Research Square

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Association of Occupational Environmental Hazardous Factors with Carotid Atherosclerosis and the Mediating Role of Apolipoprotein B: A Cross-Sectional Study in Steelworkers

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Research Article

Keywords: Occupational environmental hazardous factors, Apolipoprotein B, Carotid atherosclerosis, Steelworkers

Posted Date: June 3rd, 2025

DOI: https://doi.org/10.21203/rs.3.rs-6561180/v1

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Additional Declarations: No competing interests reported.

Abstract

Background: This study aimed to investigate the relationship between major occupational environmental hazards (heat, noise, CO, and dust) encountered by steelworkers and carotid atherosclerosis (CAS), and to explore the potential role of apolipoprotein B (ApoB) in this association.

Methods: This cross-sectional study utilized baseline data from the 2017 Beijing-Tianjin-Hebei occupational cohort, focusing on steelworkers and ultimately including 4,223 participants. Multivariable logistic regression, linear regression, and restricted cubic spline models were employed to evaluate the associations between occupational hazards, ApoB, and CAS. Mediation analysis was conducted to investigate the potential mediating role of ApoB in the relationship between occupational hazards and CAS.

Results: There were 1113 cases of CAS among 4223 steelworkers. Cumulative exposures to heat, noise, CO, and dust were each associated with an increased risk of CAS. Compared to individuals in the lowest quartile of cumulative exposure, those in the highest quartile had Odds Ratios (*ORs*) for CAS of 2.033 (95% *CI*: 1.538–2.686) for heat, 1.497 (95% *CI*: 1.201–1.864) for noise, 2.303 (95% *CI*: 1.766–3.004) for CO, and 1.376 (95% *CI*: 1.118–1.693) for dust. Workers with higher exposure to occupational hazards and elevated ApoB level faced a greater CAS risk. ApoB mediated 9.62%–48.92% of the association between occupational hazards and CAS.

Conclusion: Long-term exposure to occupational hazards may increase CAS risk among steelworkers, particularly those with high serum ApoB level. Elevated ApoB level may serves as a mechanism linking occupational hazards to CAS.

Introduction

Cardiovascular diseases (CVDs) are the leading cause of global disability and premature mortality [1, 2]. In 2015, about 422 million people globally suffered from CVDs, causing 17.9 million deaths—31% of all deaths [3]. By 2030, approximately 23.6 million people are projected to die annually from CVDs [3]. Atherosclerosis, the main pathological process behind most CVDs, often starts early and can remain asymptomatic for a long time before progressing to advanced stages [4, 5]. Atherosclerosis is an inflammatory vascular disease affecting large and medium-sized arteries, characterized by thickening, hardening, and loss of elasticity in the arterial walls. Carotid atherosclerosis (CAS), a form of carotid vascular lesion, represents the early stage of atherosclerosis [6]. Studies have confirmed that CAS not only contributes to cardiovascular diseases but is also closely linked to conditions such as psoriatic arthritis [7], renal impairment [8], hypertension [9], and fatty liver disease [10]. By 2020, about 27.6% of the global population, including an estimated 267.25 million Chinese adults aged 30 to 79, were affected by CAS, with its prevalence increasing annually due to population aging [3, 11]. Furthermore, a cohort study of occupational populations identified CADs as one of the four leading causes of death among workers [12].

Risk factors for CAS are multifaceted, encompassing lifestyle, genetic influences, and potential associations with harmful occupational exposures. Research indicates that prolonged workplace noise exposure may raise the risk of angina, coronary heart disease, and myocardial infarction by 2–3 times [13]. Compared to normal temperatures, exposure to high temperatures increases human morbidity and mortality [14]. Environmental epidemiology studies suggest that high heatwave exposure can raise CVD mortality risk by up to 1.65 times compared to low exposure [15]. The Corinthia study reported that mean carotid intima-media thickness (cIMT) of individuals in high CO (Carbon monoxide) exposure areas (1.14 ± 0.55 mm) was significantly greater than that in low-exposure areas (0.93 ± 0.24 mm), with carotid plaque burden nearly three times greater [16]. Furthermore, the Multi-Ethnic Study of Atherosclerosis showed that reducing particulate matter by 1 μg/m³ can slow cIMT progression by 2.8 μm/year [17]. China, the largest steel producer globally, employs a substantial workforce [18]. Steelworkers face various occupational hazards, such as heat, CO, dust, and noise, which may harm their health and accelerate disease progression. Although CAS risk factors have been extensively studied, research has mainly focused on lifestyle and genetics, with limited attention to occupational hazards. The relationship between occupational hazards and CAS remains an evolving area of research. Moreover, the mechanisms underlying how occupational hazards contribute to CAS remain poorly understood.

