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RESEARCH

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A cross-sectional study of serum lipids, body mass index and age relationships with breast cancer risk

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Abstract

Background Globally, the most common malignancy in women today is breast cancer. Numerous factors affect the incidence of breast cancer; therefore, we examined the connections involving age, body mass index (BMI), serum lipid levels, and breast cancer risk in women.

Methods This was a cross-sectional analytical study. 382 female patients with a breast cancer diagnosis in this study, and 11842 healthy, age-matched females who were selected from physical examination centers in the same period. Univariate analysis was conducted first, after which factors with statistically significant differences were used to construct a multi-factor binary logistic regression equation. We explored associations across different ages, BMI, triglyceride (TG), and high-density lipoprotein-cholesterol (HDL-C) levels, and breast cancer risk.

Results Age, BMI, TG, and HDL-C were the risk factors that showed the most significant association with breast cancer. Age, BMI, low-density lipoprotein-cholesterol (LDL-C) and TG levels in the breast cancer group were higher than those in the control group, but HDL-C and total cholesterol (TC) levels were lower. As BMI and TG levels increased, the risk of developing breast cancer increased, and, as HDL-C levels decreased, the risk of developing breast cancer increased. Women aged ≥ 40 years old had an increased breast cancer risk. There were no significant variations in TC and LDL-C levels between groups.

Conclusions In this study, a lower risk of breast cancer was linked to high HDL-C levels, while a higher risk of breast cancer was linked to high BMI and TG levels. Women aged ≥ 40 years old had an increased breast cancer risk.

Keywords Serum lipids, Body mass index (BMI), Age, Breast cancer

Introduction

The findings of the 2022 Global Cancer Report, which was published by the International Agency for Research on Cancer, indicate that, approximately 2.3 million females have breast cancer, accounting for 23.8% of new cases [1]. Breast cancer is now the most prevalent cancer, surpassing lung cancer. In 2022, it caused approximately 670,000 deaths, accounting for 6.8% of all cancer-related mortality [1]. In 2022, a study reported approximately 430000 new breast cancer cases in China, including approximately 124000 deaths, thus breast cancer incidence and mortality rates in China have sharply increased [2]. Breast cancer is largely influenced by increasing age, body mass index (BMI), usage of exogenous female hormones, alcohol consumption, sleep disruption and duration, physical inactivity, reproductive factors, and other factors [3]. These risk factors are important for preventing breast cancer.

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Obesity and age have critical effects on breast cancer. With studies reporting that the risk of developing breast cancer rises with age [4]. Additionally, obesity has been defined as a breast cancer risk factor by the International Agency for Research on Cancer [5]. According to Chinese population statistics, more than half of adults are overweight or obese, a figure which has tripled in the past 20 years, while approximately 41.6% are women [6, 7]. Additionally, research indicates that, relative to normal-weight patients, obese breast cancer patients presented with larger initial tumors, increased rates of lymphatic infiltration, and reduced overall survival, demonstrating an 11% decrease in overall survival among the obese breast cancer patients [8]. More so, studies have reported that a high BMI increases breast cancer development risks, with more aggressive tumors and a worse prognosis [9]. Epidemiologically, metabolic syndrome is a high-risk factor for breast cancer occurrence and development [10]. Metabolic syndrome is a collection of complex metabolic disorders typified by obesity, hypertension, dyslipidemia, and hyperglycemia. Of these, abnormal lipid metabolism has important roles in malignant tumor occurrence and development, with exogenous lipids exerting a considerable influence on breast cancer [11]. Additionally, abnormal cholesterol levels are closely related to breast cancer cell proliferation and migration. Research indicates that 27-hydroxycholesterol, a significant cholesterol metabolite, functions as a selective estrogen receptor modulator, endogenously promoting the proliferation and metastasis of estrogen receptor-positive breast cancer cells [12]. The two primary types of cholesterol, LDL-C and HDL-C, are associated with breast cancer occurrence [13]. Increased HDL-C levels can also decrease insulin-like growth factor-1 (IGF-1) expression, which in turn promotes breast cancer occurrence, while LDL-C can cause the migration and proliferation of cancer cells by triggering the P38 signaling pathway, which is closely related to breast cancer occurrence and development [14]. A previous meta-analysis showed that serum total cholesterol (TC) and HDL-C levels were negatively associated with breast cancer risk, but not LDL-C [15]. A Mendelian randomization study found that elevated LDL-C and gene-mediated HDL-C levels were connected to a high breast cancer risk [16]. Currently, the relationship between TC, LDL-C, HDL-C, BMI and breast cancer is controversial, with relatively little research on associations between TG and breast cancer risk. Therefore, to address this knowledge gap, we analyzed relationships between age, BMI, TC, TG, LDL-C, and HDL-C and breast cancer, and provided new insights for breast cancer prevention and treatment.

