      | 44.2                      | < 0.01               |     |
| Hypertension (%)\(^b\)        | 35.4           | 37.1                | 31.0           | 37.6                       | 0.88                       | 37.1                 | 34.9                      | 44.3                      | 0.56                 |     |
| Diabetes (%)\(^b\)            | 13.5           | 13.7                | 12.1           | 14.4                       | 0.81                       | 13.7                 | 12.2                      | 16.9                      | 0.55                 |     |
| BMI (IQR, kg/m\(^2\))\(^c\)   | 25.4 (23.1,27.9)| 25.6(23.3,28.2)     | 25.3(23.0,28.1)| 25.4(23.1,27.7)           | 0.01                       | 25.6(23.4,28.2)       | 25.7(23.4,28.3)          | 25.9(23.7,27.9)          | 0.44                 |     |
| Non-HDL-C (IQR,mmol/l)\(^c\)  | 3.3(2.8,3.9)   | 3.4 (2.8, 4.0)      | 3.3(2.7, 3.8)  | 3.3(2.7, 3.9)              | < 0.01                     | 3.4 (2.8, 4.0)        | 3.3 (2.8, 3.8)           | 3.3 (2.7, 3.8)           | < 0.01               |     |

Note: \(^a\)Data are presented as mean (SD). \(^b\)Data are presented as number (percentage). \(^c\)Data are presented as median (IQR). The estimates for age are not ad

Distribution of tobacco exposure status among male and female were showed Table 2. Secondhand smokers (tobacco passively exposed) were divided into 4 subgroups (0, 1 ~ 3, 4 ~ 6 and 7) according to days per week passively exposed to tobacco. Current smokers (tobacco actively exposed) were divided into 5 subgroups according to numbers of cigarettes smoked per week (< 40, 40 ~, 80 ~, 120 ~, and ≥ 160). Moreover, smoking intensity (light, moderate, heavy) and smoking burden (lower, higher) among current smokers were showed in Table 2. Logistic regression models were used to explore the different distribution in the same gender groups.
### Table 2
Associated variables of tobacco exposure status among male and female

| Variable                                         | Total (n, weighted %)<sup>a</sup> | Subgroup analysis (n, weighted %)<sup>b</sup> |
|--------------------------------------------------|-----------------------------------|-----------------------------------------------|
|                                                  | Male | Female |
| Passively exposure among non-smokers (day/week, %) |      |        |
| 0                                                | 1512 (55.7) | 3427 (52.4) |
| 1–3                                              | 643 (20.6)  | 1111 (16.3) |
| 4–6                                              | 225 (6.9)   | 303 (4.4)   |
| 7                                                | 546 (6.8)   | 1745 (27.0) |
| $\chi^2$                                         | 684.7 | 785.3 |
| P value for trend                                  | < 0.01 | < 0.01 | < 0.01 |
| Actively exposure among current smokers (n/week, %) |      |        |
| < 40                                             | 545 (17.8)  | 84 (38.1)   |
| 40~                                              | 915 (28.1)  | 70 (34.8)   |
| 80~                                              | 351 (12.2)  | 15 (5.6)    |
| 120~                                             | 985 (33.3)  | 41 (18.7)   |
| $\geq$ 160                                       | 261 (8.6)   | 7 (2.7)     |
| $\chi^2$                                         | 322.8 | 50.9 |
| P value for trend                                  | < 0.01 | < 0.01 | < 0.01 |
| Smoking intensity status (n, %)<sup>b</sup>       |      |        |
| Light                                            | 772 (24.9)  | 106 (49.1) |
| Moderate                                         | 1073 (33.8) | 64 (29.5)  |
| Heavy                                            | 1212 (41.3) | 47 (21.4)  |
| $\chi^2$                                         | 43.2 | 13.8 |
| P value for trend                                  | < 0.01 | < 0.01 | 0.001 |
| Smoking burden status (n, %)<sup>c</sup>         |      |        |
| Lower burden                                     | 1278 (42.7) | 109 (65.3) |
| Higher burden                                    | 1316 (57.3) | 62 (34.7)  |
| $\chi^2$                                         | 20.7 | 7.6 |
| P                                                | < 0.01 | < 0.01 | 0.01 |

