Association between obese phenotypes and risk of carotid artery plaque among Chinese male railway drivers

Jia Pan1†, Zihang Wang2†, Chaohui Dong1, Bo Yang1, Lei Tang1, Peng Jia3,4, Shujuan Yang1,2,4* and Honglian Zeng1*

Abstract

Background  China has the world’s highest rail transportation network density, and the prevalence of obesity among railway workers in China is more than twice that of adults in the world. Carotid artery plaque (CAP) is a simple and noninvasive predictor of early atherosclerosis, while the association between different obese phenotypes and CAP risk among Chinese male railway drivers is unclear.

Methods  This cross-sectional study was performed among 8,645 Chinese male railway drivers. Obese phenotypes were assessed based on the obesity status (the body mass index ≥ 28 kg/m² as obesity vs. < 28 kg/m² as non-obesity) and metabolic status (metabolically healthy vs. metabolically unhealthy). Metabolically unhealthy was defined as the presence of at least one dysfunction, including elevated blood pressure, elevated fasting blood glucose, elevated triglyceride, and reduced high-density-lipoprotein cholesterol. Four obese phenotypes were defined based on the body mass index and metabolic status, i.e., metabolically healthy non-obesity (MHNO), metabolically healthy obesity (MHO), metabolically unhealthy obesity (MUO), and metabolically unhealthy non-obesity (MUNO). Multivariable logistic regression was employed to estimate the association between different obese phenotypes and the risk of CAP. Subgroup analysis was performed to examine the variation of the association by age, circadian rhythm disorders, and history of smoking and drinking.

Results  The prevalence of CAP among male railway drivers in MHO, MUO, MUNO, and MHNO was 8.75%, 18.67%, 17.82%, and 5.36%, respectively. Compared to those with MHNO, an increased risk for CAP was observed among those with MHO (OR = 2.18, 95% CI: 0.82, 5.10), MUO (OR = 1.78, 95% CI: 1.44, 2.21), and MUNO (OR = 2.20, 95% CI: 1.67, 2.89). The subgroup analysis showed that both of the metabolically unhealthy groups (MUNO and MUO) aged < 45 years were prone to a higher risk of CAP (for the MUNO group, OR = 4.27, 95% CI: 2.71, 7.10; for the MUO group, OR = 4.00, 95% CI: 2.26, 7.17).

Conclusion  The obese phenotypes are associated with CAP risk in male railway drivers, especially those with metabolically unhealthy conditions aged < 45 years.

Full list of author information is available at the end of the article.
Keywords: Metabolic abnormality, Obese phenotypes, Carotid artery plaque, Male railway drivers

**Introduction**

China has the world’s highest rail transportation network density [1]. The increasing traffic volume results in a heavy workload for railway drivers in China. Compared to workers in other occupations, railway drivers have longer working hours, less physical activity, extreme mental stress, poor sleep quality, and circadian rhythm disorders [1]. These occupational characteristics lead to a higher incidence of obesity [2, 3]. The prevalence of obesity among railway workers in China is more than twice that of adults globally [4, 5].

Obesity is a growing global public health issue [5], and one of the leading causes of cardiovascular disease (CVD) worldwide [6]. There is an increasing concern about the health and well-being of railway workers, as their safety is of utmost importance in maintaining the country’s transportation infrastructure. Therefore, reducing the number of obese workers in critical positions, such as train operators, is an urgent priority for the transportation industry. Obesity is considered a pivotal contributor to metabolic abnormalities [7]. It is associated with a constellation of metabolic abnormalities, including glucose abnormalities, high blood pressure, and high triglycerides, all of which are considered risk factors for CVD [8–10]. However, only 10–30% of obese individuals are reported to be metabolically healthy [11]. According to National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria [12, 13], four types of metabolic obese phenotypes have been well described, including metabolically healthy obesity (MHO), metabolically unhealthy obesity (MUO), metabolically healthy non-obesity (MUNO), and metabolically unhealthy non-obesity (MUNO). These metabolic obese phenotypes may be deemed more accurate predictors of CVD risk than obesity alone [14].

