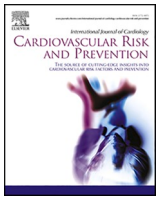
International Journal of Cardiology Cardiovascular Risk and Prevention 25 (2025) 200406

Contents lists available at ScienceDirect

International Journal of Cardiology Cardiovascular Risk and Prevention

journal homepage: www.journals.elsevier.com/international-journal-of-cardiology-cardiovascular-risk-and-prevention



Analysis of the association between long-term exposure to low-dose ionizing radiation and dyslipidemia and its components in medical radiologists: The mediating role of inflammatory markers

Changyong Wen [a,b], Xiaolian Liu [b], Yiqing Lian [b], Weizhen Guo [b], Lingyu Zhang [b], Yanting Chen [b], Xin Lan [b], Mingfang Li [b], Sufen Zhang [b], Weixu Huang [b], Jianming Zou [b], Huifeng Chen [a,b,*]

[a] School of Public Health, Guangzhou Medical University, Guangzhou, 511436, Guangdong, China
[b] Guangdong Province Hospital for Occupational Disease Prevention and Treatment, Guangzhou, 510300, Guangdong, China

ARTICLE INFO

Handling editor: D Levy

Keywords:
Medical radiologists
Low-dose ionizing radiation
Dyslipidemia
Triglyceride

ABSTRACT

Introduction: Our study aimed to explore the association between long-term exposure to low-dose ionizing radiation (LDIR) and dyslipidemia and its components among medical radiologists, and to identify the mediating role of inflammatory markers.

Methods: This cross-sectional study was conducted on 3918 medical radiologists, with data collected through questionnaires and occupational external exposure dosimeters. The multifactorial logistic regression and restricted cubic spline model were used to analyze the association between long-term exposure to LDIR and dyslipidemia and its components among medical radiologists, and mediation analysis was used to identify potential mediation effects.

Results: Of 3918 medical radiologists, 995 (25.4 %) had dyslipidemia. The gender, age, body mass index (BMI), and smoking status were influential factors for dyslipidemia of medical radiologists. After adjusting for confounders, the OR and 95 % CI for the occurrence of dyslipidemia and high TG in the highest tertile group (Q3) were 1.32 (95 % CI: 1.04, 1.67) and 1.51 (95 % CI: 1.11, 2.07), respectively. Restricted cubic spline model showed that the cumulative effective dose was linearly associated with both dyslipidemia and high TG, and the risk of dyslipidemia and high TG increased with the cumulative effective dose. Mediation analysis suggested that the inflammatory marker SII significantly mediated the association between cumulative effective dose and TG levels.

Conclusion: Our study shows that medical radiologists have a high detection rate of dyslipidemia, and the risk of dyslipidemia and high TG increases with increasing cumulative effective dose. Inflammatory marker SII may play a mediating role in the association between cumulative effective dose and TG levels.

Metabolic disease is a general term for a group of diseases caused by the accumulation or lack of nutrients due to abnormal metabolism of substances in the body, such as fats, sugars, proteins, purines, etc., mainly including hypertension, diabetes, and dyslipidemia, etc. [1]. Dyslipidemia is a common metabolic disease, primarily characterized by abnormal lipid levels, including elevated total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C), and lowered high-density lipoprotein cholesterol (HDL-C) [2]. Dyslipidemia is an important risk factor for the development of atherosclerotic cardiovascular disease and can increase the risk of cardiovascular disease and stroke mortality [3]. Data from the China Adult Nutrition and Chronic Disease Surveillance show that the prevalence of dyslipidemia in Chinese adults is 40.40 %, and its prevalence has been on the rise in the past few decades [4]. Recent research evidence suggests that in addition to the increasing of in the prevalence of dyslipidemia attributable to factors such as genetics, unhealthy diet, and lack of physical activity [5], long-term exposure to environmental or occupational low-dose ionizing radiation (LDIR) also plays an important role in the development of dyslipidemia [6,7].

LDIR refers to X or γ rays with an external exposure dose of less than

* Corresponding author. Guangdong Province Hospital for Occupational Disease Prevention and Treatment, Guangzhou, 510300, Guangdong, China.
E-mail address: hfchen2001@163.com (H. Chen).

https://doi.org/10.1016/j.ijcrp.2025.200406
Received 6 February 2025; Received in revised form 20 March 2025; Accepted 10 April 2025
Available online 12 April 2025

100 mGy or a dose rate of less than 0.1 mGy/min [8]. Compared with medium and high doses of ionizing radiation, LDIR rarely causes severe acute radiation damage effects in the short term, and its effects are characterized by complexity and uncertainty, so previous studies have focused more on the effects of medium and high doses of radiation [9]. With the wide application of radiation technology and the continuous development of medical care, the number of occupational personnel exposed to radiation work has increased year by year, and the effects of long-term exposure to LDIR on the long-term health of the populations are receiving more and more attention from scholars. Previous studies have shown that the health effects of long-term exposure to LDIR are mainly carcinogenic, such as leukemia [10], thyroid cancer [11], lung cancer [12], etc., and the focus of research in recent years has gradually shifted from cancer effects to chronic non-communicable diseases. A 40-year follow-up study of atomic bombing survivors found that radiation dose was significantly associated with the incidence of diabetes [13]. A 30-year cohort study of workers at the Mayak nuclear enterprise in Russia also found a significant positive linear correlation between the incidence of hypertension and the cumulative absorbed dose [14]. However, a study on the relationship between non-cancer incidence and occupational radiation exposure among Korean radiation workers did not find an association between radiation exposure and dyslipidemia [15]. Previous studies have focused more on the health effects of workers in nuclear enterprise and survivors of atomic bombings, but fewer studies have been conducted in relation to medical radiologists. In addition, it remains unclear whether long-term exposure to LDIR increases the risk of dyslipidemia.

In recent years, novel inflammatory markers such as white blood cells (WBC), neutrophils (NEU), neutrophil-lymphocyte ratio (NLR), and systemic immune-inflammatory index (SII) [16] have provided new ideas for monitoring inflammatory responses and reflecting inflammatory status in a variety of diseases, and can predict a wide range of physical and mental disorders, especially cardiovascular diseases [17, 18]. Studies have shown that multiple inflammatory factors significantly mediated the association between metabolism-related markers and dyslipidemia [19–21]. It is suggested that inflammation may be a predictor of dyslipidemia and play a mediating role in the development of dyslipidemia. There are few studies on the association between LDIR and dyslipidemia, and the mediating role of inflammation levels in this relationship remains unclear. Based on this, the aim of the present study is to analyze the potential dose-effect relationship between chronic LDIR exposure and dyslipidemia and its components among medical radiologists, and to explore the mediating role of inflammatory markers.

1. Study subjects and methods

1.1. Study subjects

A total of 4400 medical radiologists in Guangdong Province who participated occupational health checkups from January 2022 to December 2023 were randomly selected as survey subjects, excluding incomplete data from questionnaires (n = 258), missing radiation doses (n = 214), and missing results of blood indexes (n = 10), finally, 3918 medical radiologists were included. Fig. S1. Informed consent was obtained from the study subjects and approved by the Medical Ethics Committee of Guangdong Province Hospital for Occupational Disease Prevention and Treatment (Approval No. GDHOD MEC 2024030).