Note:  
- <sup>a</sup> n was expressed as un-weighted number and proportion was weighted.  
- <sup>b</sup> Smoking intensity was divided into three categories: current light smoker (< 70 cigarettes per week), moderate smoker ($\geq$ 70 and < 140 cigarettes per week) and heavy smoker ($\geq$ 140 cigarettes per week).  
- <sup>c</sup> Smoking burden was divided into two categories according to the median level: lower burden (< 20 pack-years) and higher burden ($\geq$ 20 pack-years).

Average non-HDL-C levels of subjects in each subgroup was calculated and plotted. The ANOVA analysis was conducted to compare the differences between groups (Fig A-B).

Participants were divided into two groups according to the mean value of serum non-HDL-C level with the cut-off value as 3.35mmol/l. Multivariate logistic regression analysis controlling for confounding factors including age, education status, alcohol drinking within 30 days, hypertension, type 2 diabetes and BMI was also performed to examine the association between non-HDL-C level and tobacco exposed status, smoking intensity or burden. Data are presented as odds ratio (OR) with 95% confidence interval (CI) (Table 3–4).
Table 3

Logistic regression results to estimate of association between higher non-HDL-C levels (cut-off value: 3.35mmol/l) and tobacco exposed status

| Participants | Tobacco exposed status | higher non-HDL-C |
|--------------|------------------------|------------------|
|              |                        | $\chi^2$ | OR (95%CI) | $P$   |
| Total        | Unexposed              | Reference      |          |       |
|              | Passively exposed      | 0.03     | 0.98 (0.78,1.23) | 0.87  |
|              | Actively exposed       | 11.34    | 1.34(1.20,1.59)  | 0.001 |
| Male         | Unexposed              | Reference      |          |       |
|              | Passively exposed      | 0.04     | 0.96 (0.82,1.13)  | 0.84  |
|              | Actively exposed       | 8.00     | 1.30 (1.08,1.56)  | 0.005 |
| Female       | Unexposed              | Reference      |          |       |
|              | Passively exposed      | 0.21     | 0.97 (0.82,1.14)  | 0.65  |
|              | Actively exposed       | 0.51     | 1.13 (0.79,1.62)  | 0.48  |

Note: Adjusted for age, education status, alcohol drinking within 30 days, hypertension, type 2 diabetes, physical inactivity and BMI.

Table 4

Logistic regression results between higher non-HDL-C levels (cut-off value: 3.43mmol/l) and smoking intensity and burden among current smokers

| Participants | Total          | Male           | Female          |
|--------------|----------------|----------------|-----------------|
|              | $\chi^2$ | OR (95%CI) | $P$ | $\chi^2$ | OR (95%CI) | $P$ | $\chi^2$ | OR (95%CI) | $P$ |
| Smoking intensity: Light smoker as reference |
| Moderate smoker | 0.96 | 1.09 (0.92,1.29) | 0.33 | 1.16 | 1.10 (0.92,1.33) | 0.28 | 0.44 | 0.76 (0.33,1.77) | 0.51 |
| Heavy smoker | 4.7     | 1.30 (1.02,1.67) | 0.03 | 4.11 | 1.30 (1.00,1.70) | 0.04 | 0.62 | 1.60 (0.48,5.35) | 0.43 |
| Smoking burden: Lower burden as reference |
| Higher burden | 27.48 | 1.85 (1.46,2.35) | <0.001 | 25.03 | 1.88 (1.46,2.43) | <0.001 | 0.34 | 1.43 (0.41,4.98) | 0.57 |

Note: Adjusted for age, education status, alcohol drinking within 30 days, hypertension, type 2 diabetes, physical inactivity and BMI.