Methods

Participants

Based on our experimental design, 382 female patients were given a breast cancer diagnosis at the Fifth Affiliated Hospital of Southern Medical University, from September 2017 to September 2024 and aged ≥ 18 years old, were recruited and assigned to the breast cancer group. Over the same period (from September 2017 to September 2024), 11842 age-matched healthy women were selected from physical examination centers as the control group according to a 1:31 ratio. Participants with a prior malignant tumor history were excluded from the breast cancer group, while participants with a prior malignant tumor history or currently suffering from malignant tumors were excluded from the control group, and participants lacking laboratory serum lipid data, and pregnant and lactating participants were excluded from both groups. Breast ultrasound results in the control group showed no breast lesions, with normal results.

Sample collection

Information included breast cancer group and control participant age, breast ultrasound data, height, weight, BMI [weight (kg)/height $(m)^2$], and serum lipids (TC, LDL-C, HDL-C, and TG).

Laboratory methods

Fasting 8 h venous bloods were collected from the breast cancer group at 3 days before their operation and from healthy subjects (control group) on the morning of their physical examination. Bloods were centrifuged after complete coagulation, and TG and TC was tested using an enzyme colorimetry method, while LDL-C and HDL-C levels were tested using a direct method-surface activity elimination method. To ensure instrument accuracy, external quality control products were also tested. When measuring height, individuals in both groups removed their shoes and hats, straightened their bodies, and leveled their eyes. The distance between the horizontal plane and the foot plane was taken as the height (units = cm, accurate to 1 decimal place). For weight measurements, individuals in both groups stood on a scale with their feet shoulder-width apart. The body was held upright with no support (units = kg, accurate to 1 decimal place).

Serum lipid, BMI, and age classification

Due to possible racial differences, study participants were all Chinese. Therefore, we used Chinese adult dyslipidemia prevention guidelines [17] for serum lipid level

stratification standards (mmol/L) to stratify breast cancer and control groups; TG < 1.7 mmol/L = appropriate level and TG ≥ 1.7 mmol/L = elevated level.

LDL-C < 2.6 mmol/L = ideal level, LDL-C < 3.4 mmol/L = appropriate level, 3.4 mmol/L ≤ LDL-C < 4.1 mmol/L = marginally increased, and LDL-C ≥ 4.1 mmol/L = increased levels.

HDL-C < 1.0 mmol/L = decreased levels.

TC < 5.2 mmol/L = appropriate levels and TC ≥ 5.2 mmol/L = elevated levels.

Due to possible racial differences, study participants were all Chinese. Therefore, according to a consensus of Chinese resident obesity prevention experts [18], recommended BMI stratification criteria (kg/m^2) were used to stratify both groups; BMI < 18.5 kg/m^2 was considered under-weight, 18.5 kg/m^2 ≤ BMI ≤ 23.9 kg/m^2 was normal, 24 kg/m^2 ≤ BMI ≤ 27.9 kg/m^2 was overweight, and BMI ≥ kg/m^2 was obese.

Studies have shown that being 40 years old is a critical point for breast cancer [19]. Therefore, TG < 1.7 mmol/L, LDL-C < 2.6 mmol/L, HDL-C ≥ 1.0 mmol/L, TC < 5.2 mmol/L, 18.5 kg/m^2 ≤ BMI ≤ 23.9 kg/m^2, and 18 years ≤ age ≤ 39 years were used as references.