1.2. Questionnaires and quality control

A general questionnaire was designed according to the characteristics of medical radiologists to collect basic information on demographic characteristics, work characteristics, lifestyle, and dietary habit. Where body mass index (BMI) was defined as <24.00 kg/m^2 group and BMI ≥24.00 kg/m^2 group [22]. Smoking was defined as cumulative smoking for 6 months or more [23]; alcohol use was defined as drinking alcohol 3 times a week or more [24]; and physical activity was defined as exercising 2 times a week or more [25].

1.3. Estimation of cumulative effective dose

Dosimetric reconstruction was performed for each medical radiologist by collecting occupational external exposure dose data from 1980 to 2022. The cumulative effective dose of external occupational exposure of the medical radiologist included in this study was calculated as the sum of the annual effective dose. The cumulative effective dose was expressed as Dose:

$$Dose_i = \sum_{t=t_i^0}^{2022} Dose_{i,t}$$

Where $Dose_i$ is the sum of the annual effective dose to medical radiologists, Where t_i^0 is the onset of radiation exposure to medical radiologists, and $Dose_{i,t}$ is the annual effective dose to medical radiologists. Survey subjects wear personal dosimeters during work and monitored on a three monthly cycle. Strict operating procedures were followed for the issuance, wearing, transport, retrieval and storage of personal dosimeters to avoid exposure of personal dosimeters to artificial radiation during non-working hours.

1.4. Blood cell count and lipid level testing

Fasting peripheral venous blood was collected from the study subjects and counts of WBC, NEU, platelet, and lymphocyte were performed using a Beckman Coulter counter. The levels of TC, TG, and HDL-C in serum were detected using a fully automated biochemical analyzer. The level of LDL-C was calculated by Friedewald's formula: LDL-C = TC—HDL-C-—TG/2.2 (mmol/L).

1.5. Criteria for determining dyslipidemia

Referring to the Chinese Guidelines for Prevention and Control of Dyslipidemia in Adults [26], dyslipidemia was diagnosed if the study subjects met any of the following: (1) TC ≥ 6.22 mmol/L; (2) TG ≥ 2.26 mmol/L; (3) LDL-C ≥ 4.14 mmol/L; (4) HDL-C < 1.04 mmol/L; and (5) self-reported taking cholesterol-lowering drugs.

1.6. Calculation of inflammatory markers NLR and SII

NLR was calculated as neutrophil (NEU)count/lymphocyte count. SII was calculated as platelet count × neutrophil (NEU) count/lymphocyte count [27].

1.7. Statistical analysis

Data were entered using EpiData 3.1 software, and the measurement data were described using $\bar{x} \pm s$, and the comparison of count data rates was performed using the Pearson χ^2 test or trend χ^2 test. Due to the right-skewed distribution of the inflammation indicator data, the data will be log-transformed before subsequent statistical analysis. The cumulative effective dose was divided into 3 dose groups using tertiles (Q1 was the reference), and the influence factors of dyslipidemia were analyzed using multifactorial logistic regression. Logistic regression adjusted models were used to analyze the relationship between cumulative effective dose and dyslipidemia and its component; Model 1 was a crude model without adjustment for covariates; Model 2 was adjusted for gender and age; Model 3 was further adjusted based on Model 2 for marital status, education level, monthly income, BMI, occupation, shift work, type of work, cumulative effective dose, smoking, alcohol use, dietary habit, and physical activity. Considering the advantages of the restricted cubic spline model in fitting nonlinear associations between

independent and dependent variables, the model (4 nodes: 5th, 35th, 65th, and 95th percentile) was used to further analyze the non-linear dose-response relationship between the cumulative effective dose and dyslipidemia and its components. In this study, a sensitivity analysis was performed to analyze the association between the cumulative effective dose and the levels of the four components of dyslipidemia using a restricted cubic spline linear regression model, taking into account that dichotomizing the levels of dyslipidemia and its components may result in the loss of information about their original data as continuous variables. Based on the hypothesis that the cumulative effective dose mediates dyslipidemia through inflammatory stimuli, mediation analysis was performed using the "mediation" package in R software, using a quasi-Bayesian Monte Carlo method based on normal approximation and 1000 simulations [28]. Four inflammatory markers (WBC, NEU, NLR, SII) were set as mediators of inflammatory factors. All statistical analysis in this study were performed using R software, version 4.3.0, and a difference of $P < 0.05$ was considered statistically significant.

2. Results

2.1. Basic information

A total of 4400 questionnaires were distributed and 4142 valid questionnaires were recovered, with a valid recovery rate of 94.14 %, and a total of 3918 participants were finally included according to the exclusion criteria. Among them, 2324 (59.3 %) were males, 1594 (40.7 %) were females, the average age was 35.4 ± 8.1 years. Table 1.

2.2. Dyslipidemia in medical radiologists with different individual characteristics

The detection rate of dyslipidemia was 25.4 % (995/3918) among medical radiologists. Differences in the detection rate of dyslipidemia among medical radiologists of different gender, age, marital status, BMI, occupation, cumulative effective dose, smoking, alcohol use, and dietary habit were statistically significant ($P < 0.05$). Table 1.

2.3. Analysis of factors influencing dyslipidemia among medical radiologists

Multifactorial logistic regression analysis was performed with the presence of dyslipidemia as the dependent variable and all variables in Table 1 as independent variables. The risk of dyslipidemia was 2.735 (95 %CI: 2.199, 3.403) times higher in males than in females. The risk of dyslipidemia was 1.640 (95 %CI: 1.237, 2.174), 1.871 (95 %CI: 1.344, 2.605), and 3.170 (95 %CI: 2.058, 4.883) times higher in those aged 30 ~, 40 ~, and 50 ~, respectively, compared with those aged <30 years. The risk of dyslipidemia was 2.169 (95 %CI: 1.846, 2.549) times higher in the BMI ≥24 group than in the BMI <24 group. The risk of dyslipidemia in the 2.23–10.0 mSv group was 1.351 (95 %CI: 1.065, 1.715) times higher than that in the <0.72 mSv group. The risk of dyslipidemia was 1.319 (95 %CI: 1.055, 1.650) times higher in the smoking group than in the non-smoking group. Fig. 1.

2.4. Correlation analysis between cumulative effective dose and dyslipidemia and its components

In logistic regression-adjusted models, the cumulative effective dose was associated with an increase in the detection rate of dyslipidemia and TG levels after correcting for all confounders ($P < 0.05$). Further subgroup analyses showed a higher risk of dyslipidemia 1.35 (95 %CI: 1.07, 1.72) and high TG 1.50 (95 %CI: 1.10, 2.04) in the highest tertile (Q3) group, using the lowest tertile subgroup of cumulative effective dose (Q1) as a reference. Table 2.

Table 1
Dyslipidemia in medical radiologists with different individual characteristics.