Apolipoprotein B (ApoB) is the primary protein component of low-density lipoprotein (LDL) particles. Elevated ApoB level indicates an increase in LDL particles, which penetrate the vascular endothelium, promote cholesterol deposition in arterial walls, and drive CAS development [19]. Elevated ApoB level is commonly associated with an increased risk of cIMT thickening and plaque formation [20, 21]. ApoB is recognized as an independent predictor of cardiovascular events. Elevated Elevated ApoB level reflects atherosclerosis progression and are strongly linked to cardiovascular events, such as myocardial infarction and stroke [22, 23]. While ApoB level changes are influenced by both genetic and environmental factors [24, 25], most studies have focused on daily living environments. Growing awareness of occupational hazards highlights their potential role as significant environmental contributors to elevated ApoB level. However, the combined effects of occupational hazards, ApoB, and CAS remain unexamined.

This study is based on the Beijing-Tianjin-Hebei occupational cohort and aims to evaluate the impact of occupational hazardous factors on the health of occupational populations. Baseline data from the steelworker cohort were used to examine the association between occupational hazards and CAS risk. Furthermore, the mediating role of ApoB in the connection between occupational hazards and CAS was examined.

Methods

Study population

Participants were drawn from the baseline survey of steelworkers in the Beijing-Tianjin-Hebei occupational cohort study, funded by the Ministry of Science and Technology of China. The steelworker

survey was conducted at the Tangsteel Company of HBIS Group in Tangshan, China. Participants were steelworkers who underwent occupational health examinations at Tangshan Hongci Hospital from January to September 2017. Data from 8,616 workers were collected, with 4,513 undergoing carotid ultrasound examinations. Participants were screened using the following exclusion criteria: (1) Non-permanent employees or those employed for less than one year. (2) Incomplete survey responses or missing laboratory test data. (3) Lack of occupational hazard monitoring data. The participant selection process is shown in Fig. 1, resulting in a final sample of 4,223 individuals.

This study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of North China University of Science and Technology (No. 15006). All participants provided written informed consent.

Questionnaire data

The survey was administered through one-on-one interviews conducted by research team members who had undergone standardized training. The questionnaire included questions on age, gender, education level, marital status, household size, monthly family income, smoking habits, alcohol consumption, dietary habits, physical activity, job type, work schedule changes, daily working hours, monthly rest time, and medical history.

Physical examination

Height and weight were measured with an infrared height-weight measurement device. Workers removed coats, shoes, phones, and other items that could affect the measurements, and readings were recorded after the device stabilized. Blood pressure was measured with the UNEX-TWIN fully automated electronic sphygmomanometer. Participants rested for 5 minutes in a quiet environment and removed clothing that could interfere with the measurement. Blood pressure was measured 3 times at intervals of at least 3 minutes, and the average was calculated.

Laboratory testing

Participants fasted for 12 hours before the test and visited the clinical laboratory at Tangshan Hongci Hospital the next morning for fasting blood collection. Blood samples were collected and delivered to specialist doctors for biochemical analysis.

Evaluation of carotid ultrasonography

Bilateral carotid ultrasound assessments were conducted by two experienced hospital physicians, blinded to the study's purpose and design, using a portable color Doppler ultrasound system with a 5–12 MHz probe frequency. Participants were positioned supine, and the examination began at the base of the neck, with the probe directed laterally opposite to the head's direction. This study adopted the 2017 Chinese Consensus on the Diagnosis of Head and Neck Arterial Atherosclerosis [26], as the diagnostic criteria for CAS, defining cIMT ≥ 1.0 mm as CAS.

Field hygienic investigation

The steel company's primary manufacturing process uses iron ore as raw material, progressing through sintering, blast furnace ironmaking, converter steelmaking, continuous casting/mold casting, and rolling. Field hygiene monitoring data, derived from the manufacturing processes of the steel company's main production units and the associated occupational hazards, included exposures to heat, noise, CO, and dust.

 Heat measurement was conducted using the LY-09 wet bulb globe temperature (WBGT) index meter at various work locations within the main production units of a steel company, following the national standard GBZ/T 189.7–2007, "Measurement of Physical Factors in the Workplace, Part 7: High Temperature." For each workplace, 4 to 6 measurement points were selected, with their layout determined according to specific worksite conditions. Each observation point was measured three times, and the average value was calculated. All instruments were calibrated before use and certified as qualified by the Provincial Bureau of Metrology.