Carotid artery plaque (CAP), as detected by carotid ultrasound, is considered a simple and noninvasive predictor of early atherosclerosis and CVD [15, 16]. A cohort study indicated that CAP was associated with incident CVD events after adjustment for traditional risk factors [17]. Another study reported an association of different obese phenotypes with CAP events in a Chinese population [18]. Nevertheless, few studies reported the association between metabolic obese phenotypes and the risk of CAP in railway drivers with a high prevalence of unhealthy lifestyle behaviors.

In this cross-sectional study based on a large sample of male railway drivers in southwest China, we aim to investigate the association between obese phenotypes and CAP risk, as well as the modification effect of age, circadian rhythms, and history of drinking and smoking on their associations. The findings could contribute to a better understanding of the role of obese phenotypes in the development of CVD among male railway drivers, and identify the groups of participants that may be at high risk for CAP.

**Methods**

**Study design and participants**

This was a cross-sectional study recruiting 14,354 male railway drivers from the Chengdu Bureau of China Railway Administration, including 50 railway stations in Sichuan Province, Guizhou Province, and Chongqing. All the male railway workers ≥18 years old underwent physical examination in the Affiliated Hospital of Chengdu University between January and December 2019.

**Inclusion and exclusion criteria**

The on-job railway drivers who received physical examinations were included in this study. Exclusion criteria were participants with incomplete clinical information (e.g., blood pressure, fasting blood glucose, blood lipid, body mass index, etc.) and a history of severe diseases (e.g., renal or liver failure, and malignant). Finally, 8,645 male railway drivers were included in this survey.

All individuals voluntarily participated in this study, and their informed consent was obtained before the survey.

**Data collection and measurement**

**Definitions of metabolic status and body weight**

According to the NCEP ATP III criteria, metabolically unhealthy parameters were defined as follows [12]: (1) elevated blood pressure: systolic blood pressure (SBP) ≥130 mmHg or diastolic blood pressure (DBP) ≥85 mmHg or using antihypertensive medications; (2) elevated fasting blood glucose (FBG): FBG level ≥5.60 mmol/L or on antidiabetic treatment; (3) elevated triglyceride (TG): TG level ≥1.7 mmol/L or using lipid-lowering medications; and (4) reduced high-density lipoprotein cholesterol (HDL-C): HDL-C level <1.04 mmol/L, or using lipid-lowering medications. Obesity was defined as a body mass index (BMI) ≥28 kg/m² based on the criteria developed by the Working Group on Obesity in China [13].

According to these criteria, all the participants were classified into four groups [19]: (1) MHO was designated for those with BMI ≥28 kg/m² and none of the metabolically unhealthy parameters; (2) MUO represented those with BMI ≥28 kg/m² and one or more metabolically unhealthy parameters; (3) participants with BMI <28 kg/m² and none of the metabolically unhealthy...
parameters were denoted as MHNO; (4) participants with BMI < 28 kg/m² and one or more metabolically unhealthy parameters were denoted as MUNO.

Blood pressure (BP) was measured using electronic sphygmomanometers. BP was taken with the right upper arm kept at the level of the heart. After resting for 5 min, two measurements were taken at 1-min intervals, with the participants in a sitting position. If the difference between the two BP values was more than 10 mmHg, the measurement was recorded for the third time, and the final reading was the mean of the two closest measurements. Laboratory tests were conducted by laboratory physicians of Affiliated Hospital of Chengdu University per standard protocols. After overnight fasting of at least 8 h, venous blood was performed to measure FBG, TG, HDL-C, FBG, and other biochemical indicators.

**Definition of CAP**
A digital ultrasonic diagnostic system (EPIQ CX, Philips Ultrasound Inc., USA) was utilized to evaluate the presence/absence of CAP. The common carotid artery, the carotid artery bulb, and the internal carotid artery near and far wall segments were scanned bilaterally. The images were reviewed blindly by two physicians with more than five years of experience in vascular ultrasound imaging. According to the Mannheim criteria [20], CAP is defined as a focal region encroaching into the arterial lumen by at least 0.5 mm, > 50% of surrounding intima-media thickness values, or thickness ≥1.5 mm above the distance of the interface between the lumen-intima and the media-adventitia.