Variables	Total	Dyslipidemia		χ^2 value	*P* value
	(N = 3918)	No(N = 2923)	Yes(N = 995)		
Gender				243.390	<0.001
Female	1594(40.7)	1398 (87.7)	196 (12.3)		
Male	2324(59.3)	1525 (65.6)	799 (34.4)		
Age, year				132.751	<0.001[b]
<30	1051(26.8)	904 (86.0)	147 (14.0)		
30 ~	1672(42.7)	1238 (74.0)	434 (26.0)		
40 ~	986(25.2)	662 (67.1)	324 (32.9)		
50 ~	209(5.3)	119 (56.9)	90 (43.1)		
Marital status				81.650	<0.001
Single[a]	1212(30.9)	1018 (84.0)	194 (16.0)		
Married	2706(69.1)	1905 (70.4)	801 (29.6)		
Education level				2.328	0.127
College and below	724(18.5)	524 (72.4)	200 (27.6)		
Bachelor's degree and above	3194(81.5)	2399 (75.1)	795 (24.9)		
Monthly income, yuan				3.610	0.164
<10000	2146(54.8)	1610 (75.0)	536 (25.0)		
10000 ~	1054(26.9)	797 (75.6)	257 (24.4)		
15000 ~	718(18.3)	516 (71.9)	202 (28.1)		
BMI, kg/m^2				255.943	<0.001
<24	2488(63.5)	2066 (83.0 %)	422 (17.0 %)		
≥24	1430(36.5)	857 (59.9 %)	573 (40.1 %)		
Occupation				45.394	<0.001
Nurse	692(17.7)	570 (82.4)	122 (17.6)		
Doctor	1958(50.0)	1376 (70.3)	582 (29.7)		
Technician	1268(32.3)	977 (77.1)	291 (22.9)		
Shift work				0.015	0.902
No	1814(46.3)	1355 (74.7)	459 (25.3)		
Yes	2104(53.7)	1568 (74.5)	536 (25.5)		
Type of work				2.262	0.520
Diagnostic radiology	2263(57.8)	1680 (74.2)	583 (25.8)		
Nuclear medicine	192(4.9)	150 (78.1)	42 (21.9)		
Radiotherapy	599(15.3)	455 (76.0)	144 (24.0)		
Interventional radiology	864(22.0)	638 (73.8)	226 (26.2)		
Cumulative radiation dose, mSv				94.905	<0.001[b]
<0.72	1420(36.2)	1156 (81.4)	264 (18.6)		
0.72–2.22	1485(37.9)	1122 (75.6)	363 (24.4)		
2.23–10.0	1013(25.9)	645 (63.7)	368 (36.3)		
Smoking				84.290	<0.001

(continued on next page)

Table 1 (*continued*)

Variables	Total	Dyslipidemia		χ^2 value	*P* value
	(N = 3918)	No(N = 2923)	Yes(N = 995)		
No	3456(88.2)	2659 (76.9)	797 (23.1)		
Yes	462(11.8)	264 (57.1)	198 (42.9)		
Alcohol use				26.275	<0.001
No	3651(93.2)	2759 (75.6)	892 (24.4)		
Yes	267(6.8)	164 (61.4)	103 (38.6)		
Dietary habit				13.876	0.001[b]
Vegetable	303(7.7)	239 (78.9)	64 (21.1)		
Balanced diet	2633(67.2)	1993 (75.7)	640 (24.3)		
Meat	982(25.1)	691 (70.4)	291 (29.6)		
Physical activity				1.655	0.198
No	941(24.0)	717 (76.2)	224 (23.8)		
Yes	2977(76.0)	2206 (74.1)	771 (25.9)		

Notes.
[a] Single (unmarried, divorced, widowhood).
[b] Trend chi-square test.

2.5. Dose-response relationship between cumulative effective dose and dyslipidemia and its components

Restricted cubic spline model analysis showed that after adjusting for all confounders, the cumulative effective dose was linearly correlated with both dyslipidemia and high TG (P_1 for non-linearity = 0.398, P_2 for non-linearity = 0.667), and the risk of dyslipidemia and high TG increased with the cumulative effective dose. Fig. 2.

2.6. Sensitivity analysis

Sensitivity analysis demonstrated the robustness of the results. First, dyslipidemia was redefined according to the criteria for determination of dyslipidemia by the U.S. National Cholesterol Education Program (Adult Treatment Panel III) (Table S1). Second, dyslipidemia and its component levels were converted from dichotomous to continuous variables. Fig. 3.

2.7. Mediation analysis

In this study, the mediating role of LDIR-mediated inflammatory markers was estimated based on the hypothesis that LDIR causes dyslipidemia through inflammatory stimuli. The results showed that no potential mediating role of inflammatory markers (WBC, NEU, NLR, SII) was found between long-term exposure to LDIR and dyslipidemia as well as TC, HDL-C and LDL-C levels. However, the association between cumulative effective dose and TG levels was observed to be mediated by the inflammatory marker SII (*P* values for all mediators were less than 0.05), with a mediating effect of 15.4 %. Fig. 4 and Table S2.

3. Discussion

In this study, we found that the detection rate of dyslipidemia among medical radiologists in Guangdong Province, China, was 25.4 %, which was higher than the detection rate of dyslipidemia among radiation workers in South Korea, which was 10.6 %, as reported by Park [15] et al. It may be related to differences in sample size, occupational category, lifestyle, dietary habit, and geographical characteristics. The results of multifactorial logistic regression analysis found that the risk of dyslipidemia was higher in males than in females, which was consistent with the findings of Xi [29] et al. on dyslipidemia in adults over 35 years of age in northern China. This may be due to gender differences in lifestyle and dietary habit and related to the fact that female estrogen promotes the degradation and metabolism of blood lipids [30].The present study found that the risk of dyslipidemia increased with age, which is consistent with the findings of Moosazadeh [31] et al. on age-related lipid levels in a population of males aged 35–70 years. With the increase of age, the function of various organs of the body decreases gradually, the metabolic level also gradually decreased, and the composition and proportion of lipids in the body changes [32], especially when the amount of physical activity is decreased, it is more likely to cause dyslipidemia. The risk of dyslipidemia was higher in smokers than in non-smokers in this study, which is consistent with the results of a study on the effect of smoking on blood lipids in Korean males reported by Kim [33] et al. Smoking is a risk factor for dyslipidemia, and nicotine produced by tobacco combustion can influence lipid metabolism [34], leading to increased levels of fatty acids and cholesterol in the blood [35]. This study also found that a higher BMI was associated with a higher risk of dyslipidemia. This is consistent with the findings of Chen et al. [36] who analyzed the relationship between BMI and dyslipidemia. This may be due to the fact that radiation workers with a higher BMI consumed more fats, sugars, and meats, and less fresh fruits and vegetables containing high levels of phytochemicals (carotenoids, isoflavones), vitamins, and dietary fiber that are beneficial in lowering blood lipid levels [37,38].

This study found that the cumulative effective dose was positively associated with the risk of dyslipidemia and high TG, and this association persisted after adjustment for confounders. Further analysis using a restricted cubic spline model showed that the cumulative effective dose was linearly and positively associated with dyslipidemia and high TG, and the risk of dyslipidemia and high TG increased with increasing cumulative effective dose. This is consistent with the findings of Nakajima S [39] et al. on dyslipidemia in residents of the nuclear power plant accident in Japan. This may be related to the following mechanisms: first, LDIR increases oxidative stress in vivo, leading to lipid peroxidation, and TG is more sensitive to oxidative stress compared to TC, LDL-C, HDL-C, and is therefore more susceptible to the effect; second, LDIR may trigger inflammation, and inflammatory factors, such as TNF-α and IL-6, promote TG synthesis but have a weaker effect on TC, LDL-C, and HDL-C; finally, LDIR may alter the expression of genes related to TG metabolism but have a lesser effect on genes related to TC, LDL-C, and HDL-C metabolism [40]. However, this study is inconsistent with the findings of Park [15] et al. who reported that occupational radiation exposure of radiation workers in Korea is not associated with dyslipidemia. It may be related to different criteria for determining dyslipidemia and sample size. In addition, it may also be related to the different types of rays to which the subjects were exposed. The subjects in the Korean study were mainly nuclear power plant workers exposed to neutrons, and α, β, and γ rays, whereas the subjects in the present study were medical radiation workers exposed mainly to X or γ rays.