Results

Demographics and clinical characteristics of the study population

The demographics and clinical characteristics of the study population were shown in Table 1. Among the 13,240 participants, 442 subjects were excluded because of the important information lost, such as smoking status and laboratory examinations. The remaining 12,798 participants were divided into three categories according to tobacco exposure status, actively exposed (n = 3286), passively exposed (n = 4573) and unexposed (n = 4939), respectively.

Of all participants, the mean age of tobacco actively exposed, passively exposed and unexposed groups were 46.3 ± 0.5, 45.5 ± 0.6 and 51.2 ± 0.9 years old, respectively. Of specific interest, tobacco actively exposed, passively exposed subjects were younger than unexposed subjects ($P < 0.01$). While a majority of men were current smokers (51.2%), and 50.4% of women were tobacco unexposed subjects. There were differences in age and education status among three subgroups after adjusting for age and sex.

The tobacco actively exposed subjects had lower proportions of physical inactivity (50.9% vs. 60.0% vs. 52.7%, $P < 0.01$) and higher proportions of current drinkers (57.4% vs. 33.3% vs. 21.7%, $P < 0.01$) than other two subgroups adjusted for age and gender. Median BMI and Non-HDL-C levels were significant higher in current smokers (BMI: 25.6kg/m$^2$, IQR: 23.3-28.2kg/m$^2$; Non-HDL-C: 3.4mmol/L, IQR: 2.8-4.0mmol/L) than other two subgroups.

Characteristics Of Tobacco Exposure Status Among Exposed Participants

Of the tobacco passively exposed subjects, 21.7% had to exposed to second-hand smoke every day and the proportion among female was up to 27.0%. Among current smokers, 41.2% were smoking more than 120 cigarettes per week, and 56.6% were heavy burden smokers who smoked more over 20 pack-years (Table 2).

Mean Non-hdl-c Levels According To Tobacco Exposed Status

The tendency for average non-HDL-C levels according to numbers of cigarettes smoked per week in smokers, and days per week passively exposed among non-smokers are respectively showed in Fig A-B. With the increasing of the number of cigarettes smoked per week, the serum level of non-HDL-C increased
gradually among male smokers ($F = 2.83, P = 0.04$) (Fig A). However, there was no difference therein among passively exposed subjects (Fig B).

TSE With Elevated Non-HDL-C Levels

The results of stratification analysis and multi-factor logistic regressions between non-HDL-C and tobacco exposed status are presented in Table 3. Male current smokers had significantly more risk of having higher non-HDL-C level than tobacco unexposed participants ($OR_{total}=1.34$, $95\% CI = 1.20, 1.59$, $P < 0.001$; $OR_{male}=1.30$, $95\% CI = 1.08, 1.56$, $P = 0.005$). However, passively smoking was not associated with non-HDL-C level regardless of gender ($OR_{total}=0.98$, $95\% CI = 0.78, 1.23$, $P = 0.87$; $OR_{male}=0.96$, $95\% CI = 0.82, 1.13$, $P = 0.84$; $OR_{female}=0.97$, $95\% CI = 0.82, 1.14$, $P = 0.65$).

Current Smokers With Elevated Non-HDL-C Levels

Table 4 showed the logistic regressions results between non-HDL-C status and smoking intensity or burden among current smokers. Compared with lighter smokers, heavy smokers had significantly higher non-HDL-C levels ($OR = 1.30$, $95\% CI = 1.02, 1.67$, $P = 0.03$) after adjusting for age, sex, education status, alcohol drinking within 30 days, hypertension, type 2 diabetes, physical inactivity and BMI. Higher burden smokers had more risk of higher non-HDL-C levels ($OR = 1.85$, $95\% CI = 1.46, 2.35$, $P < 0.001$) compared with other current smokers. Subgroup analysis of different sex explored these relationships among male smokers remain significant. However, of the female smokers, the association between smoking intensity or burden and non-HDL-C levels were not found.