**Covariates**
Based on previously published studies [18, 21–23], indicators affecting the association between obese phenotype and CAP were considered covariates. In this respect, demographic characteristics (e.g., age), history of CVD (e.g., coronary atherosclerotic heart disease and myocardial infarction), lifestyle habits (e.g., smoking and alcohol drinking habits, and circadian rhythms), and some clinical biomarkers were collected by trained physicians and nurses to minimize bias. Circadian rhythm disorders were defined as working during the evening and overnight hours (6 P.M.–8 A.M) [24] and were self-reported by the participants. The working rhythms were also double-checked by the Social Security Department of Chengdu Railway Bureau. Trained investigators measured the body height and weight on standard methods. BMI was calculated as weight (in kilograms) divided by the square of height (in meters). Smokers were defined as those who smoked more than 1 cigarette/day for more than 1-year; other situations were considered nonsmokers. Alcohol drinkers were regarded as those drinking more than 1 time/week for over 6 months; other conditions were considered as nondrinkers. Some clinical biomarkers, such as serum uric acid (SUA), Total cholesterol (TC), and low-density lipoprotein cholesterol (LDL-C), were collected per standard protocols.

**Statistical analysis**
According to the literature review data, the prevalence of CAP in the general Chinese population was 20.15% [25]. Assuming 80% power, a 2-sided α error of 0.05, and the allowable error was 3%; finally, a sample size of 687 was obtained. Considering a dropout rate of 20%, we decided on a minimum total sample size of 825. Thus, the sample size of our study has reached this standard.

Categorical variables were expressed as numbers and percentages, and the chi-square test was used to analyze differences in categorical variables. If the numerical values were not normally distributed, they were described as median (interquartile range) and analyzed by a rank-sum test. Multiple regression models were employed to estimate the associations between obese phenotypes and CAP risk after adjusting all the covariates, including age, TC, LDL-C, SUA, current smoking, drinking, history of CVD, and circadian rhythms. Subgroup analysis was performed by age, circadian rhythms, and history of drinking and smoking to investigate their modification effect. Odds ratios (ORs) and their 95% confidence intervals (CIs) to obtain the effect estimates.

Sensitivity analysis was conducted in this study. Two different criteria of obesity to classify the obese phenotypes, based on Asia Pacific criteria [26] and WHO criteria [27], were used to estimate the robustness of the results.

All statistical analyses were conducted in R Studio (Version 4.0.5).

**Results**

**Baseline characteristics**
A total of 8,645 subjects were enrolled in the study. There was a significant difference in the baseline characteristics among different obese phenotypes, including age, medical history of diabetes, hypertension, hyperlipidemia, and CVD (P < 0.001). The prevalence of CAP was higher in the metabolically unhealthy groups (MUNO 17.82% and MUO 18.67%) compared to the metabolically healthy groups (MHNO 5.36% and MHO 8.75%). (Table 1).