The inflammatory response is a natural defense reaction of the body and is essential for the maintenance of physiological homeostasis [41]. However, if the inflammatory response is not properly controlled and persists over a long period of time, it may lead to chronic inflammation, which is potentially harmful to the body and can lead to serious illness such as cardiovascular and cerebrovascular diseases [42]. Inflammatory factors have been found to be associated with an increased risk of dyslipidemia and can be used as predictors of dyslipidemia [43]. Therefore, early identification of the potential role of inflammatory factors in the pathogenesis of metabolic disorders, including dyslipidemia, is important [44]. It has been found that, compared with traditional inflammation indicators such as CRP and IL-6, complex inflammatory indices based on blood cell counts such as NLR, PLR, and systemic immunoinflammatory indices (SII, SIRI), especially SII and SIRI indices containing

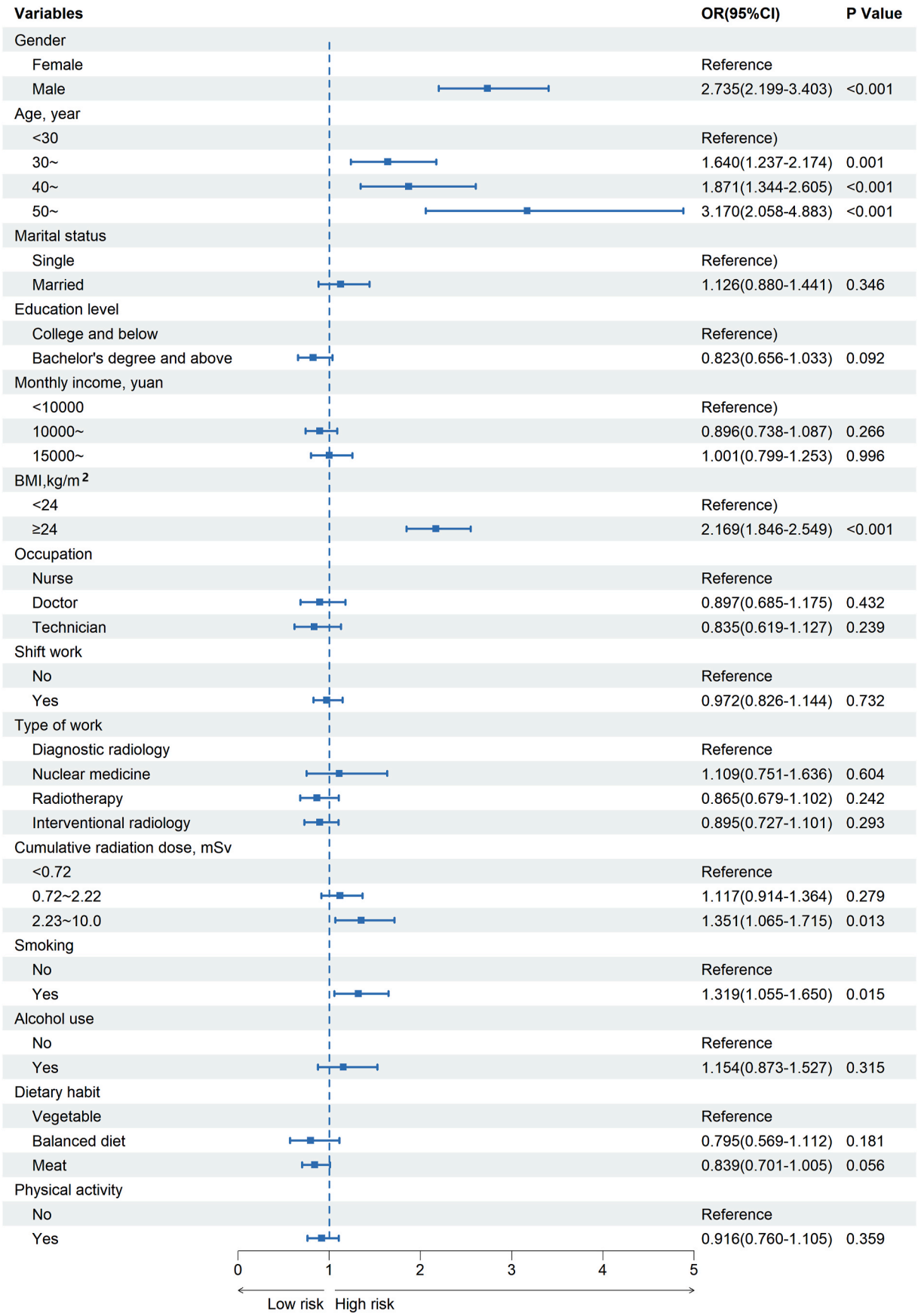


Fig. 1. Forest plot of multifactorial logistic regression analysis of dyslipidemia
Notes: Female, age <30, single, College and below, monthly income<1000, BMI<24, nurse, no shift work, diagnostic radiology, mSv<0.72, no smoking, no alcohol use, vegetable, no physical activity as reference group.

Table 2
Correlation analysis between cumulative effective dose and levels of dyslipidemia and its components.

Variables	Model 1[a]		Model 2[b]		Model 3[c]	
	OR (95 %CI)	*P* value	OR (95 %CI)	*P* value	OR (95 %CI)	*P* value
Dyslipidemia						
Q1	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Q2	1.42 (1.19–1.69)	0.001	1.11(0.92–1.35)	0.281	1.12 (0.91–1.36)	0.279
Q3	2.50 (2.08–3.01)	0.001	1.31(1.05–1.63)	0.018	1.35 (1.07–1.72)	0.013
TC						
Q1	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Q2	1.19(0.88–1.62)	0.252	0.99(0.72–1.36)	0.958	1.00(0.73–1.39)	0.989
Q3	2.01(1.49–2.72)	0.001	1.10(0.78–1.56)	0.588	1.05(0.73–1.52)	0.797
TG						
Q1	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Q2	1.26(0.98–1.62)	0.072	0.96(0.74–1.25)	0.756	0.97(0.74–1.27)	0.826
Q3	2.59(2.04–3.30)	0.001	1.44(1.09–1.92)	0.011	1.50(1.10–2.04)	0.010
HDL-C						
Q1	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Q2	1.56(1.19–2.04)	0.001	1.25(0.94–1.66)	0.132	1.23(0.92–1.66)	0.169
Q3	1.76(1.32–2.34)	0.001	1.12(0.80–1.56)	0.525	1.14(0.80–1.65)	0.468
LDL-C						
Q1	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Q2	1.25(0.94–1.65)	0.127	1.06(0.79–1.42)	0.703	1.07(0.79–1.45)	0.663
Q3	1.92(1.45–2.56)	0.001	1.16(0.83–1.61)	0.398	1.14(0.80–1.63)	0.479

Notes.
[a] Model 1 was a crude model without adjustment for covariates.
[b] Model 2 was adjusted for gender, age.
[c] Model 3 was further adjusted for marital status, education level, monthly income, BMI, occupation, shift work, type of work, smoking, alcohol use, dietary habit, and physical activity based on Model 2.

