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Review article

Relationship between body fat ratio and inflammatory markers in a Chinese population of adult male smokers

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ABSTRACT

Objective: To explore the correlation between changes in the body fat ratio (BFR) and peripheral blood inflammatory markers according to smoking status in the adult Chinese male population.
Methods: A total of 865 participants (aged 20–70 years) were included. All participants underwent a physical health examination at Xiguzhou Central Hospital between October 2015 and July 2016, including measurements of body mass index (BMI), BFR, white blood cell [WBC] count, and neutrophil–lymphocyte ratio [NLR].
Results: WBCs count and NLR were significantly higher in adult male smokers than in non-smokers ($P = 0.00$). According to the BFR stratification analysis, WBC count and NLR significantly increased in accordance with BFR ($P = 0.00$). This finding remained significant after adjusting for relevant confounding factors ($P < 0.05$). Two-factor stratified analysis of smoking status and BFR showed that WBC count and NLR in the smoking population were higher than in nonsmokers, regardless of BFR. The interaction model showed that BFR and smoking status affected WBC count and NLR changes ($P < 0.05$). A significant positive correlation was found between WBC count, NLR, and BFR in adult male smokers; however, there was no significant correlation with BMI. There was an interaction between smoking and BFR, both of which synergistically affected changes in inflammatory markers, including WBC count and NLR.
Conclusion: WBC count and NLR of smokers with a high BFR were significantly higher than those of nonsmokers with a low BFR. It is important to provide evidence-based medical evidence for social tobacco control and to reduce BFR.

1. Introduction

In 2016, the Non-communicable Disease Risk Factor Cooperative Organization (NCD-RisC) estimated that the prevalence of overweight and obesity in adults (those with BMI $\geq$ 25 kg/m^2) was 38.5% in men and 39.2% in women, affecting approximately 2.01 billion adults globally (Risk Factor Collaboration and (NCD-RisC), , 2017). Obesity is prevalent in many countries, and its complications have become one of the most important causes of death worldwide (Khosravi et al., 2016). As obesity is a metabolic disorder, one of the main pathophysiological changes in obese individuals is an increase in circulating inflammatory markers (Saltiel and Olefsky, 2017).

A *meta*-analysis showed that white blood cell (WBC) count is a significant inflammatory biomarker associated with metabolic syndrome (Saltiel and Olefsky, 2017; Aguilar-Valles et al., 2015). WBC count has received extensive attention in the pathogenesis of metabolic-related diseases. In recent years, the neutrophil-to-lymphocyte ratio (NLR) has been favored as a reliable inflammatory biomarker of systemic inflammation and is currently widely used in the assessment of inflammation and neoplastic diseases because of its ease of monitoring and identification (Tang et al., 2017; Mozos et al., 2017). Studies have shown that NLR is a high-risk factor for coronary artery disease (CAD) associated with atherosclerosis and is also affected by risk factors for atherosclerosis, such as metabolic syndrome and its components (overweight/obesity, hypertension, and diabetes) (Küçük et al., 2016; Kim et al., 2018; Kim et al., 2013). Currently, few studies exist regarding

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WBC count and NLR in an obese population.

Currently, various methods are used to assess obesity, including BMI, waist circumference (WC), and waist-to-hip ratio (WHR) (Huiming and Sen, , 2017). With advances in technology, the determination of the body fat ratio (BFR) has gradually begun to be applied in clinical practice. BFR refers to the proportion of body fat to total body weight, also known as body fat percentage, and reflects the amount of fat in the body (Huiming and Sen, 2017). A domestic study comparing BFR with BMI suggested that BFR reflects fat content (Huiming and Sen, 2017). We found that the majority of smokers showed central obesity. However, BMI reflects only the overall level of obesity. The prevalence of obesity and its subsequent cardiovascular and other complications is significantly higher in men than in women. The main reason for this is because smoking is widespread among men (Gonghuan, 2009). Smoking is a risk factor of metabolic disorders and atherosclerosis (Shen et al., 2018). Current smokers are at four times greater risk of developing cardiovascular disease than those who have never smoked or even those who have recently quit (Pirie et al., 2013).