**Association between CAP and metabolic obese phenotypes**
When compared with the MHNO group, the MUO group (2.20 [95%CI: 1.67, 2.89]) had a higher risk of CAP, followed by the MHO (2.18 [95%CI: 0.82, 5.10]) and MUNO (1.78 [95%CI: 1.44, 2.21]) groups after adjustment for age, TC, LDL-C, SUA, current smoking,
| Variable | Median (p25, p75) or percentage (%) | P value |
|----------|-------------------------------------|---------|
| **Sociodemographics** | | |
| Age, year | 44 (29.00, 48.00) | (25.00, 45.00) | (24.00, 43.00) | (38.00, 49.00) | (33.75, 48.00) | < 0.001 |
| **Age subgroup** | | | | | | < 0.001 |
| < 45 years | 4,716 (54.55) | 1,640 (72.66) | 61 (76.25) | 2,489 (46.44) | 526 (55.49) | |
| ≥ 45 years | 3,929 (45.45) | 617 (27.34) | 19 (23.75) | 2,871 (53.56) | 422 (44.51) | |
| **Medical history** | | | | | | |
| Diabetes | 404 (4.67) | 0 (0.00) | 0 (0.00) | 345 (6.44) | 59 (6.22) | < 0.001 |
| Hypertension | 1,071 (12.39) | 0 (0.00) | 0 (0.00) | 801 (14.94) | 270 (28.48) | < 0.001 |
| Hyperlipidemia | 316 (3.66) | 0 (0.00) | 0 (0.00) | 231 (4.30) | 85 (8.97) | < 0.001 |
| CVD | 76 (0.88) | 13 (0.58) | 0 (0.00) | 52 (0.97) | 11 (1.16) | < 0.001 |
| **Lifestyle behaviors** | | | | | | |
| Current smoker | 5,404 (62.51) | 1,257 (55.69) | 49 (61.25) | 3,474 (64.81) | 624 (65.82) | < 0.001 |
| Current drinker | 1,420 (16.41) | 194 (8.60) | 10 (12.25) | 1,039 (19.38) | 177 (18.67) | < 0.001 |
| Circadian rhythm disorders | 4,929 (57.02) | 1,334 (59.11) | 46 (57.50) | 2,975 (55.50) | 574 (60.55) | < 0.001 |
| **Clinical variables** | | | | | | |
| BMI, kg/m² | 24.04 (21.99, 26.26) | 22.03 (20.10, 23.95) | 29.37 (28.58, 30.30) | 24.13 (22.46, 25.79) | 29.46 (28.68, 30.74) | < 0.001 |
| 28 ≤ BMI < 30 kg/m² | 625 (7.23) | 0 (0.00) | 52 (65.00) | 0 (0.00) | 573 (60.44) | < 0.001 |
| BMI ≥ 30 kg/m² | 403 (4.66) | 0 (0.00) | 28 (35.00) | 0 (0.00) | 375 (39.56) | < 0.001 |
| SBP, mmHg | 124 (114.00, 134.00) | 114 (107.00, 120.00) | 118 (112.75, 124.25) | 128 (119.00, 137.00) | 132 (124.00, 140.00) | < 0.001 |
| DBP, mmHg | 81 (74.00, 89.00) | 73 (68.00, 78.00) | 75.5 (70.75, 80.00) | 85 (77.00, 91.00) | 88 (81.00, 95.00) | < 0.001 |
| TC, mmol/L | 4.65 (4.08, 5.24) | 4.27 (3.79, 4.80) | 4.43 (3.95, 4.91) | 4.77 (4.21, 5.38) | 4.87 (4.33, 5.48) | < 0.001 |
| TG, mmol/L | 1.59 (1.10, 2.38) | 1.06 (0.83, 1.30) | 1.31 (1.15, 1.52) | 1.32 (1.26, 1.65) | 2.3 (1.67, 3.22) | < 0.001 |
| HDL-C, mmol/L | 1.34 (1.17, 1.54) | 1.46 (1.31, 1.67) | 1.35 (1.23, 1.50) | 1.31 (1.13, 1.51) | 1.23 (1.06, 1.38) | < 0.001 |
| LDL-C, mmol/L | 2.89 (2.41, 3.38) | 2.53 (2.14, 2.97) | 2.78 (2.48, 3.16) | 2.98 (2.52, 3.48) | 3.15 (2.74, 3.61) | < 0.001 |
| FBG, mmol/L | 5.2 (4.86, 5.63) | 4.95 (4.68, 5.20) | 4.96 (4.80, 5.22) | 5.33 (4.95, 5.79) | 5.43 (5.04, 6.01) | < 0.001 |
| SUA, mmol/L | 387 (337.00, 447.00) | 367 (325.00, 419.00) | 438 (381.00, 488.00) | 389 (340.00, 447.00) | 434 (374.00, 496.25) | < 0.001 |
| **Metabolic risk components** | | | | | | |
| Elevated BP | 4,021 (46.51) | 0 (0.00) | 0 (0.00) | 3,334 (62.20) | 687 (72.47) | < 0.001 |
| Elevated FBG | 2,301 (26.62) | 0 (0.00) | 0 (0.00) | 1,894 (35.34) | 407 (42.93) | < 0.001 |
| Elevated TG | 3,969 (45.91) | 0 (0.00) | 0 (0.00) | 3,273 (61.06) | 696 (73.42) | < 0.001 |
| Reduced HDL-C | 1,016 (11.75) | 0 (0.00) | 0 (0.00) | 816 (15.22) | 200 (21.10) | < 0.001 |
| **Number of abnormal metabolisms** | | | | | | |
| 1 | 2,702 (31.26) | 0 (0.00) | 0 (0.00) | 2,451 (45.73) | 251 (26.48) | < 0.001 |
| 2 | 2,237 (25.88) | 0 (0.00) | 0 (0.00) | 1,867 (34.83) | 370 (39.03) | < 0.001 |
| 3 | 1,170 (13.53) | 0 (0.00) | 0 (0.00) | 899 (16.77) | 271 (28.59) | < 0.001 |
| 4 | 199 (2.30) | 0 (0.00) | 0 (0.00) | 143 (2.67) | 56 (5.91) | < 0.001 |
**Table 1 (continued)**