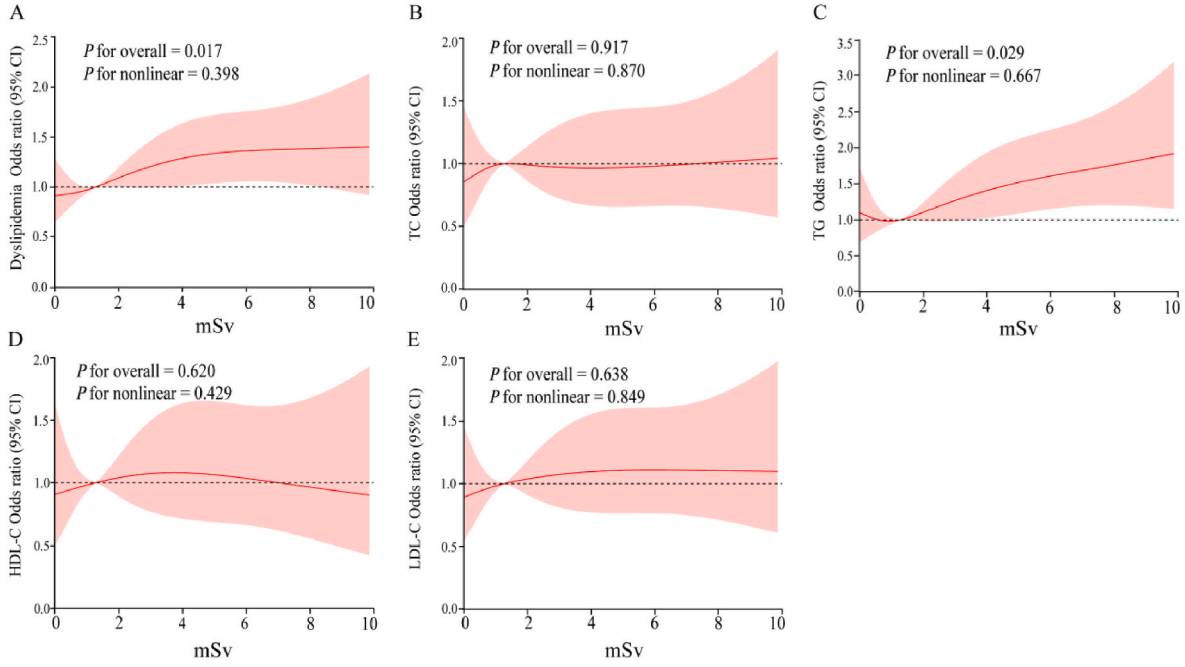


Fig. 2. Restricted cubic spline logistic regression modeling between cumulative effective dose and dyslipidemia (A) and its components TC (B), TG (C), HDL-C (D), LDL-C (E).
Note: Adjusted for gender, age, marital status, education level, monthly income, BMI, occupation, shift work, type of work, smoking, alcohol use, dietary habit, and physical activity.
TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; OR, odds ratio; CI, confidence interval.

three levels of inflammatory cells, can effectively and comprehensively reflect the complex inflammatory status in the organism [45,46], and better predict certain inflammation-related diseases [47]. A population-based study based on NHANES found a significant positive correlation of 1.03 (95 %CI, 1.01, 1.05) between systemic immune-inflammatory index SII and dyslipidemia [48]. Interestingly, the present study found that SII plays a mediating role between LDIR and TG association, which provides a potential new perspective on the association between long-term exposure to LDIR and dyslipidemia. This is consistent with the findings of Tapio S et al. on inflammation, oxidative stress-mediated adverse effects of low and moderate doses of ionizing radiation and metabolic syndrome [49]. Although the mechanisms by which LDIR affects dyslipidemia have not been fully elucidated, several possible pathways may exist based on in vitro and animal studies. First,

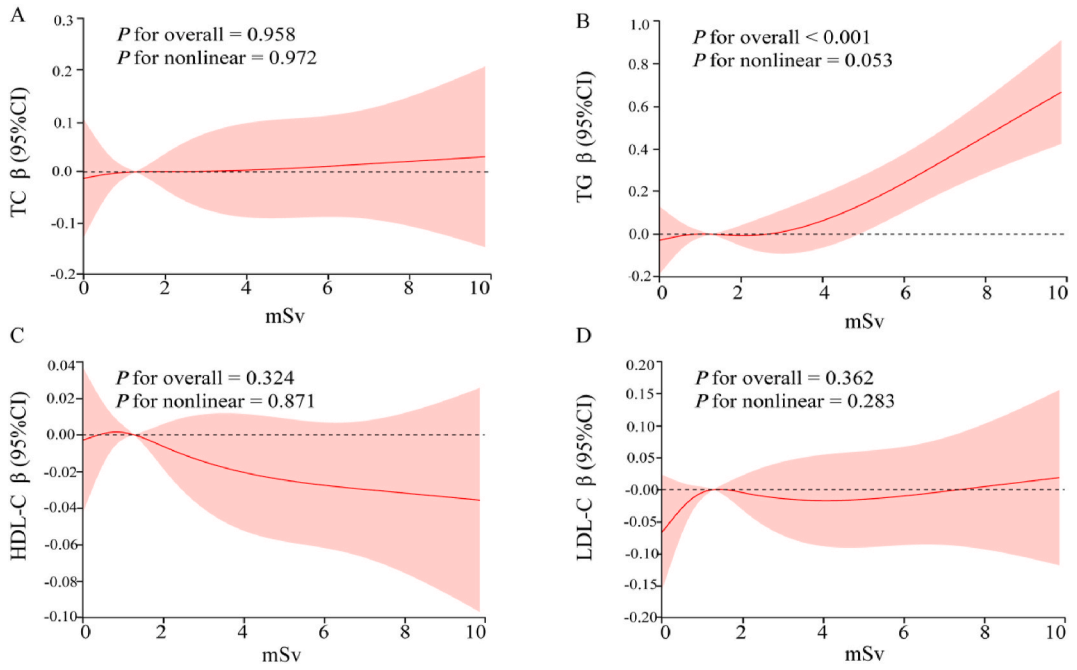


Fig. 3. Restricted cubic spline linear regression model between cumulative effective dose and TC (A), TG (B), HDL-C (C), LDL-C (D).
Note: Adjusted for gender, age, marital status, education level, monthly income, BMI, occupation, shift work, type of work, smoking, alcohol use, dietary habit, and physical activity.
TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; OR, odds ratio; CI, confidence interval.