Many studies have examined the effect of smoking on peripheral WBC count. The putative effect of smoking on inflammatory processes was first identified in the 1960s. Recent evidence strongly suggests that the molecular mechanisms underlying smoking-induced modulation of inflammation mainly involve the nuclear factor-kappa B (NF-kB) family, through the activation of both inhibitor of IkB kinase (IKK)-dependent and -independent pathways. In addition to NF-kB activation, a number of transcription factors, including GATA, PAX5, and Smad 3/4, have also been implicated (Gonçalves et al., 2011). A recent study demonstrated that nicotine stimulates neutrophil IL-8 production via nicotinic acetylcholine receptors by generating peroxynitrite and subsequently activating NF-kB, contributing to leukocytosis in smokers (Iho et al., 2003). However, little research has been performed on the relationship between peripheral blood inflammatory factors and BFR with consideration of smoking status.

We selected BFR as an obesity-related index and peripheral blood WBC count and NLR as inflammatory factors. We aimed to analyze the correlation between these two inflammatory markers and BFR, explore the correlation between the change in body fat percentage and peripheral blood inflammation index in adult men under smoking conditions, and provide evidence-based medical evidence for social tobacco control and weight loss efforts.

2. Participants and methods

2.1. Participants

A total of 1300 participants (aged 20–70 years) were enrolled. All participants underwent a physical health examination at Xuzhou Central Hospital from October 2015 to July 2016. Participants were excluded if they had physiological or pathological conditions causing leukocytosis (Wei et al., 2014), such as.

(1) acute and chronic infectious diseases, including upper respiratory infections, parasitic infections, and inflammation of various systems;

(2) state of tissue damage, including burns, trauma, and post-major surgery;

(3) hemorrhage from various causes that may result in an increased WBC count;

(4) severe primary disease, including diabetes, diseases of the biliary and hematopoietic systems, and diseases of the pancreas, heart, brain, and kidney;

(5) psychiatric disorders, the inability to communicate with physicians, or incomplete information;

(6) a history of drug use or food allergies;

(7) autoimmune diseases;

(8) inhalation or oral corticosteroid therapy or lung disease; or.

(9) anti-inhibitory or cytotoxic drugs administered within one year.

Ultimately, 865 participants were included in the analysis. All participants provided informed consent to participate. This study was reviewed and approved by the Ethics Committee of Central Hospital of Xuzhou.

2.1.1. Data collection

All body measurements, including height, weight, WC, and blood pressure (BP), were performed by professional nurses and physicians.

Height and weight were determined with the participants wearing light clothing and not wearing shoes. BMI was calculated as weight in kilograms divided by the square of height in meters. WC was measured at the midpoint between the lowest rib margin and the iliac crest in the standing position. Trained doctors measured BP using a mercury sphygmomanometer on the dominant arm after a resting period of at least 5 min in the supine position. The participant's arm was placed at the level of the heart, and BP values were calculated as the mean of three measurements.

2.1.2. Questionnaire survey

Qualified physicians and researchers used a questionnaire to record information concerning each participant's general situation, sex, age, past medical history (e.g., cardiovascular disease, hypertension, diabetes), family history, income status, education level, alcohol and tobacco use, and medication use in the preceding six months. All staff members underwent intensive training to standardize the operational procedures and methods prior to commencement of the study.

2.1.3. Determination of biochemical indicators

Blood samples were collected after participants fasted overnight (at least 10 h). Participants were also required to empty their bladders. After blood was drawn, samples were allowed to clot at room temperature for 1–3 h. Immediately after samples clotted, they were centrifuged for 15 min at 3000 rpm to separate the serum. Blood samples were collected to measure counts of WBCs, neutrophils, lymphocytes, red blood cells, and platelets, as well as fasting blood glucose, cholesterol, triglyceride, low-density lipoprotein, and high-density lipoprotein levels using an autoanalyzer (Type7600; Hitachi Ltd., Tokyo, Japan).

2.1.4. Determination of body fat ratio

The BFR was measured using the electrical impedance method with an Inbody 3.0 body composition analyzer (Biospace, Seoul, Korea). For the measurements, participants were in a fasted state, calm, and with no shoes or socks. While maintaining balance, participants placed their feet on the foot electrodes and held the hand electrodes in both hands. After the age, height, and sex of the subjects were sequentially entered using a keyboard, their BFR was measured (Man and Jingmin, 2005).