| Variable | Median (p25, p75) or percentage (%) | P value |
|----------|-------------------------------------|---------|
| **Outcome variables** | | |
| CAP | 1,260 (14.57) | 121 (5.36) | 7 (8.75) | 955 (17.82) | 177 (18.67) | <0.001 |
| BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; FBG, fasting blood glucose; SUA, serum uric acid; CAP: Carotid artery plaque; MHNO: metabolically healthy non-obesity; MUNO: metabolically unhealthy non-obesity; MUO: metabolically unhealthy obesity

**Table 2** Odds ratios and 95% confidence intervals for the risk of CAP across different obese phenotypes

| Variable | Crude model | P value | Adjusted model¹ | P value |
|----------|-------------|---------|-----------------|---------|
| MHNO | 1.00 (ref) | 1.00 (ref) | | |
| MHO | 1.69 (0.70, 3.51) | 0.2 | 2.18 (0.82, 5.10) | 0.09 |
| MUNO | 3.83 (3.16, 4.68) | <0.001 | 1.78 (1.44, 2.21) | <0.001 |
| MUO | 4.05 (3.17, 5.19) | <0.001 | 2.20 (1.67, 2.89) | <0.001 |

MHNO: metabolically healthy non-obesity; MHO: metabolically healthy obesity; MUNO: metabolically unhealthy non-obesity; MUO: metabolically unhealthy obesity. ¹ Adjustment for age, TC, LDL-C, SUA, current smoking, current drinking, history of CVD and circadian rhythms

**Table 3** Association between components of obese phenotypes and CAP risk

| Variable | Crude model | P value | Adjusted model¹ | P value |
|----------|-------------|---------|-----------------|---------|
| BMI | 1.07 (1.05, 1.09) | <0.002 | 1.04 (1.02, 1.06) | <0.001 |
| SBP | 1.05 (1.04, 1.05) | <0.001 | 1.03 (1.02, 1.03) | <0.001 |
| DBP | 1.06 (1.05, 1.07) | <0.001 | 1.04 (1.03, 1.04) | <0.001 |
| FBG | 1.29 (1.25, 1.34) | <0.001 | 1.14 (1.10, 1.18) | <0.001 |
| TG | 1.10 (1.07, 1.13) | <0.001 | 1.02 (0.99, 1.06) | 0.22 |
| HDL-C | 1.10 (0.91, 1.33) | 0.34 | 0.84 (0.68, 1.05) | 0.13 |

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; FBG, fasting blood glucose. ¹ Adjustment for age, TC, LDL-C, SUA, current smoking, current drinking, history of CVD and circadian rhythms

Current drinking, history of CVD, and circadian rhythms (Table 2). For the components of obese phenotypes, we observed that the BMI (1.04 [95%CI: 1.02, 1.06]), SBP (1.03 [95%CI: 1.02, 1.03]), DBP (1.04 [95%CI: 1.03, 1.04]) and FBG (1.14 [95%CI: 1.09, 1.18]) were significantly associated with CAP risk while no significant association was observed for TG and HDL-C (Table 3).