Statistical analyses

Data analysis was conducted using SPSS 29.0 software, with all data tested for normality. Skewed distribution data were expressed by the median and quartile distance M (Q1, Q3), and because the data were all skewed distribution, the rank sum test was used for comparison. Classification data were described by frequency and percentages (n, %), and rank sum tests were utilized to check for variations in serum lipid subtype, BMI, and age distribution between breast cancer and control groups. The threshold for statistical significance was $p < 0.05$. Also, to examine the impact of various factors (age, BMI, etc.) on the incidence of breast cancer, multivariate binary logistic regression analysis was employed to incorporate clinically significant variables and concurrently investigate the relationship between different TG, HDL-C, age, BMI levels and breast cancer occurrence.

Results

In our study, 12224 subjects were recruited and matched according to a 1:31 ratio, including 382 subjects within the breast cancer group and 11842 in the control group (Table 1). When compared with the control group, breast cancer patients had higher median ages and BMI indices, both of which were statistically significant ($p < 0.001$). Also, in serum lipid level comparisons between groups, in comparison to the control group, median TG and LDL-C indices in breast cancer patients were higher, while TG was increased by 0.36 mmol/L ($p < 0.001$). Median TC and HDL-C indices were lower, and HDL-C was decreased by 0.25 mmol/L ($p < 0.001$). The levels of TG and HDL-C were statistically significant. There were no significant variations in TC ($p = 0.502 > 0.05$) and LDL-C ($p = 0.547 > 0.05$) levels between groups.

Table 1 Comparing the traits of the control and breast cancer groups

Characteristic	Breast Cancer Group M (Q1, Q3) n %	Control Group M (Q1, Q3) n %	*p* value
Age (years)	51 (45, 59)	40 (31, 52)	< 0.001
18 ~ 39	43 11.3	5709 48.2	
40 ~ 49	126 33.0	2566 21.7	
50 ~ 59	121 31.7	2181 18.4	
≥ 60	92 24.1	1386 11.7	
BMI (kg/m^2)	23.80 (21.40, 26.00)	22.44 (20.42, 24.77)	< 0.001
< 18.5	25 6.5	946 8.0	
18.5 ~ 23.9	171 44.8	7077 59.8	
24 ~ 27.9	136 35.6	2979 25.2	
≥ 28	50 13.1	840 7.1	
TG (mmol/L)	1.34 (0.93, 2.07)	0.98 (0.71, 1.42)	< 0.001
< 1.7	243 63.6	9879 83.4	
≥ 1.7	139 36.4	1963 16.6	
TC (mmol/L)	4.90 (4.29, 5.70)	4.93 (4.33, 5.61)	0.502
< 5.2	225 58.9	7177 60.6	
≥ 5.2	157 41.1	4665 39.4	
LDL-C (mmol/L)	2.92 (2.32, 3.58)	2.91 (2.40, 3.52)	0.547
< 2.6	134 35.1	4083 34.5	
2.6 ~ 3.4	123 32.2	4268 36.0	
≥ 3.4 ~ < 4.1	79 20.7	2212 18.7	
≥ 4.1	46 12.0	1279 10.8	
HDL-C (mmol/L)	1.31 (1.09, 1.57)	1.56 (1.34, 1.81)	< 0.001
≥ 1.0	319 83.5	11552 97.6	
< 1.0	63 16.5	290 2.4	

Multivariate binary logistic regression analysis was used to produce odds ratios (OR) and confidence intervals (CI) for 95% in order to describe the associations between age, BMI, TG, and HDL-C with the risk of developing breast cancer (Table 2). When the 18–39 age range was used as a reference, the risk of developing breast cancer in subjects aged 40–49 was approximately 6.52 times higher (OR = 6.52, 95% CI: 4.60–9.25, $p < 0.001$, statistically significant), the risk of developing breast cancer in subjects aged 50–59 was approximately 7.37 times higher (OR = 7.37, 95% CI: 5.18–10.47, $p < 0.001$, statistically significant), and the risk of developing breast cancer in subjects aged ≥ 60 was approximately 8.81 times higher (OR = 8.81, 95% CI: 6.11–12.72, $p < 0.001$, statistically significant).