Discussion

Cigarette smoking has been linked to progression of AS, which may necessitate different measures of exposures and bio-markers. Given the long latency period between tobacco exposure and AS, the identification of sensitive bio-markers of dyslipidemia is important for assessment of potential AS. Several studies have recommended the use of non-HDL-C as a valuable predictor for dyslipidemia and cardiovascular risks in the current study, we demonstrated that tobacco actively exposed (current smoking), smoking intensity and burden were all positively associated with higher levels of the AS bio-marker non-HDL-C in the general adult population in Beijing, even after adjusting for several parameters that might affect the result. These data suggested that non-HDL-C may be useful bio-markers of smoking-induced AS, which might predict the risk and process of AS diseases and carry out preventive measures among smokers.

The relations between non-HDL-C and TSE in this study were consistent to some previous studies. Cigarette smoking was observed to increase non-HDL-C levels in middle-age male Japanese subjects. Smoking was associated with high non-HDL-C in non-drinkers at middle and elderly ages (40s or older) and the authors suggested that elderly smokers had better stop or reduce smoking from the viewpoint of prevention of dyslipidemia. Moreover, Yang et al. discovered an increased smoking amount was significantly associated with a decreased HDL-C level. However, there were some inconsistent results from other studies. According to Srinivasan et al.’s study, non-HDL-C levels varied in terms of sex, age and ethnic group. The possible reason of these mixed findings was the significant difference on the definition of tobacco exposure status.

Smoking intensity was positively associated with hsCRP independent of the duration of exposure, which suggested that acute tobacco exposure was associated with subclinical vascular damage. Smoking burden as an indicator of chronic tobacco exposure was related to abnormalities in all markers of inflammation, plate aggregation and/or endothelial dysfunction. In this study, we found male smokers who smoked more than 140 cigarettes per week and more than 20 pack-years, had significantly higher non-HDL-C levels. Further studies and objective assessment of TSE are needed to elucidate the underlying mechanisms and the causal effects of smoking on dyslipidemia.

In the present study, no significant associations were found between second-hand smokers and elevated non-HDL-C. Several studies performed in Asian populations also did not find any consistent relationship between secondhand smoke (SHS) and lipid levels among adults, but the effects of secondhand smoke exposure on lipid index among adolescents had been confirmed by several studies. Chen et al. made meta-analyses and reported secondhand smoke (SHS) was negatively associated with HDL-C in the lower age group (7–18 years), but no effect in the upper age group (27–74 years). Children and teenagers are at a critical stage of growth and development, and their blood lipids are susceptible to living environmental factors, such as smoking, pesticides. This may explain why SHS is more likely to lead to significant dyslipidemia in younger people than in adults. Further longitudinal assessments of exposure to tobacco in adults are required to validate the findings.

Strengths And Limitations

One of the strengths of our study is a cross-sectional study based a complex, multistage probability sampling strategy, covering all 16 districts, 55 township streets, 165 villages or neighborhood committees of Beijing, which made it representative. This study has some limitations. First, the cross-sectional design of this study did not allow to obtain any causality between non-HDL-C concentration and smoking habits. Second, the information of TSE was reported by the participants. Performing objective approach to assess serum cotinine could provide more accurate information to clarify these associations. Third, we adjusted for potential confounders, but residual confounding may have occurred and unexpectedly biased our results.

Conclusions

In conclusion, there were significant association between TSE and non-HDL-C levels. Smokers had higher non-HDL-C levels than tobacco unexposed participants. With the increase of the number of cigarettes smoked per week, the serum level of non-HDL-C in current male smokers was gradually increasing.
Higher burden male smokers (> 15 pack-years) had more risk of higher non-HDL-C levels compared with other smokers. Smokers should pay attention on their blood lipid levels in order to prevent atherosclerosis.

**Declarations**

- Ethics approval and consent to participate

The study protocol was approved by the Ethical Review Board of BJCDC (The number is no. 5 of 2017). All participants signed written informed consent.

- Consent for publication

All authors have approved this manuscript for submission.

- Competing interests

There was no any potential competing interests.

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