 Noise measurements were performed using the AWA6228 multifunction sound level meter, in compliance with the national standards GBZ 2.2–2007 "Occupational Exposure Limits for Hazardous Factors Part 2: Physical Factors" and GBZ/T 189.8–2007 "Measurement of Physical Factors in the Workplace, Part 8: Noise." Measurements were taken at fixed points determined by the workplace's actual conditions. If the sound field in the work environment was relatively uniform (A-weighted sound level difference < 3 dB(A) within the measurement range), three measurement points were selected, and the average value was calculated. For uneven sound field distributions in the occupational environment, the measurement area was divided into distinct sound level zones, each with an A-weighted sound level difference of < 3 dB(A) within the same range. Two measurement points were then selected within each sound level zone, and the average value was calculated. For mobile work within the occupational environment, noise measurements were conducted at different work locations, and the equivalent sound level was calculated from the results. A record sheet was completed on-site for each measurement location. The formula for calculating the sound level is presented below:

$$L_{Aeq,T} = 10lg\left(\frac{1}{T}\sum_{i=1}^{n} T_i 10^{0.1L_{Aeq,T_i}}\right)$$

Where: $L_{Aeq,T}$ - Equivalent sound level for the entire day;

$L_{Aeq,Ti}$ - Equivalent sound level within the time period T_i;

T - The total duration of these time periods;

T_i - Duration of the time period i;

n - Total number of time periods.

 Industrial toxins include various harmful chemicals encountered by workers during production processes, with CO being the primary focus in this study. The API carbon monoxide analyzer was used following the national standard GBZ/T 160.28–2004, "Measurement of Toxic Substances in Workplace Air: Inorganic Carbon Compounds." Sampling points were established based on production processes, the number of production equipment, and the types of occupational hazards in the workplace. One sampling point was also positioned in each control room and worker rest area. All sampling points were located downwind of work locations according to actual site conditions and placed away from exhaust outlets. The sampling period was chosen to match the workers' normal working conditions and environment. Long-term sampling methods were used for all sampling points, and flow rates were recorded before and after sampling. The average flow rate was calculated to determine the 8-hour time-weighted average (TWA) concentration of CO in the workplace. All instruments were calibrated before use and certified compliant by the Provincial Bureau of Metrology. The sampler, used for full-day sampling, was operated in multiple intervals. Therefore, the 8-hour time-weighted average (8h-TWA) was calculated using the following formula:

$$TWA = \frac{C_1T_1 + C_2T_2 + \ldots + C_nT_n}{8}$$

Where: TWA – 8-hour time-weighted average concentration of CO in the work environment, mg/m^3;

C_1, C_2, C_n - Measured concentrations of CO in the work environment, mg/m^3;

T_1, T_2, T_n - The work hours of employees at the corresponding CO concentrations, h;

8 - The 8-hour time-weighted average permissible concentration.

 The IFC-2 explosion-proof dust sampler was used following the national standard GBZ/T 192.1–2007, "Measurement of Dust in Workplace Air, Part 1: Total Dust Concentration." Dust sampling points were chosen based on areas frequently accessed by workers in dust-affected environments. All instruments were calibrated before sampling and certified as compliant by the Provincial Bureau of Metrology. A 40 mm diameter polyvinyl chloride (PVC) fiber filter was used during sampling, with the sampler flow rate set to 40 L/min. Sampling at each point lasted 45 minutes, and a dust sampling record sheet was completed on-site. Dust concentration was calculated using the following formula after sampling:

$$\mathrm{C} = \frac{\mathrm{m_2} - \mathrm{m_1}}{\mathrm{Q} \times \mathrm{t} \times 1000}$$

Where: C - Dust concentration, mg/m^3;

m_1 - Filter mass before sampling, mg;

m_2 - Filter mass after sampling, mg;;

T - sampling time, min;

Q - sampling flow rate, 1/min

Data on changes in production processes and work-hour systems at the steel company were collected by reviewing historical archives and interviewing the occupational health department leadership. Workers' occupational histories were obtained through face-to-face interviews. The cumulative exposure measurement (CEM) of occupational hazards comprehensively reflects the duration and concentration of exposure to various hazards.Individual CEM was calculated using workers' occupational histories, including their exposure duration and concentration to various hazards.

$$\mathrm{CEM} = \mathrm{L_1T_1} + \mathrm{L_2T_2} \ldots + \mathrm{L_nT_n}$$

Where: L_n represents the average exposure level of the target hazard factor over a given period, T_n.

Definition and classification of covariates

Educational level: In this study, junior high school and below were classified as basic education; high school, vocational secondary, and technical school as intermediate education; and college, undergraduate, and graduate studies as advanced education.

Current smoking: Defined as smoking at least one cigarette per day for a minimum of six months, with smoking status categorized as current smoker or non-smoker.

Current drinking: Defined as consuming alcohol at least once per week for a minimum of six months, with drinking status categorized as current drinker or non-drinker.

Diet: Dietary patterns were evaluated using the Dietary Approaches to Stop Hypertension (DASH) diet [27], which focuses on eight food and nutrient categories. The intake of vegetables, fruits, whole grains, nuts and legumes, low-fat milk, sugary beverages, red meat, processed meat, and sodium was analyzed. A score of 5 indicates optimal intake, characterized by low consumption of sugary beverages, red and processed meats, and sodium, alongside high consumption of vegetables, fruits, whole grains, nuts and legumes, and low-fat milk. Participants were categorized by the median DASH score into two groups: ≤24 and >24.