**Subgroup analysis**

The risk of CAP was increased in both MUNO and MUO groups compared with the MHNO group (P<0.01). Besides, those aged<45 years with metabolically unhealthy conditions (MUNO and MUO groups) showed a higher risk of CAP, with OR (95%CI) of 4.27 (95%CI: 2.71, 7.10) and 4.00 (95%CI: 2.26, 7.17), respectively (P for difference<0.05) (Fig. 1).

**Sensitivity analysis**

When using the Asia Pacific and WHO criteria for obesity, the results were robust (Table 4). The metabolically unhealthy groups (MUNO and MUO) also had the highest risk of CAP compared with the MHNO group.

**Discussion**

In this cross-sectional study based on male railway drivers, we found that the risk of CAP was higher in metabolically unhealthy groups (MUO and MUNO) than in metabolically healthy groups (MHO and MHNO) and notably higher in metabolically unhealthy groups aged<45 years. These findings could help to better understand the risk of CAP among male railway drivers and identify the groups of participants that need early health interventions.

Both metabolic abnormalities and obesity that can be expressed by obese phenotypes may exacerbate metabolic syndrome status and increase the risk of developing CAP [28]. In addition, we revealed that metabolically unhealthy groups were associated with a high risk of CAP, identical to previous studies on the general population. A retrospective cohort study with a sample size of 32,778 Chinese adults showed that different obese phenotypes were associated with the CAP risk [18]. However, the prevalence of CAP in male railway workers was significantly higher than that in previous studies (MHNO [5.36% vs. 1.1%], MHO [8.75% vs. 2.4%], MUNO [17.82% vs. 10.6%], MUO [18.67% vs. 6.3%], respectively) [29–31]. The differences may be explained by the high prevalence of unhealthy lifestyles and occupation-related characteristics among railway drivers (e.g. longer working hours, less physical activity, and circadian rhythm disorders). Previous studies have indicated that metabolically unhealthy patients had a higher risk of CVD [14, 32, 33]. In contrast, the CVD risk among metabolically healthy
individuals, such as MHO and MHNO groups, was controversial in previous studies [33, 34]. In this study, an association between metabolically healthy obese phenotypes and CAP risk among male railway drivers was found, and prior research involving a cohort of 3.5 million individuals reported a similar result [33]. Our study indicated that obese phenotypes could be used as a more precise classification of CAP risk in male railway workers.

The possible mechanism of the association varied among the obese phenotypes, and the risk of CAP can be explained by the following reasons. On the one hand, the common characteristics of obesity and metabolic abnormalities in lipid deposition in tissues lead to lipotoxicity, inflammation, and oxidative stress. All these factors can increase the risk of CAP [35, 36]. On the other hand, those with the metabolically healthy condition but obese phenotype were associated with lower levels of adiposity, which may explain their lower risk of carotid vascular endothelial injury than those with metabolic abnormalities. The available pieces of literature showed a varied association between obese phenotypes and the risk of CVD [37, 38].

Our stratified analysis demonstrated that age modified the association between obese phenotypes and CAP, and
those less than 45 years old had a high risk of CAP. The possible reason might be that drivers aged <45 years had higher rates of poor lifestyles (e.g., for the prevalence of circadian rhythm disorders, 61.2% vs. 52.1%) and prevalence of obesity (12.4% vs. 11.22%) than those ≥45 years. Although circadian rhythms were not found as modifiers, circadian rhythms were a striking occupational characteristic among railway drivers. Circadian rhythms can affect atherosclerosis plaques through a neuro-immune axis that links sleep to hematopoiesis and atherosclerosis [29, 30]. While we did not observe a modification effect of circadian rhythms on the association between CAP risk and obese phenotypes, the MUO group with circadian rhythm disorders still had a 2.53-to-2.68-fold risk of developing CAP compared with the MHNO group. Further studies are needed to investigate the role of circadian rhythm disorders on the risk of CAP in railway drivers.