2.1.5. Statistical analysis

The statistical software SPSS (version 17.0 (SPSS Inc. Released 2008. SPSS Statistics for Windows, Version 17.0. Chicago: SPSS Inc) was used for data management and statistical analyses. All hypothesis tests were performed using a two-sided test. Measured data are expressed as means $\pm$ standard deviation ($\bar{x}\pm s$), and count data are expressed as numerical values. Two independent samples t tests were used for between-group comparisons. A general linear equation test was used to analyze grouping trends under different BFRs and BMIs. Correlations between different indicators and the BFR were analyzed by stratification. An interaction model was used to analyze the relationships among BFR, inflammatory markers, and smoking status. $P \leq 0.05$ was considered statistically significant.

3. Results

3.1. Comparison of general clinical data and biochemical indicators

A total of 865 men were included in the analysis, 527 (61%) of whom

were smokers. Baseline data were divided into two groups based on the smoking status. Table 1 shows the distribution of indicators between the two groups. WC, inflammatory indicators, and triglyceride and high-density lipoprotein levels were significantly higher in the smoking group than in the non-smoking group ($P < 0.05$). Interestingly, non-smokers had significantly higher educational levels than smokers ($P = 0.00$). Specifically, the average values of WBC, neutrophil, and lymphocyte counts and NLR in the smoking population were higher than those in the non-smoking population. This difference was statistically significant ($P = 0.00$). However, there were no significant differences in age, BMI, systolic BP, diastolic BP, fasting blood glucose level, total cholesterol level, low-density lipoprotein level, blood uric acid level, red blood cell count, platelet count, annual income, or drinking history ($P > 0.05$).

3.2. Correlation analysis between the inflammation index and BMI

We further evaluated the association between these two inflammatory markers and BMI. BMI tertiles were grouped as follows: T1, <24 kg/m^2; T2, 24–26 kg/m^2; and T3, >26 kg/m^2. The relationships between peripheral blood WBC count, NLR, and BMI were analyzed separately, as shown in Table 2. Using WBC count as the dependent variable, the WBC count corresponding to different BMIs revealed a statistically significant trend with an increase in BMI ($P = 0.00$). However, after adjusting for confounding factors such as age and BFR and further adjusting for fasting systolic BP; diastolic BP; blood glucose, triglyceride, total cholesterol, high-density lipoprotein, and high-density lipoprotein levels; annual income; education; and drinking behavior, there were no statistically significant differences ($P > 0.05$). In the equation where NLR was used as the dependent variable, NLR did not change with BMI, regardless of the adjustment for confounding factors. The differences between the three groups were not statistically significant ($P > 0.05$).

Table 1
Comparison of general clinical data and biochemical indicators.

Variable	Non-smokers	Smokers	t/χ2	*P* value
N	338	527		
Age (years)	45.49 ± 8.87	46.24 ± 90.27	1.19	0.23
BMI (kg/m^2)	24.78 ± 2.76	24.08 ± 2.95	1.49	0.14
WC(cm)	88.22 ± 7.47	89.85 ± 8.23	2.95	0.00
SBP(mmHg)	125.17 ± 14.75	126.23 ± 15.38	1.00	0.31
DBP(mmHg)	79.96 ± 11.09	81.05 ± 11.15	1.40	0.16
FPG(mmol/L)	5.25 ± 0.80	5.28 ± 1.12	0.43	0.66
TC(mmol/L)	5.03 ± 0.92	5.12 ± 0.93	1.43	0.15
TG(mmol/L)	1.80 ± 1.94	2.07 ± 1.83	2.09	0.04
HDL(mmol/L)	1.20 ± 0.29	1.16 ± 0.26	2.08	0.04
LDL(mmol/L)	2.99 ± 0.80	3.03 ± 0.79	0.60	0.55
SUA(μmol/L)	334.60 ± 73.57	334.73 ± 68.90	0.03	0.98
RBC(10*10^{12}/L)	4.95 ± 0.35	4.93 ± 0.36	1.07	0.29
PLT (10*10^9/L)	205.56 ± 45.12	219.66 ± 48.75	4.04	0.00
Educational level			17.34	0.00
Junior high school or below	12(4%)	25(7%)		
High school/secondary school	38(11%)	117(22%)		
College and above	260(78%)	353(67%)		
Annual income (RMB)			0.29	0.87
<15000	132(39%)	217(41%)		
15000 ~ 20000	80(24%)	121(23%)		
>20000	79(23%)	132(25%)		
Drinking history			3.56	0.06
Yes	290(86%)	476(90%)		
No	31(9%)	31(6%)		
Inflammation indicators				
WBC(10*10^9/L)	5.76 ± 1.31	6.40 ± 1.60	5.89	0.00
Neu(10*10^9/L)	3.15 ± 1.04	3.55 ± 1.15	5.03	0.00
Lym(10*10^9/L)	2.09 ± 0.55	2.25 ± 0.63	3.64	0.00
NLR	1.59 ± 0.70	1.75 ± 0.58	3.46	0.00

