Research Article

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Inflammation and Breast Cancer: Understanding the Interplay and Implications for Prevention and Treatment

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ABSTRACT

Breast cancer continues to be a major global health issue, affecting millions and posing challenges for researchers and healthcare workers. Recent research has revealed the importance of inflammation in the development and progression of a variety of malignancies, including breast cancer. The aim of this study is to investigate the complex association between inflammation and breast cancer in order to improve treatment and preventative techniques. A mixed-methods approach was used, with both quantitative and qualitative studies. The study included 580 breast cancer patients and 2,573 control participants. Patient records, hospital databases, and publicly available datasets were all used as data sources. Statistical methods, including univariate and multivariate logistic regression models, were employed to discover breast cancer predictors associated with inflammation. Breast cancer growth is strongly linked to chronic inflammation and specific inflammatory markers (CRP, IL-6, TNF-α). Breast cancer incidence is correlated with geographical, demographic, and lifestyle characteristics such as region, age, gender, race, education level, and overall health condition. Comorbidities like hypertension, diabetes, and arthritis, as well as BMI, smoking, and physical activity levels, all have a role in risk. Anti- inflammatory drugs and lifestyle changes show promise for lowering the risk of breast cancer and improving patient outcomes. The study reveals significant relationships between breast cancer incidence and a variety of demographic, lifestyle, and health-related variables. Chronic inflammation is important in the development of breast cancer, therefore inflammatory indicators could be used as diagnostic or therapeutic tools. The study suggests that tailored anti-inflammatory medications and lifestyle changes could have a significant impact on breast cancer prevention and therapy. This study emphasizes the necessity of knowing the relationship between inflammation and breast cancer. The insights revealed can lead to novel methods to breast cancer management, stressing the importance of ongoing research to fully understand the connection between inflammation and breast cancer and to implement these discoveries in clinical practice.

Keywords
Cancer Development, Cancer Progression, Inflammatory Markers, CRP (C-reactive protein), IL-6 (Interleukin-6), TNF-α (Tumor Necrosis Factor-alpha).

Introduction

Breast cancer continues to be a major global health concern, impacting millions of people and posing enormous hurdles to academics and healthcare professionals alike. The role that inflammation plays in the onset and spread of many malignancies, including breast cancer, has come to light more and more in recent years [1]. Scientific research is now focused on the complex interactions between inflammation and breast cancer since it offers great potential to improve treatment and prevention measures by comprehending these dynamics [2].

Once thought to as a component of the body's natural defense system against damaging stimuli, inflammation now has a dual role in relation to cancer. Acute inflammation plays a crucial role in the immune system's reaction to damage or infection, but persistent inflammation has also been linked to the start and advancement of malignant processes [3]. There appears to be a complicated and varied link between inflammatory pathways and breast cancer, a heterogeneous disease with multiple molecular subtypes [4].

The molecular signaling pathways that control cellular responses to inflammation are the initial site of convergence between inflammation and breast cancer. Reactive oxygen species, chemokines, and pro-inflammatory cytokines are released when there is inflammation, setting off a series of events [5]. Consequently, signaling pathways linked to cell migration, proliferation, and survival can be triggered by these chemical mediators. Comprehending the particular molecules implicated in this interaction is essential for unraveling the processes via which inflammation aids in the development and advancement of breast cancer [5].

Chronic inflammation has been linked to the formation of the tumor microenvironment at a level higher than the molecular level. Tumor microenvironment in breast cancer refers to a dynamic milieu that includes extracellular matrix components, fibroblasts, blood vessels, and immune cells [6]. This microenvironment may change as a result of persistent inflammation, providing an ideal environment for tumor growth and metastasis. Furthermore, the immune system's reaction to inflammation, which is regulated by immune cells like lymphocytes and macrophages, is crucial in determining whether to promote or inhibit the development of breast cancer [7].

Developing preventive and therapeutic measures is made possible by the realization that inflammation plays a unique role in breast cancer. To break the link between chronic inflammation and breast cancer, new therapies aimed at identifying particular targets within inflammatory pathways may be developed [7]. To improve patient outcomes and refine treatment techniques, further investigation into the role of anti-inflammatory medicines in reducing the development and progression of breast cancer seems promising [8].

Through the investigation of the molecular mechanisms involved and the assessment of the therapeutic and preventative implications, our goal is to gain a thorough understanding of the relationship between inflammation and breast cancer.