It should be noted that there were some limitations in the study. Firstly, caution should be taken in making causal interpretations between CAP risk and obese phenotypes since this study was a cross-sectional design. Further prospective studies are warranted to obtain the incidence of CAP in this population. Secondly, due to the occupational characteristics of railway workers, high pressure, and irregular lifestyles, practically all employees were male workers, which limited the generalization of our findings. For this reason, future multicenter studies are required to include female employees and extend these findings. Thirdly, although the results have adjusted for several important confounding factors, there still have many unselected or unmeasured factors, such as socioeconomic variables (e.g., income level, education level, exercise habit, etc.), some clinical biomarkers (e.g., creatinine, c-reactive protein, homocysteine, etc.), and personal history of diseases (e.g., chronic kidney disease). Therefore, further studies on this population are greatly needed to collect these confounding factors.

Conclusion

This study is the first of its kind to investigate the association between obese phenotypes and CAP among male railway workers, and participants with MHO, MUNO, and MUO were associated with a high risk of CAP, especially in those with the metabolically unhealthy condition aged <45 years.

Acknowledgements

We are particularly grateful to the participants. We also thank all staff involved in this study for their painstaking efforts in conducting the data collection.

Authors’ contributions

H.Z. was responsible for study design and data collection. S.Y. was responsible for study design, data analysis, manuscript preparation, and revision. J.P. was responsible for study design, data analysis, manuscript preparation and revision, and obtaining funding. Z.W. was responsible for study design, data analysis, and manuscript preparation and revision. C.D., B.Y., and L.T. were responsible for data acquisition.

Funding

This study was supported by the Youth Fund of Chengdu University (2018ZX2B13) and the Research Fund of the Affiliated Hospital of Chengdu University (2020Y1Z42), and the Regional Innovation Cooperation Program of Science and Technology Commission Foundation of Sichuan Province (2021YFQ0031), the Chengdu Technological Innovation Research and Development Project (2021-YF05-0086-SN), the Sichuan University-Dazhou Cooperation Project (2020CDZ2-26).

Data availability

The datasets are available from the corresponding authors upon reasonable request.

 declarations

Ethics approval and consent to participate

The Ethical Committee of the Affiliated Hospital of Chengdu University approved this study (No. PI 2019-015-02). All individuals voluntarily participated in this study, obtaining their informed consent. This study was conducted per the Declaration of Helsinki.

Competing interests

The authors declare that they have no competing interests.

Consent for publication

Not applicable.

Author details

1Department of Health Management Center, Affiliated Hospital of Chengdu University, Chengdu, Sichuan, China
2West China School of Public Health and West China Fourth Hospital, Sichuan University, Chengdu, Sichuan, China
3School of Resource and Environmental Sciences, Wuhan University, Wuhan, China
4International Institute of Spatial Life Course Health (ISLE), Wuhan University, Wuhan, China
5Cooperation Project (2020CDDZ-26).

Received: 23 May 2022 / Accepted: 27 September 2022
Published online: 05 October 2022

References

1. Jiang Y, Wu C, Hu T, Chen M, Liu W, Zhou Y, Chen Z, Xu X. Association for combined exposure to job strain, shift work on mental health among Chinese railway workers: a cross-sectional study. BMU Open. 2020;10(10):e037544.
2. Li Y, Ma J, Yao K, Su W, Tan B, Wu X, Huang X, Li T, Yin Y, Tosi G, et al. Circadian rhythms and obesity: Timekeeping governs lipid metabolism. J Pineal Res. 2020;69(3):e12682.
3. Puttonen S, Harmà M, Hublin C. Shift work and cardiovascular disease—pathways from circadian stress to morbidity. Scand J Work Environ Health. 2010;36(2):96–108.
4. Wen X, Liu Xu D, X, Song S, Lu Y. Relationship between overweight/obesity and hypertension, hyperlipidemia and fatty liver among railway workers in Jinan. Chin J Prevent Control Chronic Dis. 2015;23:588–91.
5. Atfin A, Forouzanfar MH, Reitsma MB, Sur P, Estep K, Lee A, Marczak L, Mokdad AH, Moradi-Lakeh M, Naghavi M, et al. Health Effects of Overweight and Obesity in 195 Countries over 25 Years. N Engl J Med. 2017;377(1):13–27.
6. Virani SS, Alonso A, Benjamin EJ, Bittencourt MS, Callaway CW, Carson AP, Chamberlain AM, Chang AR, Cheng S, Delling FN, et al. Heart Disease and Stroke Statistics-2020 Update: A Report From the American Heart Association Circulation. 2020;141(8):e139–596.
7. Atrawi RT, Bunch KL, Toque HA, Caldwell RB, Caldwell RW. Mechanisms of obesity-induced metabolic and vascular dysfunctions. Front Biosci (Landmark Ed). 2019;24:890–934.
8. Son JS, Choi S, Lee G, Jeong S-M, Kim SM, Kim K, Yun YM, Park SM. Blood Pressure Change from Normal to 2017 ACC/AHA Defined Stage 1 Hypertension and Cardiovascular Risk. J Clin Med. 2018(6):820.
9. Musunuru K. Atherogenic dyslipidemia: cardiovascular risk and dietary intervention. Lipids. 2010;45(10):907–14.
10. Hoppin AG. Obesity and the liver: developmental perspectives. Semin Liver Dis. 2004;24(4):381–7.