BMI: body mass index; WC:waist circumference;SBP: systolic pressure; DBP: diastolic blood pressure; FPG: fasting blood glucose;TC: total cholesterol; TG: triglycerides; HDL: high-density lipoprotein; LDL: low-density lipoprotein;SUA: serum uric acid; RBC: red blood cell; PLT: platelet; WBC: white blood cell; Neu: neutrophils; Lym: lymphocytes; NLR: the neutrophil–lymphocyte ratio;

Table 2
Correlation analysis between the inflammation index and BMI.

	Variable	BMI			t value	*P* value
		T1 group <23.73	T2 group 23.73–26.05	T3 group >26.05		
WBC (10*10^9/L)	Model1	5.88 ± 0.10	6.14 ± 0.10	6.40 ± 0.10	3.86	0.00
	Model2	6.06 ± 0.10	6.15 ± 0.09	6.23 ± 0.10	1.07	0.29
	Model3	6.18 ± 0.11	6.16 ± 0.09	6.11 ± 0.10	0.58	0.57
NLR	Model1	1.57 ± 0.04	1.73 ± 0.04	1.58 ± 0.04	0.20	0.84
	Model2	1.61 ± 0.04	1.75 ± 0.04	1.54 ± 0.04	1.18	0.24
	Model3	1.61 ± 0.04	1.74 ± 0.04	1.54 ± 0.04	1.12	0.26

BFR: body fat ratio; WBC: white blood cell.
NLR: the neutrophil–lymphocyte ratio.
Model 1: unadjusted.
Model 2: adjusted for age and BFR.
Model 3: adjusted for age, BFR, FPG, SBP, DBP, TG, TC, HDL, LDL, Educational level, Annual income and Drinking history.

3.3. Correlation analysis between the inflammation index and BFR

BFR tertiles were grouped into BF1 (<23%), BF2 (23–27%), and BF3 (>27%) groups. Relationships among peripheral blood WBC count, NLR, and BFR were analyzed separately, as shown in Table 3. Using WBC count as the dependent variable, WBC count corresponding to different BFRs showed a statistically significant difference among the three groups. Model 1 showed that as BFR increased, WBC count also increased, and there was a statistically significant trend ($P = 0.00$). After adjusting for age and BMI, this trend remained ($P = 0.00$). When further adjusted for systolic BP; diastolic BP; total cholesterol, triglyceride,

Table 3
Correlation analysis between the inflammation index and BFR.

	Variable	BFR(%)			t value	*P* value
		BF1 group <23%	BF2 group 23%-27%	BF3 group >27%		
WBC (10*10^9/L)	Model1	5.92 ± 0.10	6.10 ± 0.10	6.47 ± 0.10	4.03	0.00
	Model2	5.97 ± 0.11	6.11 ± 0.10	6.41 ± 0.11	2.70	0.00
	Model3	5.99 ± 0.11	6.11 ± 0.10	6.42 ± 0.11	2.02	0.03
NLR	Model1	1.54 ± 0.04	1.65 ± 0.04	1.70 ± 0.04	3.10	0.02
	Model2	1.51 ± 0.04	1.65 ± 0.04	1.73 ± 0.04	3.02	0.00
	Model3	1.51 ± 0.05	1.65 ± 0.04	1.73 ± 0.04	3.12	0.00

BFR: body fat ratio; WBC: white blood cell.
NLR: the neutrophil–lymphocyte ratio.
Model 1: unadjusted.
Model 2: adjusted for age and BMI.
Model 3: adjusted for age, BMI, FPG, SBP, DBP, TG, TC, HDL, LDL, Educational level, Annual income and Drinking history.

high-density lipoprotein, and high-density lipoprotein levels; annual income; education; and drinking behavior, this trend remained (P = 0.03). In the equation using NLR as the dependent variable, NLRs corresponding to the different BFR tertiles also increased, and the trend was statistically significant (P = 0.02). This trend remained statistically significant after further correction for relevant confounders (P = 0.00).