Physical activity [28] : The Physical Activity Questionnaire (long version) was used to gather data on steelworkers' physical activity, covering daily work, commuting, daily activities, exercise and recreation, sedentary time, and sleep. The total metabolic equivalent (MET) for each worker was calculated based on activity frequency, and physical activity was classified as light, moderate, or vigorous according to scoring standards.In this study, sufficient physical activity was defined as moderate or vigorous activity.

Body mass index (BMI): BMI = weight (kg) / height (cm)².

Hypertension: Hypertension was defined based on the "2018 Revised Edition of the Chinese Guidelines for the Prevention and Treatment of Hypertension" [29], as a systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg, or a prior diagnosis of hypertension.

Dyslipidemia was defined based on the "Chinese Guidelines for the Prevention and Treatment of Dyslipidemia in Adults (2016 Revised Edition)" [30], as total cholesterol (TC) > 6.22 mmol/L, triglycerides (TG) ≥ 2.26 mmol/L, low-density lipoprotein cholesterol (LDL-C) ≥ 4.14 mmol/L, high-density lipoprotein cholesterol (HDL-C) < 1.04 mmol/L, or a prior diagnosis of dyslipidemia.

Diabetes: Diabetes was defined based on the "Chinese Guidelines for the Prevention and Treatment of Type 2 Diabetes (2022 Edition)" [31] as a fasting plasma glucose (FPG) ≥ 7.0 mmol/L or a prior diagnosis of diabetes.

Statistical method

Continuous data for participants' basic characteristics are presented as mean (standard deviation), and categorical data as frequency (percentage). Group differences were analyzed using t-tests or chi-square tests. Spearman correlation analysis was conducted to evaluate the correlation among various occupational hazardous factors. A restricted cubic spline regression model with four knots (5th, 35th, 75th, and 95th percentiles) was constructed to visualize the exposure-response relationships among occupational hazardous factors, ApoB, and CAS. The weighted quantile sum (WQS) method was applied to evaluate the joint effect of mixed occupational hazardous exposures on CAS, adjusting for covariates.

ApoB level was log-transformed prior to regression analysis. The regression analysis was conducted in three parts. In the first part, occupational hazardous factors were categorized into four quartile-based groups (Q1, Q2, Q3, Q4). A multivariable logistic regression model was applied to evaluate the association between occupational hazardous factors (as both continuous and categorical variables), ApoB, and the risk of CAS. Covariates included gender, age, marital status, monthly household income per capita, smoking habits, alcohol consumption, physical activity, diet, BMI, family history of CAS, FG, SBP, DBP, and serum creatinine (Cr). In the second part, a linear regression model was applied to evaluate the relationship between occupational hazardous factors (as both continuous and categorical variables) and ApoB. In the third part, occupational hazardous factors and ApoB were categorized into low and high concentrations based on the median (< median, ≥ median).The reference group comprised participants with low ApoB levels and low exposure to occupational hazardous factors. A multivariable logistic regression model was used to examine the association between low ApoB combined with high exposure to occupational hazardous factors, high ApoB combined with low exposure, and high ApoB combined with high exposure to occupational hazardous factors, and CAS. To further explore whether ApoB mediates the effect of occupational hazardous factors on CAS, a mediation analysis was conducted.

The sensitivity analysis examined the impact of occupational hazardous factors on carotid atherosclerosis across various demographic groups, including gender, smoking, alcohol consumption,

hypertension, diabetes, and dyslipidemia.

All data were analyzed using R 4.0.3 software, with statistical significance set at two-sided *P* values < 0.05.

Results

Baseline feature

Table 1 showed that this study included 4,223 steelworkers, of whom 1,113 (26.36%) had CAS.The average age of participants was 43.22 ± 7.91 years, comprising 3,858 males (91.36%) and 365 females (8.64%). Cumulative exposures to heat (599.70 ± 221.94 ℃/year), noise (1108.21 ± 809.89 dB(A)/year), CO (77.92 ± 33.96 mg/m^3/year), and dust (26.36 ± 18.60 mg/m^3/year) were significantly higher in the CAS group than in the non-CAS group (*P* < 0.05). Serum ApoB level (0.98 ± 0.21 g/L) was significantly higher in the CAS group than in the non-CAS group (*P* < 0.05). Significant differences were also observed between the CAS and non-CAS groups in gender, age, education level, income, smoking, alcohol consumption, BMI, FPG, SBP, DBP, LDL-C, TC, diabetes, hypertension, and dyslipidemia (*P* < 0.05).