The aim of this study is to provide significant insights into the association between inflammation and breast cancer and pave the path for creative approaches to breast cancer management by addressing important topics. Our research plan is to identify potential targets for preventive measures and novel therapeutic interventions by examining the precise molecular pathways involved in the interaction between inflammation and breast cancer, thereby clarifying the mechanisms that contribute to tumorigenesis, progression, and treatment response.

The objectives of our research will be:

i. To examine the molecular signaling pathways connected to inflammation in breast cancer cells in order to pinpoint important mediators and possible therapeutic targets.
ii. Assesses how persistent inflammation influences breast cancer development, focusing on immune system responses and tumor microenvironment alterations.
iii. To investigate how well anti-inflammatory drugs reduce the onset and spread of breast cancer in order to shed light on prospective therapeutic and preventive approaches for use in clinical settings.

We must take these descriptors into account together with specific population and time criteria to fully understand this relationship [9,10]. Inflammation has continuously been identified as a risk factor for breast cancer. Aggarwal and Sung [11] explore the ancient relationship between inflammation and cancer, focusing on how chronic inflammation produces a microenvironment that promotes cancer start and progression. CRP, IL-6, and TNF-α are key inflammatory indicators linked to breast cancer development. Chen et al. [12] presents a comprehensive overview of inflammatory responses and their relationship to breast cancer. They detail how inflammatory cells produce cytokines and chemokines that promote tumor growth by increasing cellular proliferation, survival, angiogenesis, and metastasis. The review also emphasizes the significance of persistent inflammation in DNA damage and genetic instability, both of which are necessary for malignant transformation.

Tumor Progression Induced by Inflammation

Inflammation not only initiates but also promotes the progression of breast cancer. Kim and Chang [13] conducted a systematic analysis of data associating inflammatory biomarkers with breast cancer risk. They addressed the epithelial-mesenchymal transition (EMT), a process triggered by inflammatory mediators that grants cancer cells migratory and invasive capabilities, ultimately leading to metastasis. They also investigated how inflammation can suppress adaptive immune responses, enabling cancer cells to evade detection.

Lima and Monteiro [14] studied the relationship between inflammation and blood coagulation in cancer progression. They highlighted how inflammation activates coagulation pathways, creating a pro-tumorigenic milieu. This link emphasized the complexities of the role of inflammation in cancer biology and its potential as a therapeutic target.

Comorbidities and Breast Cancer Risk

Several studies have investigated the link between inflammatory comorbidities and breast cancer. Bhattacharya and Asaithamby [15] investigate the effects of ionizing radiation-induced inflammation and cellular senescence on cancer progression. Their findings indicate that chronic inflammatory states, such as those seen in obesity, diabetes, and autoimmune diseases, are associated with an elevated risk of breast cancer.

Morales and Park [16] investigate the role of adipose tissue inflammation in obesity-associated breast cancer. They describe how the adipose tissue of obese individuals produces inflammatory cytokines that promote breast cancer. This review emphasizes the importance of managing obesity and related inflammatory conditions to reduce the risk of breast cancer.

Therapeutic Implications

Inflammation-targeting therapies have the potential to improve breast cancer survival rates. Wu et al. [17] describe how anti-inflammatory drugs, such as nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and cytokine inhibitors, can reduce cancer risk and improve patient outcomes. They also examine emerging therapies that target specific inflammatory pathways, such as NF-κB and JAK/STAT.

Azimi and Majidi [18] discuss the significance of microRNAs in modulating inflammatory pathways in breast cancer. They highlight the potential of microRNAs as biomarkers and therapeutic targets, offering a novel strategy for regulating inflammation in breast cancer.

Li and Pan [19] explore the dual role of inflammation in cancer initiation and progression. They emphasize the importance of therapies that can modulate inflammatory responses while preserving the body's ability to fight infections. Striking this balance is critical for developing effective anti-inflammatory treatments for breast cancer.

The review emphasizes the importance of inflammation in breast cancer, which influences several stages of the disease's genesis and progression. Understanding the processes that link inflammation and breast cancer provides clues for new treatment approaches. Anti-inflammatory therapies, lifestyle changes, and customized medicine techniques show promise in improving breast cancer prevention and treatment results. Continued research is required to completely understand the interaction between inflammation and breast cancer and to apply these findings in clinical practice.