11. Blüher M. Metabolically Healthy Obesity. Endocr Rev 2020, 41(3).

12. Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, Frucht JC, James WP, Loria CM, Smith SC. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention, National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. Circulation. 2009, 120(16):1640–65.

13. Mi YJ, Zhang B, Wang JW, Yan J, Han W, Zhao J, Liu DW, Tian QB. Prevalence and Secular Trends in Obesity Among Chinese Adults, 1991–2011. Am J Prev Med. 2015;49(5):661–9.

14. Sánchez-Villegas L, Navarro-González D, Fernández-Montero A, Pastorana-Delgado J, Martínez JA. Risk of incident ischemic stroke according to the metabolic health and obesity states in the Vascular-Metabolic CUN cohort. Int J Stroke. 2017;12(2):187–91.

15. Polak JF, O'Leary DH. Carotid Intima-Media Thickness as Surrogate for and Predictor of CVD. Global Heart. 2016;11(3):295–312.e293.

16. Polak JF, Pencina MJ, Pencina KM, O'Donnell CJ, Wolf PA, D'Agostino RB, Sr.: Carotid-wall intima-media thickness and cardiovascular events. N Engl J Med 2011, 365(3):213–221.

17. Brunner G, Virani SS, Sun W, Liu L, Dodge RC, Nambi V, Coresh J, Mosley TH, Sharrett AR, Boerwinkle E, et al. Associations Between Carotid Artery Plaque Burden, Plaque Characteristics, and Cardiovascular Events: The ARIC Carotid Magnetic Resonance Imaging Study. JAMA Cardiol. 2021;6(1):79–86.

18. Shen P, Zhou Y, Song A, Wan Y, Fan Z, Xu R. The association of metabolic health obesity with incidence of carotid artery plaque in Chinese adults. Nutr Metab Cardiovasc Dis. 2021;31(8):2376–81.

19. Lavié CJ, Laddu D, Arena R, Ortega BF, Alpert MA, Kushner RF. Healthy Weight and Obesity Prioritization. JACC Health Promotion Series. J Am Coll Cardiol. 2018;72(13):1506–11.

20. Touboul PJ, Hennerici MG, Meairs S, Adams H, Amarenco P, Bornstein N, Lavie CJ, Laddu D, Arena R, Ortega FB, Alpert MA, Kushner RF. Healthy Weight and Obesity Prevention: JACC Health Promotion Series. J Am Coll Cardiol. 2018;72(13):1506–31.

21. Bhatnagar A. Environmental Determinants of Cardiovascular Disease. Circ Res. 2011, 365(3):213–221.

22. Sharrett AR, Boerwinkle E, et al. Metabolically healthy but obese phenotype: cardiovascular prognosis. Frontiers in Pharmacology 2015, 6(71).

23. Ortega LF, Lavelle J, Lauweryns J, et al: Mannheim recommendations. Asia Pac J Clin Nutr. 2008;17(3):370–4.

Publisher's note
Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.