3.4. Correlation analysis between BFR and inflammatory markers by smoking status

The data were divided into three groups according to BFR tertiles. We then separately analyzed the relationship between WBC count and smoking status, BMI, and WC. Table 4 shows that with the change in BFR, changes in WBC count in smokers and non-smokers showed a statistically significant increasing trend ($P < 0.05$). Moreover, there was an interaction between BFR and smoking status, which affected changes in WBC count (P value of interaction = 0.04). In the T3 group, there was a significant difference between the distribution of WBC and the change in BFR (P = 0.01). However, in the T1 group, there was no significant correlation between WBC count and BFR changes. Further interaction analysis revealed no interaction between BMI and BFR with WBC count (P value of interaction = 0.68). In participants with a WC ≥ 85 cm, changes in WBC count were observed in concert with changes in BFR (P = 0.00). However, in participants with a WC < 85 cm, this trend was not statistically different (P = 0.80). Similarly, no interaction of WBC count with WC and BFR was observed (P value of interaction = 0.19).

We also analyzed the correlation of NLR with smoking status, BMI, and WC according to BFR tertiles. As shown in Table 4, with a change in the BFR, there was a statistically significant increasing trend in NLR changes among smokers (P = 0.01). A similar statistically significant increasing trend in the changes in NLR in nonsmokers was also osbserved(P = 0.04). Meanwhile, the interaction of smoking status and BFR with NLR was statistically significant (P value = 0.00). In the low BMI group, NLR increased with BFR (P = 0.00), whereas in the high BMI group, this trend was not statistically different (P = 0.73). In addition, there was no interaction among BMI, BFR, and NLR (P value of interaction = 0.87). However, in participants with WC ≥ 85 cm, NLR did not change in accordance with BFR (P = 0.29). Similarly, in participants with WC < 85 cm, NLR did not change with changes in BFR (P = 0.11). No interaction among WC, BFR, and NLR (P = 0.49).

3.5. Changes in BFR in smokers and non-smokers interact with WBC count and NLR, respectively

Fig. 1 and Fig. 2 show the effects of smoking status and BFR interactions on WBC count and NLR, respectively. Fig. 1 shows that with an increase in BFR, the increase in WBC count in the smoking group was significantly higher than that of the non-smoking group, and the difference was statistically significant (P = 0.04). Fig. 2 shows that with an increase in BFR, the increase in NLR in the smoking group was significantly higher than that of the non-smoking group, and the difference was statistically significant (P = 0.00). The two sets of graphs show that this interaction was more pronounced in the smoking population. In smokers, the two inflammatory markers (WBCs count and NLR) changed with the BFR values, and this change was more significant than that in non-smokers. The increase in the inflammatory index was significantly higher in smokers than in non-smokers.

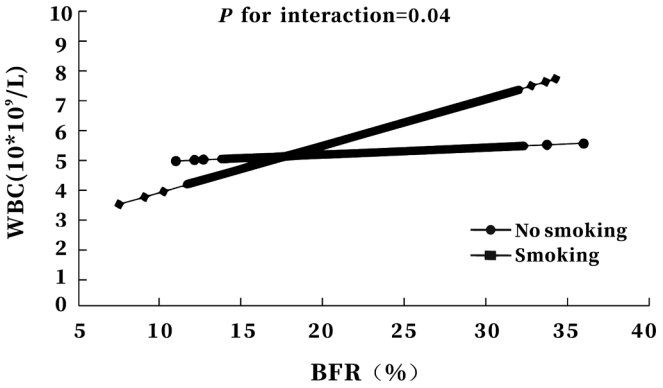

Fig. 1. Changes in BFR in smokers and non-smokers interact with WBC Note: BFR: body fat ratio; WBC: white blood cell.