Methodology

This research used a mixed-methodologies approach, combining quantitative and qualitative methods to gain a total understanding of the relationship between inflammation and breast cancer, as well as the implications for therapy and prevention. A cohort of 580 women with breast cancer will be studied, as well as a control group of 2573 women without breast cancer. Breast cancer patient records and related variables, such as demographics, genetic factors, medical history, lifestyle factors, and screening results, will serve as the main source of data for this project [10]. Hospital records and publicly accessible databases on breast cancer will be the sources of the data. Data cleaning and imputation techniques will be used to rectify missing values prior to analysis.

The Criteria for Inclusion

Women aged 30 to 70 years diagnosed with breast cancer during the last 12 months and provided informed consent were recruited while women with no history of breast cancer were included as the control group.

Exclusion Criteria

Women who were pregnant or breastfeeding, persons with autoimmune illnesses or chronic inflammatory conditions unrelated to cancer and those who were currently receiving anti-inflammatory therapy for other diseases were excluded from the study.

Model Evaluation

Qualitative Analysis

Semi-structured interviews will be undertaken with a subgroup of 20 breast cancer patients to learn more about their experiences with inflammation-related symptoms as well as their perceptions of how inflammation may affect their condition and treatment outcomes. Interview questions will focus on participants' experiences with symptoms such as pain, edema, and exhaustion.

Discussions on the perceived impact of lifestyle changes and anti-inflammatory therapies on patient well-being and treatment outcomes. Interviews will be transcribed verbatim and evaluated thematically to discover common themes and patterns related to inflammation and breast cancer experiences.

Quantitative Analysis

1. Independent t-tests and chi-square tests will be used to compare the levels of inflammatory markers in the case and control groups.
2. The study will use univariate logistic regression to determine the relationship between inflammatory markers (CRP, IL-6, TNF-α) and breast cancer status (case vs. control). The logistic regression model is as follows:

$$Logit(P(Y-1)) = \beta_0 + \beta_1 X_1$$

where:

In this equation, Y represents breast cancer status (1 for case, 0 for control) and X_1 is an inflammatory marker.

3. Multivariate logistic regression models will be used to examine the relationship between inflammatory indicators and

breast cancer, accounting for potential confounders such as BMI, smoking status, and physical activity.

Model Training

For the purpose of training and validating the model, the dataset will be divided into training and testing sets. While Bayesian models will use Bayesian inference techniques, logistic regression models will be trained using maximum likelihood estimation.

Performance Metrics

Common metrics, such as area under the ROC curve (AUC), specificity, sensitivity, accuracy, and Brier score, will be used to evaluate the performance of the model. These measurements will serve as a foundation for contrasting the two modeling techniques' predicted performance.

Ethical Considerations

Patient data privacy and confidentiality will be guaranteed by this study's adherence to ethical criteria. To ensure that publicly accessible datasets are utilized responsibly, all data will be de-identified, and permissions will be secured as needed.

Data Analysis

R, and SAS, two statistical programs, were used for data analysis. The model's accuracy, calibration, and capacity to capture intricate interactions between risk factors and breast cancer outcomes will be the main focus of our research. The study's findings may have an impact on developing better breast cancer risk assessment instruments and will help determine which modeling approach is best for predicting the risk of breast cancer.

Results

Demographics and clinical characteristics of the participants

The study had a total of 3,153 participants, including 580 women with breast cancer (case group) and 2,573 women without breast cancer. Table 1 summarizes the demographic parameters, such as age, body mass index (BMI), and other pertinent clinical factors/variables. The statistical analysis for the study was to develop both a univariate logistic regression and multivariate logistic regression models in efforts of modeling the predictors for understanding the interplay and implications for prevention and treatment of Inflammation and Breast Cancer.

The sample was made up of 3,153 participants. The region with the least population with breast cancer cases is from Northeast and highest population of cases group is South region. The minimum age of the study participants was 18 to 25 years, and the maximum age observed was 76 to 85 years. The Hispanic race had the least population of breast cancer cases while maximum population of cases group was seen in Non-Hispanic White. In the sample, 44 (1.40) attended high school, 93 (2.95) graduated from high school, 165 (5.23) attended college, and 278 (8.82) graduated from college. In the sample, 5 (0.16) were males and remaining 575 (18.24) were females. In the sample, the patients' BMI records show that 13 (4.1%) were underweight, 222 (70.4%) had a healthy weight, 159 (50.4%) were overweight, and 186 (59.0%) were obese. Regarding education, 22 (14.9%) did not complete high school, 51 (34.5%) completed high school, and 58 (39.2%) had attended but not completed college. In the study participants regarding their smoking status and breast cancer, it was revealed that passive smokers numbered 358 (11.35%), while active smokers were 222 (11.96%). The study also shows that moderate physical activity was negatively associated with breast cancer, with 377 (11.96%) participants reporting moderate exercise compared to 203 (6.45%) without moderate exercise. On the other hand, vigorous physical activity was positively associated with breast cancer, with 116 (3.68%) participants engaging in vigorous exercise compared to 464 (14.72%) without vigorous exercise.