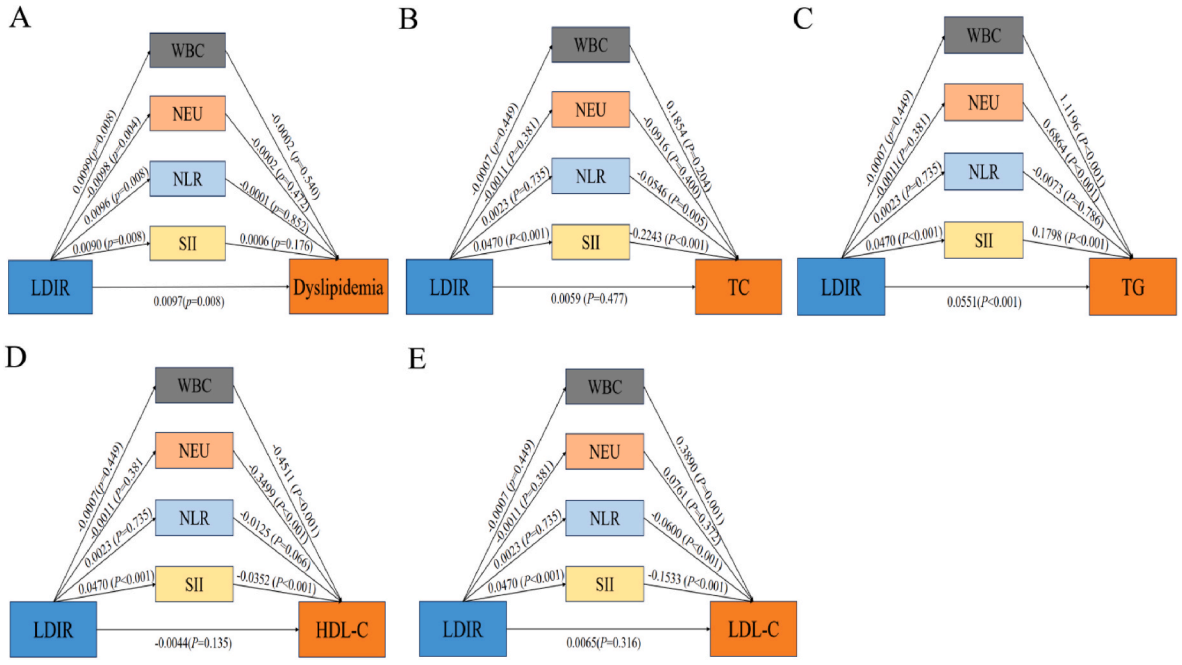


Fig. 4. Mediating role of inflammatory markers in the association of LDIR with dyslipidemia (A) and its components TC (B), TG (C), HDL-C (D), LDL-C (E).
Note: Adjusted for gender, age, marital status, education level, monthly income, BMI, occupation, shift work, type of work, smoking, alcohol use, dietary habit, and physical activity.
TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

LDIR induces an immune-inflammatory response, triggering an inflammatory response through activation of immune cells, such asTNF-α, IL-6, and the systemic immune-inflammatory index, SII, which in turn affects the expression of lipid functions [50]. Second, due to chronic exposure to LDIR, the body produces large amounts of ROS, which causes damage to macromolecules such as proteins, lipids, and DNA. Damage to unrepaired or improperly repaired cellular macromolecules is mainly manifested at the cellular and tissue level, such as cellular senescence, apoptosis, and inflammation, leading to persistent oxidative stress, which in turn triggers metabolic diseases [49].

This study has the following strengths. First, two sensitivity analysis were used and demonstrated the robustness of the results. Second, the

association between long-term exposure to LDIR and dyslipidemia and its components was found with large sample data, providing a reliable basis for radiation epidemiological studies of long-term exposure to LDIR and health. Third, the true external exposure dose can directly and accurately reflect the association between long-term exposure to LDIR and dyslipidemia by measuring personal dose equivalent of occupational external exposure through a personal dosimeter worn by medical radiologists. Finally, we explored the potential mediating role of inflammatory markers in the association between long-term exposure to LDIR and dyslipidemia and its components, and found that the inflammatory marker SII mediated the association between long-term exposure to LDIR and high TG levels, which provides new clues for mechanistic studies of the association between long-term exposure to LDIR and dyslipidemia. This study also has certain limitations. First, only the personal dose of external radiation was obtained in this study, but not the dose of internal radiation. In the future, the association between long-term exposure to LDIR and dyslipidemia may be considered by combining the internal and external dose. Second, the lowest tertile of cumulative effective dose was selected as the control group in our study, which may not be fully representative of the unexposed group, and future studies should consider non-radiation-exposed- healthcare workers as a control group. In conclusion, as the present study was only a cross-sectional study, it was not possible to infer a causal relationship between long-term exposure to LDIR and dyslipidemia, and it is recommended that future prospective cohort studies be conducted to provide important evidence of a causal relationship between long-term exposure to LDIR and dyslipidemia among medical radiologists.

4. Conclusion

The detection rate of dyslipidemia among medical radiologists was high. Gender, age, BMI, cumulative effective dose, and smoking were the factors influencing dyslipidemia. The risk of dyslipidemia and high TG increased with increasing cumulative effective dose. The inflammatory marker SII may mediate the relationship between long-term exposure to LDIR and TG levels. Future large-scale population-based radiation epidemiologic studies are urgently needed to confirm our findings and to elucidate the potential association between long-term exposure to LDIR and dyslipidemia in medical radiologists.

CRediT authorship contribution statement

Changyong Wen: Writing – original draft, Methodology, Formal analysis, Data curation, Conceptualization. **Xiaolian Liu:** Methodology, Data curation, Conceptualization. **Yiqing Lian:** Methodology, Data curation. **Weizhen Guo:** Methodology, Data curation. **Lingyu Zhang:** Methodology, Data curation. **Yanting Chen:** Methodology, Data curation. **Xin Lan:** Methodology, Data curation. **Mingfang Li:** Methodology, Data curation. **Sufen Zhang:** Methodology, Data curation. **Weixu Huang:** Methodology, Data curation. **Jianming Zou:** Resources, Methodology, Formal analysis, Conceptualization. **Huifeng Chen:** Writing – review & editing, Supervision, Resources, Methodology, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Ethics approval

This study was approved by the Medical Ethics Committee of Guangdong Province Hospital for Occupational Disease Prevention and Treatment and the research process complied with ethical standards. Informed consent was obtained from participants and the data obtained was kept confidential and anonymous to protect the privacy of the participants.

Consent for publication

Not applicable.

Data availability

The data that support the findings of this study are available on request from the corresponding author.

Funding

This work was supported by the Guangdong Basic and Applied Basic Research Foundation (number 2025A1515012483, 2023A1515010414, 2022A1515012421, 2019A1515011969), National Natural Science Foundation of China (number 81302387), and the Guangzhou Science and Technology Plan Project (number 202002030031).

Declaration of competing interest

The authors declare that they have no competing interests.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcrp.2025.200406.