Table 2 The odds ratio (OR) of breast cancer to age, BMI, TG, HDL-C and 95% confidence intervals (95% CI)

Characteristic	Breast Cancer Group	Control Group	OR	95%CI	*p* value
	n	n			
Age (years)					
18 ~ 39	43	5709	1.00	Reference	
40 ~ 49	126	2566	6.52	4.60 - 9.25	< 0.001
50 ~ 59	121	2181	7.37	5.18 - 10.47	< 0.001
≥ 60	92	1386	8.81	6.11 - 12.72	< 0.001
BMI (kg/m^2)					
< 18.5	25	946	1.09	0.72 - 1.67	0.68
18.5 ~ 23.9	171	7077	1.00	Reference	
24 ~ 27.9	136	2979	1.89	1.50 - 2.38	< 0.001
≥ 28	50	840	2.46	1.78 - 3.40	< 0.001
TG (mmol/L)					
< 1.7	243	9879	1.00	Reference	
≥ 1.7	139	1963	2.88	2.32 - 3.57	< 0.001
HDL-C (mmol/L)					
≥ 1.0	319	11552	1.00	Reference	
< 1.0	63	290	7.87	5.86 - 10.56	< 0.001

Table 3 Correlation analysis of age, BMI, TG, HDL-C and breast cancer occurrence

Variables	Characteristic	Correlation
Breast cancer occurrence (Yes = 1, No = 0)	Age (years)	0.130
	BMI (kg/m^2)	0.059
	TG (mmol/L)	0.096
	HDL-C (mmol/L)	− 0.116

Compared with the reference group (18.5 kg/m^2 ≤ BMI ≤ 23.9 kg/m^2), subjects with 24 kg/m^2 ≤ BMI ≤ 27.9 kg/m^2 had approximately 1.89 times the breast cancer risk (OR = 1.89, 95% CI: 1.50–2.38, $p < 0.001$, statistically significant), subjects with a BMI ≥ 28 kg/m^2 had approximately 2.46 times the breast cancer risk (OR = 2.46, 95% CI: 1.78–3.40, $p < 0.001$, statistically significant), and those with BMI < 18.5 kg/m^2 were not statistically significant (OR = 1.09, 95% CI: 0.72–1.67, $p = 0.68$).

When TG < 1.7 mmol/L was used as a reference, subjects with TG ≥ 1.7 mmol/L had an approximately 2.88 times higher breast cancer risk (OR = 2.88, 95% CI: 2.32–3.57, $p < 0.001$, statistically significant). When HDL-C ≥ 1.0 mmol/L was used as a reference, subjects with HDL-C < 1.0 mmol/L had an approximately 7.87 times higher breast cancer risk (OR = 7.87, 95%CI: 5.86–10.56, $p < 0.001$, statistically significant). Table 3 further demonstrates that age, BMI, and TG exhibit a low positive correlation with breast cancer, while HDL-C shows a low negative correlation with breast cancer, as determined by Spearman analysis.

From a box plot showing breast cancer and control group characteristics (Fig. 1), however, HDL-C and TC levels in the breast cancer group were lower than those in the control group. Age, BMI, LDL-C, and TG levels were all greater in the breast cancer group than they were in the control group. But there was no correlation between breast cancer risk and TC or LDL-C levels.

According to OR values (Table 2), created using a forest plot (Fig. 2), we showed that when age was between 18 and 39 years old (reference), the OR value was 1.00 and the breast cancer risk in women aged ≥ 40 years was increased. When BMI was between 18.5 and 23.9 kg/m^2 (reference), the OR value was 1.00 and the breast cancer risk increased with increased BMI. At TG < 1.7 mmol/L (reference), the OR value was 1.00 and subjects with TG ≥ 1.7 mmol/L had an increased breast cancer risk. When HDL-C ≥ 1.0 mmol/L was the reference, the OR value was 1.00 and those with HDL-C < 1.0 mmol/L had an increased breast cancer risk.