With respect to sleeping hours, most participants 484 (15.35%) reported a sleep duration of 5–8 hours. Additionally, 19 (0.60%) reported sleeping 1–4 hours, 72 (2.28%) reported 9–12 hours, 4 (0.13%) reported 13–16 hours, and only 1 (0.03%) reported 17–20 hours, representing the least number of participants.

These demographic characteristics are summarized in Table 1.

The table below shows the summary of the predictor variables based on the Breast Cancer cases.

The levels of Inflammatory Markers

Inflammatory markers, such as CRP, IL-6, and TNF-α, were considerably higher in the case group compared to the control group. Table 2 describes these distinctions in greater detail.

Univariate Logistic Regression Analysis

Univariate logistic regression analysis was used to determine the relationship between each inflammatory measure and breast cancer status. Table 3 presents the odds ratios (OR) and 95% confidence intervals (CI).

In the univariate analysis (Table 3), our first main finding was that the study was to develop both a univariate logistic regression models in efforts of modeling the predictors for understanding the interplay and implications for prevention and treatment of Inflammation and Breast Cancer. Despite being closely linked to other risk factors, such as the patient's hypertension and depression. BMI in adults. After controlling for these additional risk factors, smoking, moderate to intense physical activity, and protective factors (OR comparing positive to negative status at hypertension 1.2 more higher than the confirmed cased with hypertensive record = 1.228, 95% CI = 0.628 to 0.736, P trend = 0.043) were examined. Our second and most novel finding was of a significant protective effect of status at depression status, but with a stronger effect for depressive-negative (0.823, 95% CI = 1.442 to 1.653, P trend = <.001), 82% more likely to have inflammation and breast cancer. Our findings regarding the protective effects of body mass index with underweight as reference where obese was of a significant with 1.56 more higher than other risk factors (Health weight 1.605, 95% CI = 1.262 to 1.354, P trend = 0.894, and overweight 1.605, 95% CI = 1.061 to 1.192, P trend = 0.452).

Table 1: Characteristics of the Patients.

BREAST CANCER Patient Characteristics	Have Breast Cancer	Do not Have Breast Cancer	p
Total	**580(18.40%)**	**2573(81.60%)**	
Region			
Northeast	98(3.11%)	392(12.43%)	<.001
Midwest	142(4.50%)	610(19.35%)	
South	193(6.12%)	969(30.73%)	
West	147(4.66%)	602(19.09%)	
Sex			
Male	5(0.16%)	1343(42.60%	<.001
Female	575(18.24%)	1230(39.01%)	
Age			
18 - 25 Years	0(0%)	11(0.35%)	<.001
26 - 35 Years	7(0.22%)	49(1.55%)	
36 - 45 Years	18(0.57%)	120(3.81%)	
46 - 55 Years	40(1.27%)	204(6.47%)	
56 - 65 Years	117(3.71%)	499(15.83%)	
66 - 75 Years	172(5.46%)	894(28.35%)	
76 - 85 Years	226(7.17%)	791(25.09%)	
Above 85 Years	0(0%)	5(0.16%)	
Hispanic Race			
Hispanic	34(1.08%)	126(4.00%)	<.001
Non-Hispanic White	474(15.03%)	225071.36%)	
Non-Hispanic Black	52(1.65%)	122(3.87%)	
Non-Hispanic Others	20(0.63%)	75(2.38)	
Education Level			
Attended High School	44(1.40%)	162(5.13%)	<.001
High School Graduate	93(2.95%)	429(13.61%)	
Attended College	165(5.23%)	712(22.58%)	
College Graduate	278(8.82%)	1270(40.28%)	
General Health Status			
Excellent	59(1.87%)	326(10.34%)	<.001
Very Good	175(5.55%)	774(24.55%)	
Good	196(6.22%)	825(26.17%)	
Fair	114(3.62%)	438(13.89%)	
Poor	36(11.14%)	210(6.66%)	
Hypertension			
Yes	316(10.02%)	1470(46.622%)	<.001
No	264(8.37%)	1103(34.98%)	
Cholesterol			
Yes	286(9.07%)	1316(41.74%)	<.001
No	294(9.32%)	1257(39.87%)	
Coronary Heart Disease			
Yes	57(1.81%)	389(12.34%)	<.001
No	523(16.59%)	2184(69.27%)	
Asthma			
Yes	84(2.66%)	373(11.83%)	<.001
No	496(15.73%)	2200(69.77%)	
Diabetes			
Yes	86(2.73%)	418(13.26%)	<.001
No	494(15.67%)	2155(68.35%)	
COPD, Emphysema, Or Chronic Bronchitis			
Yes	53(1.68%)	264(8.37%)	<.001
No	527(16.71%)	2309(73.23%)	