Association between occupational environmental hazards and CAS

Table 2 showed that after adjusting for covariates, each 1-*SD* increase in cumulative exposure to heat, noise, CO, and dust was associated with *ORs* for CAS of 1.263 (1.146–1.391), 1.193 (1.105–1.288), 1.304 (1.195–1.423), and 1.178 (1.096–1.267), respectively. Participants in the highest quartile of cumulative exposure to heat (*OR*, 2.033; 95% *CI*, 1.538–2.686), noise (*OR*, 1.497; 95% *CI*, 1.201–1.864), CO (*OR*, 2.303; 95% *CI*, 1.766–3.004), and dust (*O*R, 1.376; 95% *CI*, 1.118–1.693) had a higher risk of CAS compared to those in the lowest quartile. Moreover, CAS incidence showed an increasing trend with higher cumulative exposure to heat, noise, CO, and dust ($P_{\text{for trend}}$ <0.05). Figure S1 show that WQS model identified CO and heat as the two occupational hazardous factors with the highest weights for CAS. Figure 2 show that A significant linear positive correlation was observed between cumulative exposure to heat and noise and CAS ($P_{\text{for nonlinear}}$ >0.05), whereas cumulative exposure to CO and dust exhibited a significant nonlinear positive correlation with CAS ($P_{\text{for nonlinear}}$ <0.05).

Relationship between occupational environmental harmful factors and ApoB

Table S1 showed that after adjusting for covariates, each 1-*SD* increase in cumulative exposure to heat, noise, CO, and dust was associated with ApoB increases of 0.109, 0.032, 0.079, and 0.014, respectively (*P* < 0.05). Participants in the highest quartile of cumulative exposure to heat, noise, CO, and dust showed ApoB increases of 0.228, 0.097, 0.203, and 0.077, respectively, compared to those in the lowest quartile (*P* < 0.05). Figure S1 showed that heat and CO were identified as the two occupational hazardous factors with the highest weights for ApoB. Figure S2 showed that significant nonlinear relationships were observed between cumulative exposure to heat, noise, CO, and dust and CAS ($P_{\text{for nonlinear}}$ <0.05).

The relationship between ApoB and CAS

Figure S3 showed that after adjusting for confounding factors, a significant linear relationship between ApoB and CAS was observed ($P_{\text{for nonlinear}}$ = 0.242). Table S2 showed that participants in the highest quartile of ApoB had a 118.3% higher risk of CAS (*OR*, 2.183; 95% *CI*, 1.719–2.772) compared to those in the lowest quartile. Furthermore, higher ApoB levels were significantly associated with an increased risk of CAS ($P_{\text{for trend}}$ <0.05).

The role of ApoB in the association between occupational environmental hazards and CAS

Given that occupational hazardous factors and ApoB are potential risk factors for CAS, we further examined their combined effect. Table 3 showed that, in most cases, a consistent and gradual increase in the combined effect of these two factors on CAS was observed. Compared to participants with low cumulative exposure to occupational hazardous factors (heat, noise, CO, and dust) and low serum ApoB, those with high cumulative exposure and high serum ApoB had 113.3%, 112.2%, 142.1%, and 99.7% higher risks of CAS, respectively (P< 0.05).

ApoB may also act as a mediator between occupational hazardous factors and CAS. Therefore, the mediating role of ApoB in the relationship between these factors was further analyzed. Table 4 showed that ApoB was found to mediate the relationship between cumulative exposure to heat, noise, CO, and dust and the risk of CAS, with mediation proportions of 48.92%, 19.70%, 30.00%, and 9.62%, respectively.

Sensitivity analysis

Tables S3-S6 showed that stratified analysis revealed that gender significantly modified the association between cumulative exposure to occupational hazardous factors (heat, noise, CO, and dust) and CAS risk, with stronger harmful effects observed in male workers. Table S3 showed that diabetes significantly influenced the association between cumulative exposure to heat and CAS risk, with stronger associations found in non-diabetic participants. Apart from these differences, tables S3-S6 showed that the association between cumulative exposure to occupational hazardous factors (heat, noise, CO, and dust) and CAS risk remained consistent across subgroups, with stable findings in the sensitivity analysis.

Discussion

This large-scale cross-sectional study of the steelworker population identified a positive correlation between cumulative exposure to occupational hazardous factors (heat, noise, dust, and CO) and CAS risk. ApoB is associated with CAS, and individuals with both high cumulative exposure to occupational hazardous factors and elevated ApoB levels have a higher CAS risk. Furthermore, ApoB partially mediates the relationship between occupational hazardous exposures and CAS. This study advances understanding of the complex relationship among occupational hazardous exposures, ApoB, and CAS, and sheds light on the potential mechanisms linking occupational exposures to CAS.