Discussion

Correlation studies examining serum lipids, BMI, age and breast cancer have been controversial, with no consensus on their significance. In our study, we evaluated correlations between different serum lipid subtypes, age, BMI, and breast cancer, and correlations between different levels of TG, HDL-C, age, BMI, and breast cancer. From our work, we verified the correlation between age and breast cancer. In contrast to the control group, we discovered median TG, age, BMI and LDL-C index levels were increased in breast cancer group, while median HDL-C and TC index levels were decreased. Additionally, we stratified TG, HDL-C, age, and BMI, and after multivariate binary logistic analysis, we observed the following: the breast cancer risk in women aged ≥ 40 years old was increased, and breast cancer risk increased with increased BMI and TG levels. Also, when compared with HDL-C ≥ 1.0 mmol/L, women with HDL-C < 1.0 mmol/L levels also had an increased risk of developing breast cancer.

For women, the peak age of breast cancer is gradually approaching aging. Studies have shown that the peak breast cancer population in Chinese women occurs between 50 and 54 years old, which is more than 10 years earlier when compared with Western countries [20]. In this study, the median age at which breast cancer onset was 51 years, consistent in aforementioned findings. Worldwide, the age of breast cancer onset is typically concentrated between 50 and 69 years, therefore, for women in this age range, the World Health Organization