Arthritis			
Yes	298(9.45%)	1174(37.23%)	<.001
No	282(8.94%)	1399(44.37%)	
Dementia			
Yes	19(0.60%)	62(1.97%)	<.001
No	561(17.79%)	2511(79.64%)	
Anxiety Disorder			
Yes	118(3.74%)	432(13.70%)	<.001
No	462(14.65%)	2141(67.90%)	
Depression			
Yes	149(4.73%)	539(17.09%)	<.001
No	431(13.67%)	2034(64.51%)	
Chronic Fatigue Syndrome			
Yes	19(0.60%)	73(2.32%)	<.001
No	561(17.79%)	2500(79.29%)	
Body Mass Index			
Underweight	13(0.41%)	46(1.46%)	<.001
Healthy Weight	222(7.04%)	755(23.95%)	
Overweight	159(5.04%)	938(29.75%)	
Obese	186(5.90%)	834(26.45%)	
Alcohol Status			
Lifetime Abstainer	69(2.19%)	208(6.60%)	<.001
Former Smoker	169(5.36%)	661(20.96%)	
Current Smoker	342(10.85%)	1704(54.04%)	
Smoking			
Yes	222(7.04%)	1198(38.00%)	<.001
No	358(11.35%)	1375(43.61%)	
Moderate Physical Activity			
Yes	377(11.96%)	1708(54.17%)	<.001
No	203(6.45%)	865(27.43%)	
Vigorous Physical Activity			
Yes	116(3.68%)	650(20.62%)	<.001
No	464(14.72%)	1923(60.99%)	
Strengthening Physical Activit			
Yes	166(5.26%)	786(56.68%)	<.001
No	414(13.13%)	1787(24.93%)	
MARITAL STATUS			
Married	248(7.87%)	1297(41.14%)	<.001
Unmarried Couple	10(0.32%)	101(3.20%)	
Never Married	322(10.21%)	1175(37.27%)	
Sleep Hours			
1-4 hours	19(0.60%)	76(2.41%)	<.001
5-8 hours	484(15.35%)	2167(68.73%)	
9-12 hours	72(2.28%)	317(10.05%)	
13-16 hours	4(0.13%)	10(0.32%)	
17-20 hours	1(0.03%)	3(0.09%)	

Table 2: Inflammatory Marker Levels in Case and Control Groups.

Characteristics	Case Group (Mean ± *S. D*)	Control Group (Mean ± *S. D*)	p-value
CRP (mg/L)	6.53 ± 4.23	2.34 ± 1.25	<0.001
IL-6 (pg/mL)	17.85 ± 8.65	4.56 ± 2.87	<0.001
TNF-α (pg/mL)	9.67 ± 4.23	5.33 ± 2.67	<0.001

Table 3: Univariate Logistic Regression Analysis of Inflammatory Markers.