References

[1] D. Furman, J. Campisi, E. Verdin, et al., Chronic inflammation in the etiology of disease across the life span, Nat. Med. 25 (12) (2019) 1822–1832, https://doi.org/10.1038/s41591-019-0675-0. PMID: 31806905; PMCID: PMC7147972.
[2] A. Pirillo, M. Casula, E. Olmastroni, et al., Global epidemiology of dyslipidaemias, Nat. Rev. Cardiol. 18 (10) (2021) 689–700, https://doi.org/10.1038/s41569-021-00541-4. PMID: 33833450.
[3] S.H. Suh, S.W. Kim, Dyslipidemia in patients with chronic kidney disease: an updated overview, Diabetes Metab. J 47 (5) (2023) 612–629, https://doi.org/10.4093/dmj.2023.0067. PMID: 37482655; PMCID: PMC10555535.
[4] P.K. Song, Q.Q. Man, H. Li, et al., Trends in lipids level and dyslipidemia among Chinese adults, 2002-2015, Biomed. Environ. Sci. 32 (8) (2019) 559–570, https://doi.org/10.3967/bes2019.074. PMID: 31488232.
[5] M. Arvanitis, C.J. Lowenstein, Dyslipidemia, Ann. Intern. Med. 176 (6) (2023) ITC81–ITC96, https://doi.org/10.7326/AITC202306200. PMID: 37307585.
[6] Y.V. Semenova, A.B. Karpov, R.M. Takhauov, et al., Markers of endothelial dysfunction in patients with arterial hypertension exposed to occupational irradiation of low intensity, Kardiologiia 60 (10) (2020) 73–79, https://doi.org/10.18087/cardio.2020.10.n1236.
[7] S. Fujiwara, A. Suyama, J.B. Cologne, et al., Prevalence of adult-onset multifactorial disease among offspring of atomic bomb survivors, Radiat. Res. 170 (4) (2008) 451–457, https://doi.org/10.1667/rr1392.1. PMID: 19024652.
[8] F.R. Tang, K. Loganovsky, Low dose or low dose rate ionizing radiation-induced health effect in the human, J. Environ. Radioact. 192 (2018) 32–47, https://doi.org/10.1016/j.jenvrad.2018.05.018. PMID: 29883875.
[9] E.A. Ainsbury, C. Dalke, N. Hamada, et al., Radiation-induced lens opacities: epidemiological, clinical and experimental evidence, methodological issues, research gaps and strategy, Environ. Int. 146 (2021) 106213, https://doi.org/10.1016/j.envint.2020.106213. PMID: 33276315.
[10] A. Mazzei-Abba, C.L. Folly, C. Kreis, et al., External background ionizing radiation and childhood cancer: Update of a nationwide cohort analysis, J. Environ. Radioact. 238–239 (2021) 106734, https://doi.org/10.1016/j.jenvrad.2021.106734. PMID: 34521026.
[11] D. Adliene, B. Griciene, K. Skovorodko, et al., Occupational radiation exposure of health professionals and cancer risk assessment for Lithuanian nuclear medicine workers, Environ. Res. 183 (2020) 109144, https://doi.org/10.1016/j.envres.2020.109144. PMID: 32028181.
[12] K. Kelly-Reif, S.J. Bertke, R.D. Daniels, et al., Ionizing radiation and solid cancer mortality among US nuclear facility workers, Int. J. Epidemiol. 52 (4) (2023) 1015–1024, https://doi.org/10.1093/ije/dyae025. PMID: 37253388; PMCID: PMC10527884.
[13] Y. Tatsukawa, K. Cordova, M. Yamada, et al., Incidence of diabetes in the atomic bomb survivors: 1969-2015, J. Clin. Endocrinol. Metab. 107 (5) (2022) e2148–e2155, https://doi.org/10.1210/clinem/dgab902. PMID: 34918116; PMCID: PMC9016441.
[14] T. Azizova, K. Briks, M. Bannikova, et al., Hypertension incidence risk in a cohort of Russian workers exposed to radiation at the mayak production sssociation over prolonged periods, Hypertension 73 (6) (2019) 1174–1184, https://doi.org/10.1161/HYPERTENSIONAHA.118.11719. PMID: 31046470.
[15] S. Park, D.N. Lee, Y.W. Jin, et al., Non-cancer disease prevalence and association with occupational radiation exposure among Korean radiation workers, Sci. Rep. 11 (1) (2021) 22415, https://doi.org/10.1038/s41598-021-01875-2. PMID: 34789809; PMCID: PMC8599676.
[16] A. Brinn, J. Stone, Neutrophil-lymphocyte ratio across psychiatric diagnoses: a cross-sectional study using electronic health records, BMJ Open 10 (7) (2020)