With rapid industrialization, occupational hazardous factors have emerged as a significant public health issue affecting workers' health. Certain workplaces expose workers to occupational hazards such as noise, thermal radiation, dust, heat, and CO, all of which negatively impact health [32-34]. A 10-year epidemiological study of construction workers reported that 22% of deaths during cooler seasons were attributed to cardiovascular diseases, increasing to 58% during hotter seasons. Increased mortality during hot periods is likely attributable to heat stress [35]. Our study confirms a strong association between long-term heat exposure and increased CAS risk. It provides additional evidence, highlighting the significance of occupational heat exposure in CAS. A review on noise impact summarized that long-term occupational noise exposure damages the auditory system and harms the cardiovascular system [36].Our study found that cumulative noise exposure significantly increased CAS risk. The UK timber workers cohort study reported that the high-exposure group had a 50% higher mortality rate from acute myocardial infarction compared to the low-exposure group [37]. Animal experiments suggest that noise-induced oxidative stress may be a primary biological mechanism underlying CVDs [38]. Our results indicate that workers with higher cumulative CO exposure face an increased risk of CAS. Although limited, evidence suggests that CO's strong binding with hemoglobin causes hypoxia, leading to myocardial cell changes and increased cardiovascular disease risk [39]. A review indicates that CO exposure, whether in household or occupational settings, increases the risk of vascular diseases [40]. A time-series analysis of 272 cities reported that each 1 mg/m³ increase in ambient CO concentration raised cardiovascular disease mortality by 1.12% (0.42-1.83) [41]. Previous studies have shown that occupational dust significantly harms cardiovascular health [42, 43], and our findings further support this conclusion. Mouse models suggest that long-term exposure to high dust concentrations impairs vascular relaxation, induces vascular wall inflammation, and exacerbates atherosclerosis [44]. Environmental epidemiology indicates that fine particulate matter and its components are significantly associated with increased cIMT [45]. To our knowledge, no studies have examined the link between occupational dust exposure and CAS in steelworkers. Our study addresses this gap.

Occupational hazardous factors are significant contributors to lipid metabolism abnormalities and have been linked to dyslipidemia in existing studies [46]. ApoB is a structural component of LDL and a ligand for the LDL receptor [47], closely associated with LDL and lipid metabolism. Vangelova et al. [48] reported that workers exposed to high temperatures had a 53.9% higher risk of developing high LDL-C compared to non-exposed workers. Studies indicate that exposure to hot environments rapidly increases cortisol and catecholamine secretion rates, which may indirectly influence LDL-C levels in the blood [49]. We hypothesize that physical activity under heat in steelworkers causes excessive sweating, increasing blood viscosity and inducing a hyperlipidemic state. Noise, as a social-psychological stressor, promotes cardiovascular diseases by elevating blood lipid levels [50]. We hypothesize that noise affects the endocrine system and activates the sympathetic nervous system, disrupting metabolism and hormonal regulation [51, 52]. The relationship between CO and ApoB remains unclear. A study in North Korea reported that high CO exposure affects cardiac autonomic function, with serum lipid levels as major effect modifiers [53]. A study on heating workers demonstrated that respirable coal dust causes cellular

damage, leading to lipid and protein oxidation, serum enzyme leakage, and imbalances in lipid profiles and antioxidant defense systems [54]. In a mouse model exposed to dust, visible dust deposits were observed in the lungs and liver, accompanied by a significant increase in very low-density lipoprotein (VLDL) and TC [55]. Our findings further deepen the understanding of the relationship between occupational hazardous factors and lipid metabolism abnormalities. Compared to traditional lipid indicators (such as cholesterol and HDL), ApoB, as the primary structural protein of atherosclerotic lipoproteins, more directly reflects atherosclerosis risk and exhibits abnormalities before lipid profile changes, enabling early detection of lipid metabolism imbalances caused by occupational factors [56-58].

Dyslipidemia is a well-established risk factor for CAS. Most prior studies have focused on the relationship between ApoB and CAS in the general population [59]. This occupational cohort study is the first to reveal the association between ApoB and CAS risk in steelworkers. Building on this, we further examined the combined and mediating effects of cumulative occupational hazardous exposures and ApoB on CAS. Higher CAS risk was closely associated with long-term high cumulative exposure to occupational hazardous factors and elevated serum ApoB levels. The study also revealed that ApoB partially mediates the relationship between occupational hazardous factors and CAS. Previous studies have reported that occupational hazardous factors influence lipid metabolism [48], consistent with our finding that these factors elevate ApoB levels. Epidemiological studies have further shown that CAS onset is often accompanied by elevated ApoB levels [60]. We hypothesize that ApoB may serve as a key biomarker in the mechanism linking occupational hazardous factors to CAS. The relatively high mediating proportion of ApoB provides preliminary evidence for its identification as a biomarker and offers insights for future interventions targeting CAS in steelworkers.