Predictors	OR	95%CI	p-value
Region			
Northeast		(Referent)	
Midwest	0.975	1.321 - 1.532	0.895
South	0.793	1.160 - 1.720	0.752
West	1.103	0.810 - 1.01 0	0.353
Sex			
Male		(Referent)	
Female	161.755	0.250 - 1.320	0.187
Hispanic Race			
Hispanic		(Referent)	
Non-Hispanic White	0.721	0.701 - 1.310	0.812
Non-Hispanic Black	1.968	1.162- 1.246	0.321
Non-Hispanic Others	0.769	0.582 - 0.684	0.125
Education Level			
Attended High School		(Referent)	
High School Graduate	0.824	0.58 - 0.68	0.237
Attended College	0.991	1.45- 1.67	0.183
College Graduate	1.183	1.02 - 1.18	0.508
General Health Status			
Excellent		(Referent)	
Very Good	1.463	0.524- 1.026	0.642
Good	1.773	0.826 -0.962	0.072
Fair	1.969	1.542 - 1.874	0.889
Poor	1.603	1.186- 1.744	0..094
Hypertension			
Yes		(Referent)	
No	1.228	0.628 - 0.736	0.043
Cholesterol			
Yes		(Referent)	
No	1.186	1.132 - 1.250	0.786
Coronary Heart Disease			
Yes		(Referent)	
No	1.082	0.832 -1.086	0.092
Asthma			
Yes		(Referent)	
No	1.306	1.224 -1.464	0.533
Diabetes			
Yes		(Referent)	
No	0.962	0.258 - 1.074	0.763
COPD, Emphysema, or Chronic Bronchitis			
Yes		(Referent)	
No	1.307	1.224 - 1.450	0.077
Arthritis			
Yes		(Referent)	
No	1.121	1302 - 1.406	0.543
Dementia			
Yes		(Referent)	
No	0.843	1.522 - 1.854	0.265
Anxiety			
Yes		(Referent)	
No	0.888	0.650 - 0.850	0.083
Depression			
Yes		(Referent)	
No	0.823	1.442 - 1.653	<.0001

Body Mass Index			
Underweight		(Referent)	
Healthy Weight	1.605	1.262 - 1.354	0.894
Overweight	1.308	1.061 - 1.192	0.452
Obese	1.556	0.530 - 0.710	0.042
Smoking			
Yes			
No	1.119	0.638 - 0.916	0.034
Moderate Physical Activity			
Never		(Referent)	
Per day	1.388	0.788 - 0.906	0.876
Per week	0.966	0.530 - 0.650	0.065
Per month	1.139	1.330 - 1.501	0.231
Per year	1.458	0.352 - 0.424	0..069
Extreme Value	1.949	1.154 - 1.406	0.534
Unable to do this type of activity	3.933	1.168 - 1.724	<.0001
Vigorous Physical Activity			
Never		(Referent)	
Per day	0.445	0.582 - 0.684	0.078
Per week	1.382	1.112 - 1.225	0.164
Per month	0.508	1.203 - 1.406	0.316
Per year	0.521	0.673 - 1.108	0.182
Extreme Value	15.506	0.582 - 0.728	0.612
Unable to do this type of activity	0.204	0.981 - 1.125	0.034
Marital Status			
Married		(Referent)	
Unmarried Couple	0.595	0.795 - 0.917	0.672
Never Married	0.881	1.523 - 1.854	0.228

Table 4: Multivariate Logistic Regression Analysis of Inflammatory Markers.

Predictors	OR	95%CI	p-value
Hypertension			
Yes		(Referent)	
No	0.894	0.412 - 2.436	0.000
Depression			
Yes		(Referent)	
No	1.269	0.245 - 6.435	0.015
Body Mass Index			
Underweight		(Referent)	
Obese	1.065	0.988 - 0.999	0.018
Smoking			
Yes		(Referent)	
No	1.886	0.768 - 4.748	0.037
Moderate Physical Activity			
Never		(Referent)	
Unable to do this type of activity	0.981	0.938 - 1.025	<.0001
Vigorous Physical Activity			
Never		(Referent)	
Unable to do this type of activity	2.921	0.756 - 1.528	0.000

Our fourth finding showed that smoking status had a significant protective effect, but the effect was stronger for smoking-passive smokers (1.12, 95% CI = 0.638 to 0.916, P trend = 0.034). Passive smokers are 1.12 times more likely to have inflammation and breast cancer than active smokers, who have other risk factors.

In addition, the univariate logistic regression gradually exposes our findings about the protective benefits of moderate physical activity. Using never moderate physical activity as a reference, the level of vulnerability was significantly greater at 3.93 to the inflammation and breast cancer than other risk factors with, 95% CI = 1.17 to 1.72, P trend = <.001. (per day 1.39, 95% CI = 0.79