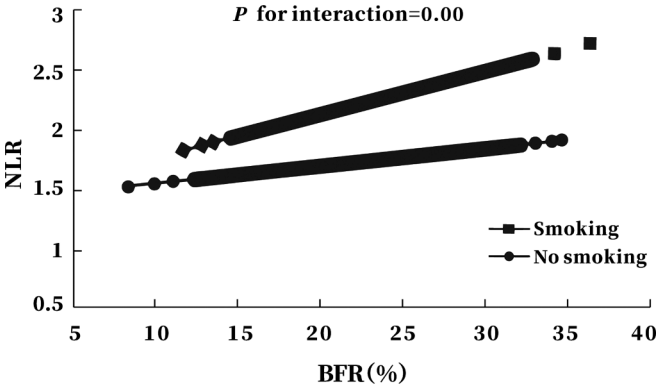

Fig. 2. Changes in BFR in smokers and non-smokers interact with NLR Note: BFR: body fat ratio; NLR: the neutrophil–lymphocyte ratio.

Table 4
Correlation analysis between BFR and inflammatory markers (WBCs and NLR) by smoking status.

Inflammatory markers	Variable		BFR(%)				*P* value of interaction
			BF1 group <23%	BF2 group 23%-27%	BF3 group >27%	Trend *P* value	
WBCs	Smoking status	No	5.45 ± 1.07	5.65 ± 1.10	6.13 ± 6.13	0.00	0.04
		Yes	6.20 ± 1.66	6.36 ± 1.43	6.67 ± 1.68	0.04	
	BMI(kg/m^2)	<28	5.83 ± 1.45	6.13 ± 1.41	6.52 ± 1.69	0.09	0.68
		≥28	6.04 ± 1.58	5.65 ± 1.10	6.13 ± 6.13	0.01	
	WC(cm)	<85	5.74 ± 0.13	5.79 ± 0.18	5.97 ± 0.31	0.80	0.19
		≥85	5.95 ± 0.10	6.10 ± 0.09	6.43 ± 10.10	0.00	
NLR	Smoking status	No	1.49 ± 0.08	1.52 ± 0.07	1.73 ± 0.08	0.04	0.00
		Yes	1.52 ± 0.05	1.71 ± 0.05	1.76 ± 0.05	0.01	
	BMI(kg/m^2)	<28	1.54 ± 0.05	1.66 ± 0.04	1.77 ± 0.05	0.00	0.87
		≥28	1.64 ± 0.15	1.50 ± 0.10	1.54 ± 0.05	0.73	
	WC(cm)	<85	1.51 ± 0.05	1.62 ± 0.07	1.44 ± 0.13	0.29	0.49
		≥85	1.57 ± 0.06	1.65 ± 0.05	1.73 ± 0.04	0.11	

WBC: white blood cell; NLR: the neutrophil–lymphocyte ratio; BFR: body fat ratio.
BMI: body mass index; WC: waist circumference.

4. Discussion

In this study, we found that differences in WBC count, neutrophil count, lymphocyte count, and NLR between smokers and non-smokers; WBC count and NLR of adult male smokers were significantly higher than those of non-smokers. This is consistent with the results of previous studies by Shahabinejad et al. (Shahabinejad et al., 2016) and King et al. (King et al., 2017). Interestingly, we found that the majority of smokers showed central obesity. Because BMI only reflects the overall obesity level, we further studied the relationship between the two indicators of inflammation and BMI. We found no significant correlation between WBC count, NLR, and BMI in adult male smokers after adjusting for confounding factors. Therefore, this study selected BFR as the obesity-related index, which was subsequently divided into three. The results showed a significant positive correlation between WBC count, NLR, and BFR levels in adult male smokers. This finding remained significant after correcting for confounding factors. In summary, this study concluded that there was a significant positive correlation between WBC count, NLR, and BFR in adult male smokers, but there was no significant correlation with BMI. Our conclusions are consistent with those of a domestic study comparing BFR and BMI (Huiming and Sen, 2017), which found that BFR is a more accurate indication of fat content (Huiming and Sen, 2017). The results of a longitudinal assessment of BFR and diabetes risk in Korea suggest that the prevalence of diabetes in individuals with high BFR is elevated and independent of BMI (Park et al., 2018). Fairchild et al. showed that increased BFR, but not BMI, reduced insulin sensitivity and insulin resistance (Fairchild et al., 2018). These findings suggest that BFR and BMI differ in the assessment of obesity and that these are different for different metabolic disorders. The BFR reflects body fat content more directly than BMI and is a more accurate indicator of obesity.