e036859, https://doi.org/10.1136/bmjopen-2020-036859. PMID: 32690528; PMCID: PMC7371128.
[17] G. Arteaga-Henríquez, J. Lugo-Marín, L. Gisbert, et al., Activation of the monocyte/macrophage system and abnormal blood levels of lymphocyte subpopulations in individuals with autism spectrum disorder: a systematic review and meta-analysis, Int. J. Mol. Sci. 23 (22) (2022) 14329, https://doi.org/10.3390/ijms232214329. PMID: 36430805; PMCID: PMC9699353.
[18] T. Bhikram, P. Sandor, Neutrophil-lymphocyte ratios as inflammatory biomarkers in psychiatric patients, Brain Behav. Immun. 105 (2022) 237–246, https://doi.org/10.1016/j.bbi.2022.07.006. PMID: 35839998.
[19] Y. Lian, L. Xie, Y. Liu, et al., Metabolic-related markers and inflammatory factors as predictors of dyslipidemia among urban Han Chinese adults, Lipids Health Dis. 18 (1) (2019) 167, https://doi.org/10.1186/s12944-019-1109-1. PMID: 31472689; PMCID: PMC6717639.
[20] J. Ma, Y. Xie, Y. Zhou, et al., Urinary copper, systemic inflammation, and blood lipid profiles: Wuhan-Zhuhai cohort study, Environ. Pollut. 267 (2020) 115647, https://doi.org/10.1016/j.envpol.2020.115647. PMID: 33254652.
[21] J. Chen, Y. Zhang, R. Wu, et al., Inflammatory biomarkers mediate the association between polycyclic aromatic hydrocarbon exposure and dyslipidemia: a national population-based study, Chemosphere[J] (2024) 142626, https://doi.org/10.1016/j.chemosphere.2024.142626. PMID: 38908446.
[22] Q. Zeng, Y. He, S. Dong, et al., Optimal cut-off values of BMI, waist circumference and waist:height ratio for defining obesity in Chinese adults, Br. J. Nutr. 112 (10) (2014) 1735–1744, https://doi.org/10.1017/S0007114514002657. PMID: 25300318.
[23] N.A. Rigotti, G.R. Kruse, J. Livingstone-Banks, Treatment of tobacco smoking: a review, JAMA 327 (6) (2022) 566–577, https://doi.org/10.1001/jama.2022.0395. PMID: 35133411.
[24] B. Guo, Y. Guo, Q. Nima, et al., Exposure to air pollution is associated with an increased risk of metabolic dysfunction-associated fatty liver disease, J. Hepatol. 76 (3) (2022) 518–525, https://doi.org/10.1016/j.jhep.2021.10.016. PMID: 34883157.
[25] P. Hartley, J.L. Keating, K.J. Jeffs, et al., Exercise for acutely hospitalised older medical patients, Cochrane Database Syst. Rev. 11 (11) (2022) CD005955, https://doi.org/10.1002/14651858.CD005955.pub3. PMID: 36355032; PMCID: PMC9648425.
[26] Joint committee issued Chinese guideline for the management of dyslipidemia in adults. 2016 Chinese guideline for the management of dyslipidemia in adults, Zhonghua Xinxueguanbing Zazhi 44 (10) (2016) 833–853, https://doi.org/10.3760/cma.j.issn.0253-3758.2016.10.005.
[27] J.P. Xu, R.X. Zeng, Y.Z. Zhang, et al., Systemic inflammation markers and the prevalence of hypertension: a NHANES cross-sectional study, Hypertens. Res. 46 (4) (2023) 1009–1019, https://doi.org/10.1038/s41440-023-01195-0. PMID: 36707716.
[28] K. Imai, L. Keele, D. Tingley, A general approach to causal mediation analysis, Psychol. Methods 15 (4) (2010) 309–334, https://doi.org/10.1037/a0020761. PMID: 20954780.
[29] Y. Xi, L. Niu, N. Cao, et al., Prevalence of dyslipidemia and associated risk factors among adults aged ≥35 years in northern China: a cross-sectional study, BMC Public Health 20 (1) (2020) 1068, https://doi.org/10.1186/s12889-020-09172-9. PMID: 32631296; PMCID: PMC7339536.
[30] R.M. Badeau, J. Metso, P.T. Kovanen, et al., The impact of gender and serum estradiol levels on HDL-mediated reverse cholesterol transport, Eur. J. Clin. Invest. 43 (4) (2013) 317–323, https://doi.org/10.1111/eci.12044. PMID: 23397902.
[31] M. Moosazadeh, P. Ebrahimnejad, M. Kheradmand, et al., Association between smoking and lipid profile in men aged 35 to 70 years: dose-response analysis, Am. J. Mens Health 18 (3) (2024) 15579883241249655, https://doi.org/10.1177/15579883241249655. PMID: 38742733; PMCID: PMC11095195.
[32] M.Y. Ou, H. Zhang, P.C. Tan, et al., Adipose tissue aging: mechanisms and therapeutic implications, Cell Death Dis. 13 (4) (2022) 300, https://doi.org/10.1038/s41419-022-04752-6. PMID: 35379822; PMCID: PMC8980023.
[33] S.K. Kim, H.C. Kim, J.S. Shim, et al., Effects of cigarette smoking on blood lipids in Korean men: cardiovascular and metabolic diseases etiology research center cohort, Korean J Intern Med 35 (2) (2020) 369–382, https://doi.org/10.3904/kjim.2019.133. PMID: 31842527; PMCID: PMC7060992.
[34] S. Wang, L. Xu, J.B. Jonas, et al., Prevalence and associated factors of dyslipidemia in the adult Chinese population, PLoS One 6 (3) (2011) e17326, https://doi.org/10.1001/jamainternmed.2022.6817. PMID: 36804760; PMCID: PMC9941971.
[35] X.X. Li, Y. Zhao, L.X. Huang, et al., Effects of smoking and alcohol consumption on lipid profile in male adults in northwest rural China, Public Health 157 (2018) 7–13, https://doi.org/10.1016/j.puhe.2018.01.003.
[36] X.Y. Chen, L. Fang, J. Zhang, et al., The association of body mass index and its interaction with family history of dyslipidemia towards dyslipidemia in patients with type 2 diabetes: a cross-sectional study in Zhejiang Province, China, Front. Public Health 11 (2023) 1188212, https://doi.org/10.3389/fpubh.2023.1188212. PMID: 37255759; PMCID: PMC10225544.
[37] J. Morze, A. Danielewicz, G. Hoffmann, et al., Diet quality as assessed by the healthy eating index, alternate healthy eating index, dietary approaches to stop hypertension score, and health outcomes: a second update of a systematic review and meta-analysis of cohort studies, J. Acad. Nutr. Diet. 120 (12) (2020) 1998–2031.e15, https://doi.org/10.1016/j.jand.2020.08.076. PMID: 33067162.
[38] E.A. Trautwein, S. McKay, The role of specific components of a plant-based diet in management of dyslipidemia and the impact on cardiovascular risk, Nutrients 12 (9) (2020) 2671, https://doi.org/10.3390/nu12092671. PMID: 32883047; PMCID: PMC7551487.
[39] S. Nakajima, E. Eguchi, N. Funakubo, et al., Trends and regional differences in the prevalence of dyslipidemia before and after the great east Japan earthquake: a population-based 10-year study using the national database in Japan, Int. J. Environ. Res. Publ. Health 20 (1) (2022) 560, https://doi.org/10.3390/ijerph20010560. PMID: 36612881; PMCID: PMC9819528.
[40] Y. Tian, Z. Xia, M. Li, et al., The relationship between microwave radiation injury and abnormal lipid metabolism, Chem. Phys. Lipids 225 (2019) 104802, https://doi.org/10.1016/j.chemphyslip.2019.104802. PMID: 31449766.
[41] A.A. Justiz Vaillant, S. Sabir, A. Jan, Physiology, immune response, in: StatPearls [Internet], StatPearls Publishing, Treasure Island (FL), 2024 Jul 27, 2025 Jan–. PMID: 30969623.
[42] L. Ferrucci, E. Fabbri, Inflammageing: chronic inflammation in ageing, cardiovascular disease, and frailty, Nat. Rev. Cardiol. 15 (9) (2018) 505–522, https://doi.org/10.1038/s41569-018-0064-2. PMID: 30065258; PMCID: PMC6146930.
[43] E. Esteve, W. Ricart, J.M. Fernández-Real, Dyslipidemia and inflammation: an evolutionary conserved mechanism, Clin. Nutr. 24 (1) (2005) 16–31, https://doi.org/10.1016/j.clnu.2004.08.004. PMID: 15681098.
[44] A.C. Dongway, A.S. Faggad, H.Y. Zaki, et al., C-reactive protein is associated with low-density lipoprotein cholesterol and obesity in type 2 diabetic Sudanese, Diabetes Metab Syndr Obes 8 (2015) 427–435, https://doi.org/10.2147/DMSO.S85451. PMID: 26379442; PMCID: PMC4567170.
[45] B. Hu, X.R. Yang, Y. Xu, et al., Systemic immune-inflammation index predicts prognosis of patients after curative resection for hepatocellular carcinoma, Clin. Cancer Res. 20 (23) (2014) 6212–6222, https://doi.org/10.1158/1078-0432.CCR-14-0442. PMID: 25271081.
[46] B.W. Tian, Y.F. Yang, C.C. Yang, et al., Systemic immune-inflammation index predicts prognosis of cancer immunotherapy: systemic review and meta-analysis, Immunotherapy 14 (18) (2022) 1481–1496, https://doi.org/10.2217/imt-2022-0133. PMID: 36537255.
[47] L. He, X. Xie, J. Xue, et al., Association of the systemic immune-inflammation index with all-cause mortality in patients with arteriosclerotic cardiovascular disease, Front. Cardiovasc. Med. 9 (2022) 952953, https://doi.org/10.3389/fcvm.2022.952953. PMID: 36172591; PMCID: PMC9510918.
[48] N. Mahemuti, X. Jing, N. Zhang, et al., Association between systemic immunity-inflammation index and hyperlipidemia: a population-based study from the NHANES (2015-2020), Nutrients 15 (5) (2023) 1177, https://doi.org/10.3390/nu15051177. PMID: 36904176; PMCID: PMC10004774.
[49] S. Tapio, M.P. Little, J.C. Kaiser, Ionizing radiation-induced circulatory and metabolic diseases, Environ. Int. 146 (2021) 106235, https://doi.org/10.1016/j.envint.2020.106235. PMID: 33157375; PMCID: PMC10686049.
[50] N.P.B. Au, T. Wu, G. Kumar, et al., Low-dose ionizing radiation promotes motor recovery and brain rewiring by resolving inflammatory response after brain injury and stroke, Brain Behav Immun. 115 (2024) 43–63, https://doi.org/10.1016/j.bbi.2023.09.015. PMID: 37774892.