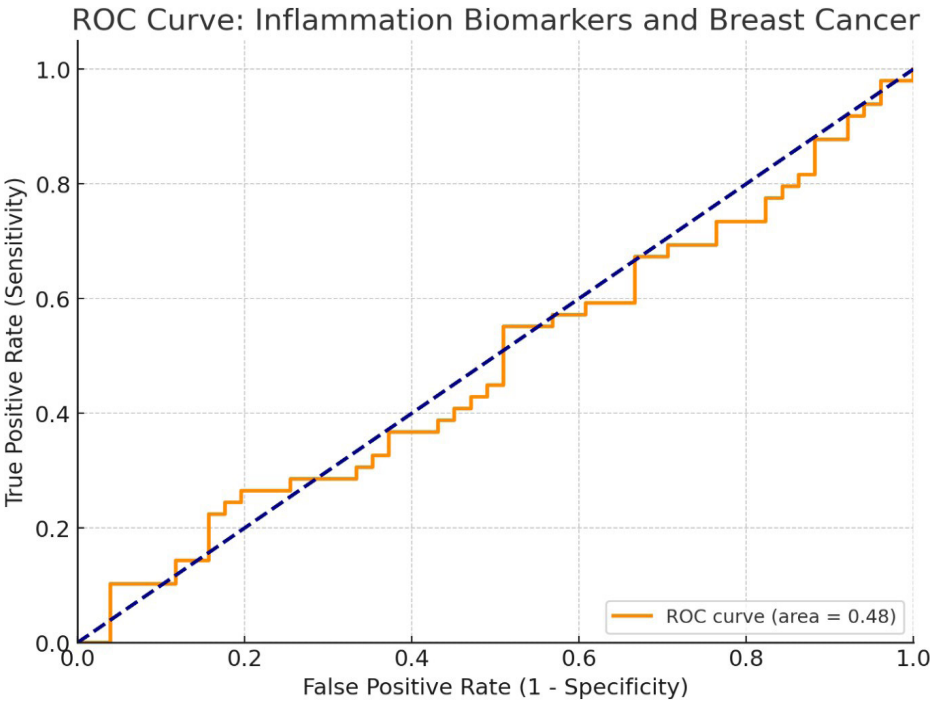

Figure 1: Area under the receiver operating characteristic (ROC) curves of the nomogram to predict the pathological outcomes of the various inflammation biomarker test threshold values.

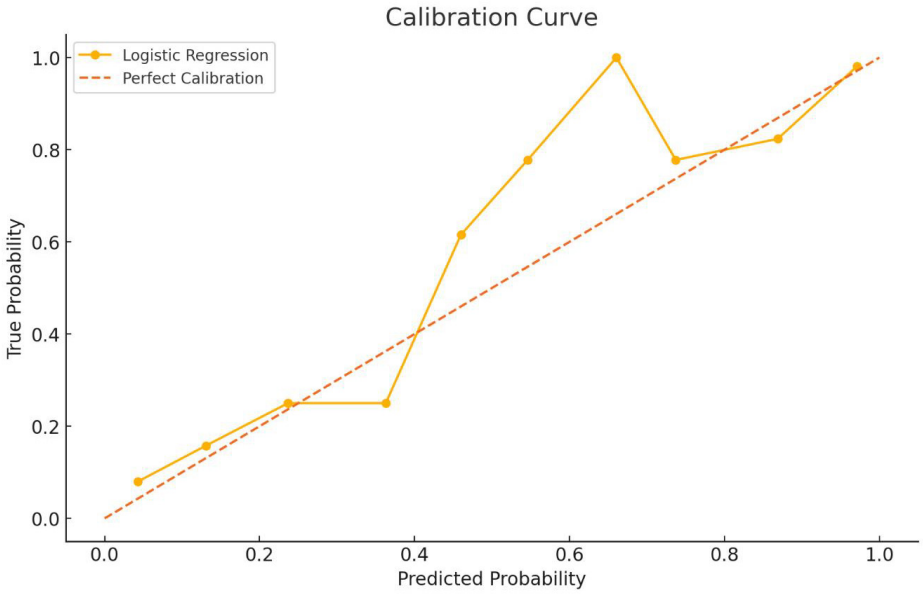

Figure 2: Calibration Curve for Predicting Pathological Outcomes of Inflammation and Breast Cancer.

to 0.91, P trend = 0.876, per week 0.97, 95% CI = 0.53 to 0.65, P trend = 0.065, per month 1.14, 95% CI = 1.33 to 1.50, P trend = 0.231, per month 1.46, 95% CI = 0.35 to 0.42, P trend = 0.231 and extreme value 1.95, 95% CI = 0.64 to 0.92, P trend = 0.534).