Our study has several strengths. First, our survey is part of a large-scale cohort study of occupational populations in the Beijing-Tianjin-Hebei region, funded by China’s Ministry of Science and Technology. We comprehensively assessed the relationship between steelworkers' exposure to various occupational hazardous factors and CAS, filling a gap in existing research. Second, we investigated the role of ApoB in CAS related to occupational hazardous factors, offering new insights into disease prevention and control among heavy industrial workers.

Our study also has several limitations. First, although our study is cohort-based, we only used baseline cross-sectional data, which limits our ability to establish causal relationships between disease and exposure. Second, we analyzed the relationship between occupational hazardous factors, ApoB, and CAS exclusively in the steelworker population, limiting the generalizability of the results. Further research in other occupational groups is needed to validate our findings. Lastly, despite adjusting for various potential confounders and performing stratified analyses, the possibility of residual or unmeasured confounding cannot be fully excluded.

Conclusion

Long-term high exposure to various occupational hazardous factors in steelworkers is associated with an increased risk of CAS, especially in those with elevated occupational exposure levels and serum ApoB concentrations. ApoB may partially mediate the relationship between occupational hazardous factors and CAS, suggesting its potential role in the mechanism of occupational exposure-induced atherosclerosis. Our findings underscore the need for stricter occupational hazardous factor control standards to reduce exposure levels, lower ApoB, and decrease CAS risk.

Declarations

Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of North China University of Science and Technology (No. 15006). All participants provided written informed consent.

Consent for publication

All the authors approved the manuscript for publication.

Competing interests

Not applicable.

Funding

This work was supported by the National Key Projects of Research and Development of China (No.2016YFC0900605).

Author Contribution

HY.C: conception; design of the work; the acquisition, analysis, and interpretation of data; the creation of new software used in the work; have drafted the work . JX.Y: conception ; the acquisition, analysis, and interpretation of data; have drafted the work or substantively revised it. All authors read and approved the final manuscript.

Acknowledgements

Thanks to all participants in this survey.