Many studies have examined the effect of smoking on peripheral blood WBC count; however, few have assessed the relationship between peripheral blood inflammatory factors and BFR according to smoking status. Therefore, this study analyzed the correlation between the BFR, WBC count, and NLR under different smoking conditions in a male population. We found an interaction between smoking and BFR, which affects changes in inflammatory indices, such as WBC count and NLR: WBC and NLR levels of smokers with high BFR were significantly higher than those of non-smokers with low BFR.

Multiple possible mechanisms underlie these findings. (1) Obesity leads to an increases the volume and number of fat cells. First, the activation of various pro-inflammatory factors, such as interleukin 1, interleukin 6, tumor necrosis factor α, monocyte chemotactic protein, leptin, and resistin, is induced by different pathways. This further promotes an increase in the levels of peripheral blood inflammatory factors (Aoshiba et al., 2001). (2) In a comprehensive analysis of 29 studies, a *meta*-analysis of 148,731 individuals (Morris et al., 2015) showed that smoking causes fat accumulation in the upper body, resulting in poor body fat distribution while increasing weight, WC, and hip circumference and leading to central obesity (Fujiyoshi et al., 2016). The mechanism of smoking-induced central obesity is unclear and may be related to the following aspects: first, cigarette smoke components antagonize estrogen, causing endocrine disruption in smokers and promoting abdominal fat accumulation (Canoy et al., 2005); second, cigarettes are rich in nicotine, which increases circulating cortisol concentration and promotes abdominal fat accumulation (Fujiyoshi et al., 2016); third, smoking promotes high lipoprotein lipase expression in adipose tissue of the buttocks, which reduces the activity of lipoprotein lipase in the blood—this thereby promotes triglyceride uptake into abdominal adipose tissue and utilization of free fatty acids, which further reduces triglyceride clearance in the blood and therefore increased blood lipid concentration (Liu et al., 2010). (3) Activation of various pro-inflammatory factors promotes the differentiation and maturation of WBCs and inhibits the migration of inflammatory factors to the extravascular space; this increases the number of circulating WBCs, which leads to an increase in peripheral blood inflammatory factors (Nannan and Yinglong, 2017; Yibo and Yu, 2004).

The main indicators used to assess body composition are BMI, WC, WHR, and BFR. As stated above, BMI mainly reflects the overall obesity level. It is widely used because its measurement method is simple and reliable. However, it has certain limitations. For individuals with strong skeletal muscles, although not obese, BMI may be high. Similarly, for obese people with low muscle mass, the BMI may be within the normal range. Second, BMI cannot be used to assess the distribution of body fat (Man and Jingmin, 2005; Hung et al., 2017). In contrast, WC and WHR are indicators of central obesity and measure the extent of abdominal obesity (Czernichow et al., 2011). Most smokers in the present exhibited central obesity, whereas BMI only reflected the overall level of obesity. For abdominal obesity, BMI may be within the normal range, which may explain why there was no significant correlation of WBC count and NLR with BMI in adult male smokers.

This study has some limitations. First, we conducted a questionnaire survey that included the smoking status and did not specifically measure the biological indicators of tobacco exposure. For example, the nicotine concentration was not measured, and the questionnaire may have led to intentional concealment, which affects the true relationship between the smoking population and the two inflammatory indicators. Second, this study selected physically healthy participants and did not analyze overweight and obese people, and the sample size was not sufficiently large; thus, the results may differ from other results. Finally, this was a cross-sectional study, and further prospective and mechanistic studies are required to explore potential causality.

5. Conclusion

We found that WBC count and NLR of adult male smokers were significantly higher than those of non-smokers. There was a significant positive correlation among WBC count, NLR, and BFR in adult male smokers, but no significant correlation was observed with BMI. There was an interaction between smoking and BFR, which affected changes in inflammatory markers, such as WBC count and NLR. Moreover, WBC count and NLR of smokers with high BFR were significantly higher than those of non-smokers with low BFR.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

The data that has been used is confidential.

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Ethics approval and consent to participate

This study was reviewed and approved by the Ethics Committee of Central Hospital of Xuzhou.

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