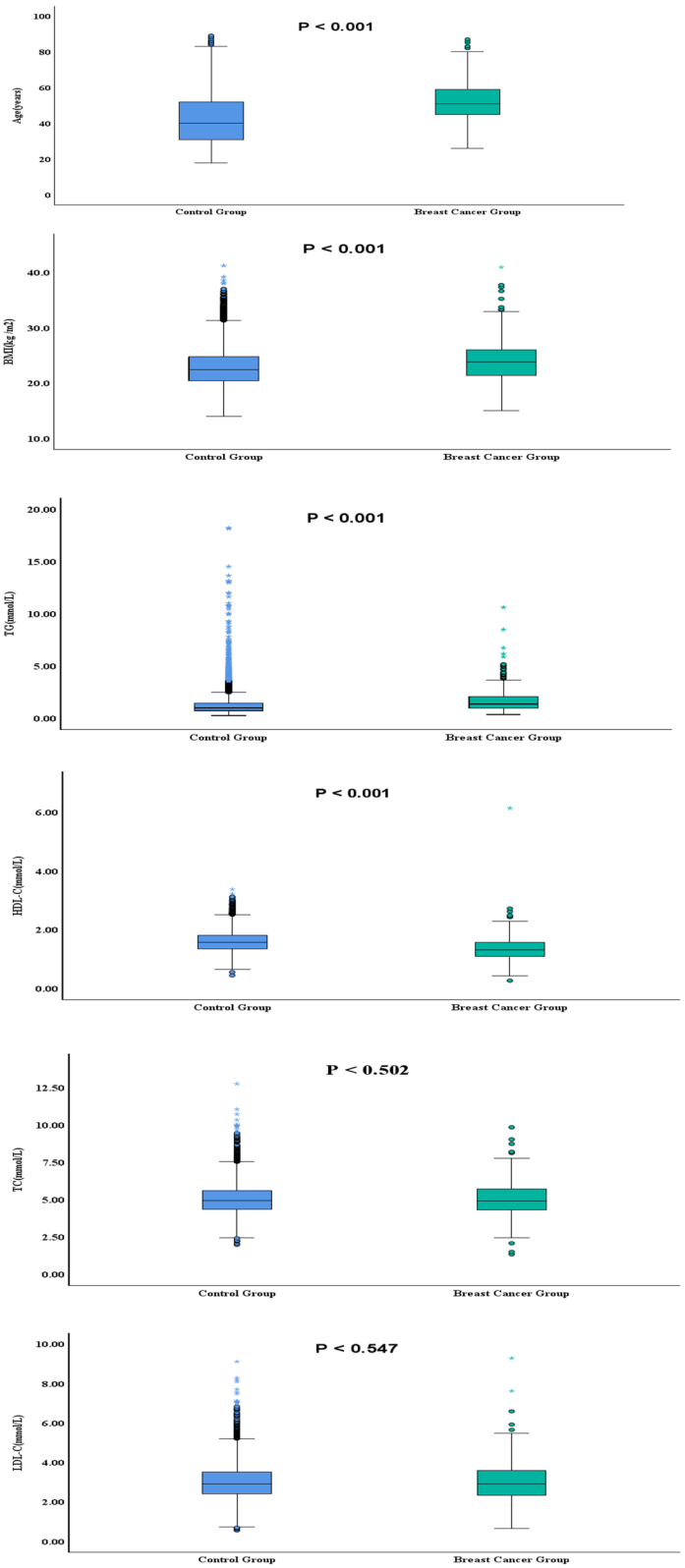

Fig. 1 Comparison box plot showing age, BMI, and serum lipids between breast cancer and control groups