Data Availability

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Figures

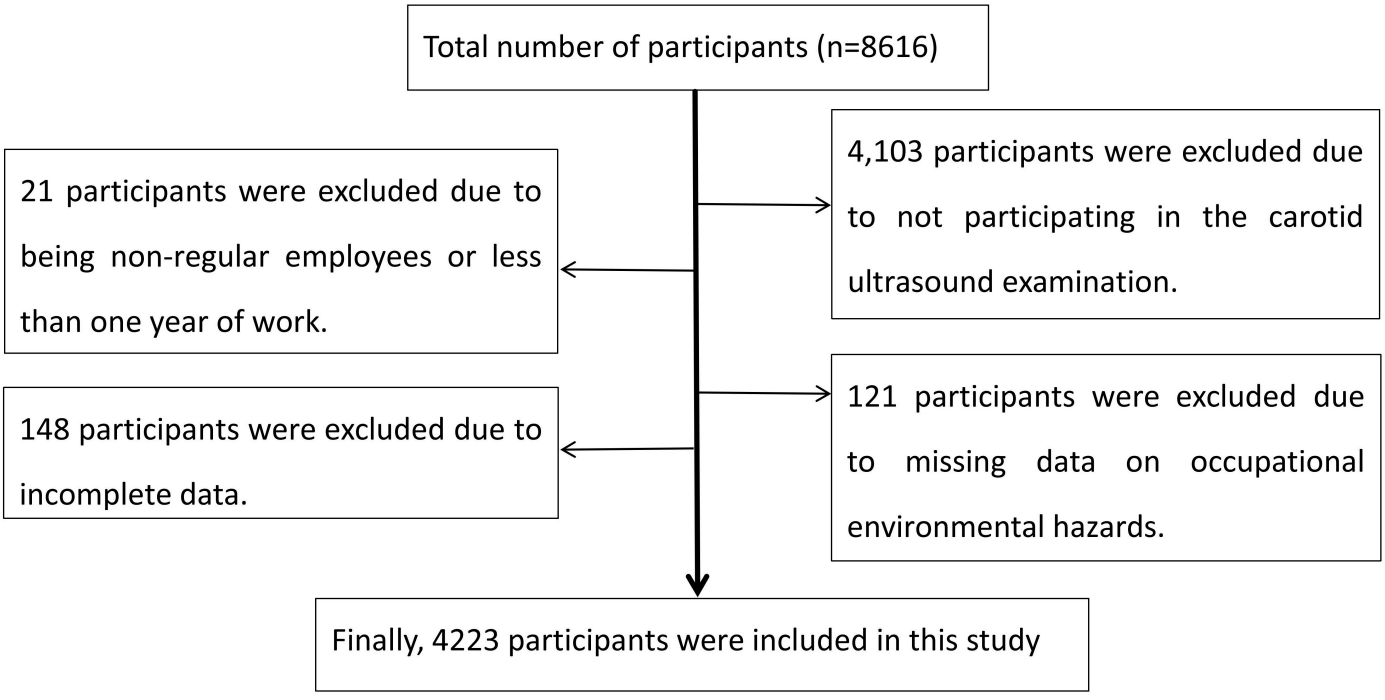


Figure 1 Screening process for participants

Figure 1

See image above for figure legend

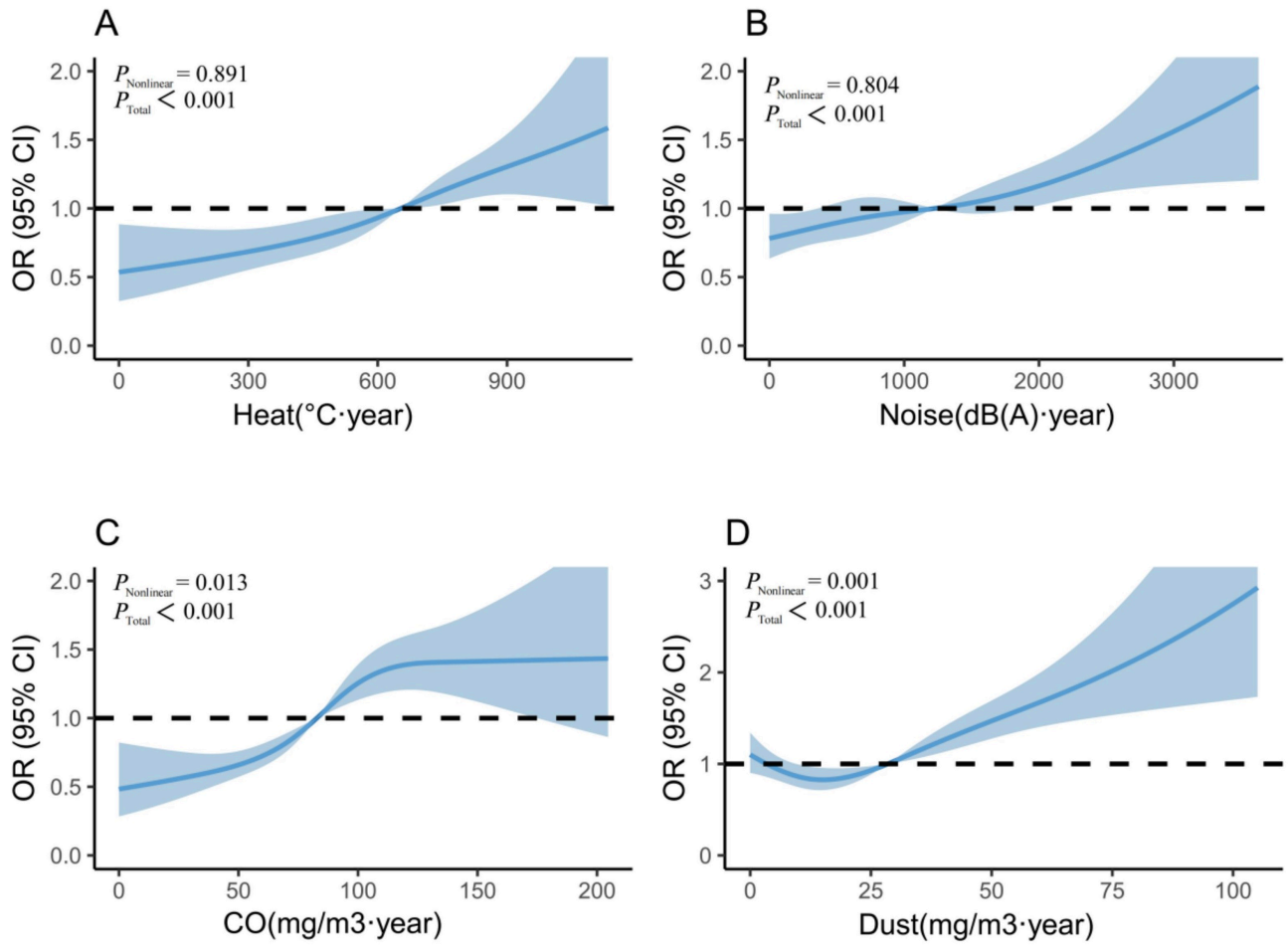


Figure 2 Dose-response relationship between harmful occupational environmental exposures and carotid atherosclerosis. Adjusted for gender, age, marital status, monthly household income per capita, current smoking, current drinking, physical activity, DASH grouping, BMI, family history of CAS, fasting glucose, systolic blood pressure, diastolic blood pressure, and serum creatinine

Figure 2

See image above for figure legend

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