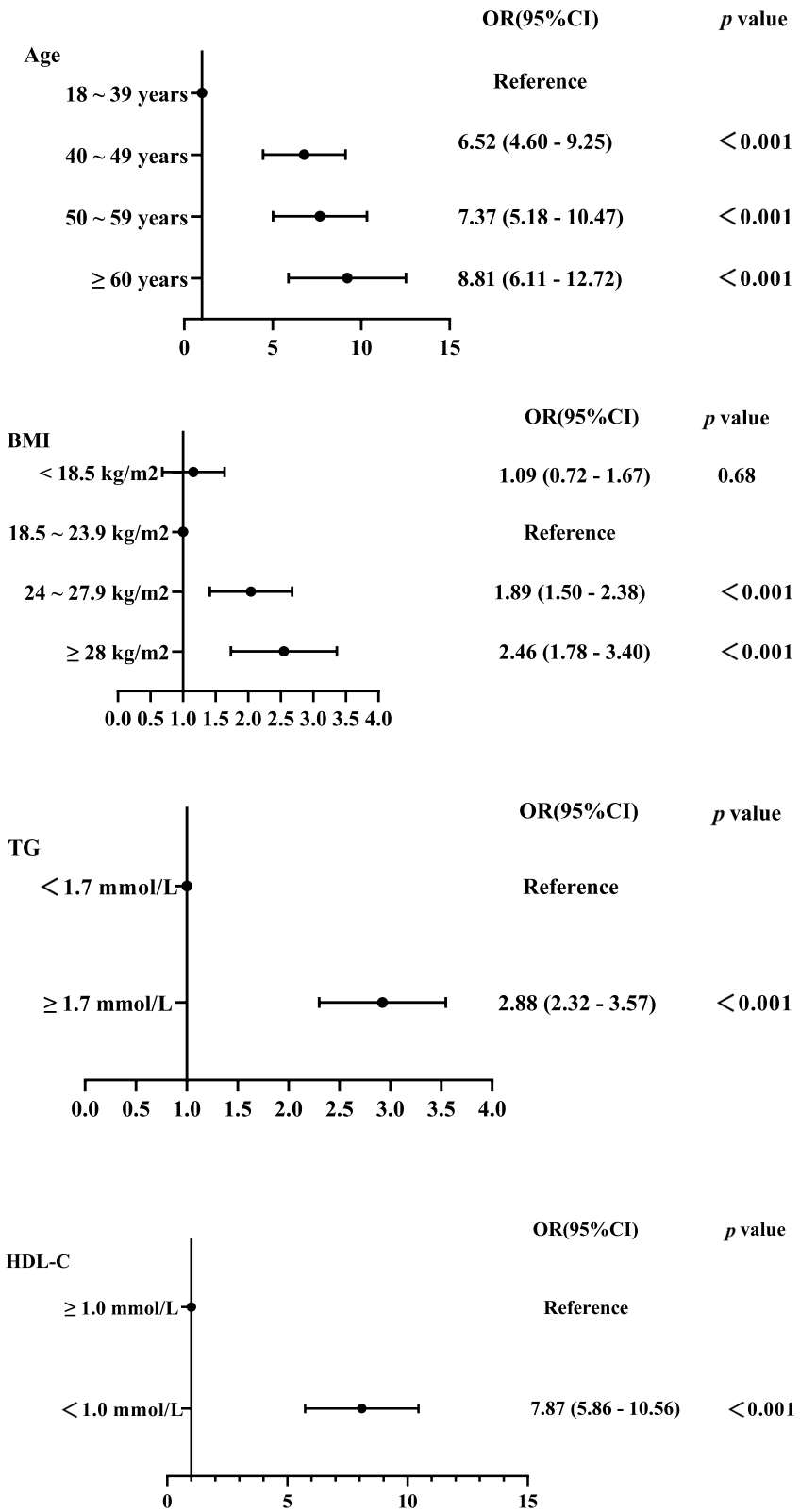

Fig. 2 Forest plot showing breast cancer odds ratios (ORs) to age, BMI, TG, and HDL-C. Note: The black dot represents the OR and the error bar represents the 95% confidence interval (95% CI)

advises mammography screening every 2 years. In China, breast cancer screening guidelines recommend screening and ultrasounds every 1–2 years for those aged 45–70 [21].

According to a previous umbrella analysis showed that obesity was linked to increased breast cancer development risk [22]. This was because adipose tissue in obese populations generates inflammatory cytokines and mediators that facilitate breast cancer progression, invasion, and metastasis by enhancing immune cell infiltration and angiogenesis [23]. In the chronic inflammatory condition of adipose tissue in obesity, pro-inflammatory proteins, including tumor necrosis factor-α (TNF-α), interleukin (IL)− 1β, interferon-γ, and transforming growth factor-β_1, inhibit the maturation of preadipocytes into adipocytes [24]. The augment angiogenesis and leptin production. Breast cancer also often expresses leptin receptors. Elevated leptin levels thus induce macrophages to generate TNF-α, fibroblast growth factor, and epidermal growth factor, thereby enhancing preadipocyte proliferation and obstructing preadipocyte maturation [23, 25]. Furthermore, macrophages secrete cytokines such as vascular endothelial growth factor, IL-6, and IL-8, which facilitate angiogenesis in the hypoxic milieu of obese adipose tissue. IL-6 additionally disrupts the equilibrium by promoting leptin synthesis and diminishing adiponectin production, thereby sustaining the chronic inflammatory state [23]. This facilitates the metastasis and advancement of breast cancer. Moreover, elevated TNF-α in obese individuals interferes with glucose and fatty acid metabolism [26], leading to insulin resistance, increased insulin and IGF-1 levels, and heightened plasma free fatty acid levels. Murphy et al. [27] found that higher IGF-1 concentrations were associated with an increased risk of breast cancer. At the 2024 UK-Breast Cancer Prevention Conference [28], most studies assessing breast cancer risk in relation to weight based their investigations on BMI and showed that whereas higher BMI was linked to lower breast cancer risk in premenopausal women, it was linked to higher breast cancer risk in postmenopausal women. However, in two meta-analyses, premenopausal breast cancer risk was positively correlated with increased BMI in Asian populations due to differences between races [29]. We also showed a statistical significance between BMI and breast cancer occurrence ($p < 0.001$), and when BMI was between 18.5 and 23.9 kg/m^2 (reference), breast cancer risk increased with increased BMI levels.

In terms of associations between serum lipids and breast cancer, a prospective study reported that HDL-C was negatively associated with breast cancer risk, while a Mendelian randomization study research revealed that there was no correlation between breast cancer risk and LDL-C [30, 31]. These results were consistent with our results. Other studies have shown that up-regulated scavenger receptor BI protein (SR-BI) is linked to a poor prognosis and accelerated breast cancer development [13]. SR-BI is the HDL receptor, which promotes cholesterol excretion from the liver; HDL-C levels are significantly reduced when SR-BI is highly expressed. This suggests that breast cancer is negatively correlated with HDL-C. These results were also confirmed by another in vitro study which showed that SR-BI deletion reduced Akt activation, thus preventing breast cancer invasion and metastasis [13]. Moreover, overall breast cancer patient survival was substantially reduced when HDL-C levels were lower. Additionally, for LDL-C, studies have reported no correlations between LDL-C and breast cancer risk [14], consistent with our results. However, a higher level of LDL-C is thought to indicate the breast cancer stage, and because it is connected to higher breast cancer histological grades and later clinical stages, it also leads to a poorer prognosis in patients; thus, an elevated LDL-C status, to some extent, is more likely to develop into lymph node metastasis [32]. Nancy et al. [33] in their case-control study reported that 83 women with breast cancer had significantly higher TG values when compared with 113 women without breast cancer. We also verified this; breast cancer risk increased with increased TG levels. Since TG is a separate source of oxidation of fatty acids, it has key roles promoting cell proliferation and tumor growth [34]. However, another study reported that HDL-C and TG were not associated with breast cancer risk [35]. Low TC has been linked in studies to a higher risk of breast cancer [36]. We found that the breast cancer group had lower TC levels than the control group, but there was no statistical significance.

Although associative mechanisms underlying serum lipid, BMI, and breast cancer processes are not fully understood, and correlations between these factors vary, they are closely related and mutually influence each other. We confirmed that age was significantly correlated with breast cancer. It also reaffirms that women aged ≥ 40 years old should maintain healthy levels of HCL-C and BMI, which is consistent with global breast screening guidelines. This study provided a new reference for early breast cancer prevention, thereby reducing breast cancer incidence and improving adverse disease outcomes in the future. Moreover, there were a lot of healthy females in our study, which strengthened data accuracy. However, there are a few limitations to consider. First off, this study did not cover significant risk variables for breast cancer, such as dietary habits, physical activity, genetic predisposition, family history, clinicopathological features, and menopausal status. Among them, menopause status

is more important, which affects the changes of estrogen level and lipid metabolism, which are related to the risk of breast cancer. Because dietary habits, physical activity, and genetic predisposition were not documented, and family history and menopausal state were partially incomplete, so they could not be adjusted in the analysis. There may be selection bias. In the future, we will reduce bias by conducting balanced grouping or stratified analysis on the aforementioned factors. In addition, clinicopathological features (TNM stage, histological grade, ER, PR, HER-2, Ki-67, etc.) of some patients were missing, so relevant features could not be further analyzed. In the future, we will gather more detailed clinicopathological features for correlation analysis with breast cancer. Secondly, the subject selection is quite simple, all sourced from the same hospital. Moreover, due to ethnic differences, BMI classification criteria and lipid level stratification criteria are different among ethnic groups, which may limit the generality of these results.

Finally, in future studies, the menopausal status of our subjects will be examined, and analyze genetic data and the association of inflammatory biomarkers (IL-6, TNF-α, and adipokines, etc.) with breast cancer. As well as more in-depth lipid detection analyses, the determination of lipoproteins and associated subclasses, and a deeper discussion on abnormal lipid metabolism in breast cancer.

Conclusions

Age, BMI, TG, and HDL-C are the main breast cancer risk factors in women. High TG and BMI were linked to increased breast cancer risk, while high HDL-C was linked to decreased breast cancer risk. Importantly, with increased HDL-C levels, breast cancer risks decreased. Women aged ≥ 40 years old had an increased breast cancer risk; with increased TG and BMI, the breast cancer risk increased. Given that the association between TC and LDL-C levels and breast cancer was not statistically significant in our findings, possibly due to the homogeneity of the demographic and inconsistent measurement standards, we may need to collect more case numbers in the future to draw meaningful conclusions.

Abbreviations

BMI	Body mass index
TG	Triglyceride
TC	Total cholesterol
LDL-C	Low-density lipoprotein-cholesterol
HDL-C	High-density lipoprotein-cholesterol
OR	Odds ratio
CI	Confidence intervals
TNF-α	Tumor necrosis factor-α
IL	Interleukin
IGF-1	Insulin-like growth factor-1

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Authors' contributions

Yvning Hu and Suoping Yang contributed to the conception and design of this study and the acquisition, and interpretation of data. Yvning Hu performed statistical analysis. The first draft of the manuscript was written by Yvning Hu. Yvning Hu and Suoping Yang critically revised the manuscript, approved the final manuscript as submitted, and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Ethical approval: This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of the Fifth Affiliated Hospital, Southern Medical University (approval number: 2024-RXK-K-001).

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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