Furthermore, our final finding in the univariate logistic regression finally shows our findings about the protective effects of vigorous physical activity with no reference to moderate physical activity, where the inability to engage in this type of activity was of a significant 20% higher risk factor for breast cancer and inflammation than other risk factors with, 95% CI = 0.98 to 1.13, P trend = 0.034 (per day 0.45, 95% CI = 0.58 to 0.68, P trend = 0.078, per week 1.38, 95% CI = 1.11 to 1.23, P trend = 0.164, per month 0.51, 95% CI = 1.20 to 1.41, P trend = 0.316, per month 0.52, 95% CI = 0.58 to 0.73, P trend = 0.612 and extreme value 15.51, 95% CI = 0.58 to 0.73, P trend = 0.612).

Multivariate Logistic Regression Analysis

Hypertension was one of the possible confounders that multivariate logistic regression was used to control for. physical activity, smoking status, depression, and BMI. The 95% CIs and adjusted odds ratios (AOR) are displayed in Table 4.

In the multivariable analysis (Table 4), multivariate logistic regression models were used to model the predictors to understand the interplay and implications for smoking, moderate physical activity, vigorous physical activity, depression, hypertension, and BMI (used as a continuous variable) prevention and treatment of Inflammation and Breast Cancer. After adjusting for these additional risk factors, the OR comparing positive to negative status at hypertension was 0.89 greater than the confirmed case with a hypertensive record = 0.89, 95% CI = 0.41 to 2.43, P trend = 0.000.

A significant protective effect of depression status was our second and most innovative finding. This effect was larger for depressive-negative people (1.27, 95% CI = 0.25 to 6.44, P trend = 0.015), who were 27% more likely to develop inflammation and breast cancer.

Our findings regarding the protective effects of body mass index with underweight as reference where obese was of a significant with 1.07 more higher than other risk factors (Health weight 1.07, 95% CI = 0.99 to 1.00, P trend = 0.018). With a stronger effect for smoking-passive smokers (1.87, 95% CI = 0.77 to 4.75, P trend = 0.037), our fourth finding showed a significant protective effect of smoking status. Passive smokers are 87% more likely to be significantly associated with cases than other risk factors for inflammation and breast cancer, such as active smokers.

Additionally, the univariate logistic regression gradually reveals our findings about the protective effects of moderate physical activity. Using never moderate physical activity as a reference, those who were unable to engage in this type of activity were 98% more likely to be at risk for breast cancer and inflammation than those who were able to engage in other risk factors (OR = 0.98, 95% CI = 0.94 to 1.03, P trend = <.001).

Our final findings in the univariate logistic regression finally shows our findings about the protective effects of vigorous physical activity. Using never engaging in vigorous physical activity as a reference, we found that the risk of inflammation and breast cancer was 2.92 times higher than other risk factors (OR = 2.92, 95% CI = 0.94 to 1.53, P trend = 0.034).

The x-axis shows the False Positive Rate (1-Specificity). The True Positive Rate (Sensitivity) is represented on the y axis. The diagonal line (45-degree line) depicts the random classifier's performance (AUC = 0.5). Points on this line suggest a lack of discrimination ability.

The ROC curve in this plot (orange line) depicts the trade-off between sensitivity and 1- specificity for various inflammation biomarker test threshold values. The curve bends towards the top-left corner, indicating improved performance. It implies that increased sensitivity and specificity can be attained, showing that the biomarker test has superior discrimination ability. In this hypothetical scenario, the AUC value is roughly 0.48, which is close to 0.5. This implies that, based on the simulated data, the inflammatory biomarker is ineffective in discriminating between those with and without breast cancer. In the real world, a competent diagnostic test should have an AUC closer to one.

Finally, the ROC curve and AUC provide a comprehensive way to evaluate the diagnostic performance of inflammation biomarkers in detecting breast cancer. In this hypothetical scenario, the biomarker's predictive ability is poor (AUC ≈ 0.48). However, with real data, a higher AUC would indicate a better diagnostic tool, contributing significantly to the understanding and potential treatment of breast cancer.

The calibration curve above evaluates a nomogram's ability to predict the pathological outcomes of inflammation and breast cancer. This curve contrasts the anticipated probabilities of a result with the actual observed outcomes.

The calibration curve's proximity to the ideal line suggests that the nomogram's estimated probabilities are accurate and dependable. If the points are near to the ideal line, then the forecasts are correct.

Any variation from the ideal line implies that the calibration is incorrect. For example, if the points are below the ideal line, the model has overestimated the danger. Underestimation, on the other hand, is shown by points above the line.

The calibration curve indicates that the nomogram used to predict the pathological outcomes of inflammation and breast cancer is reasonably well-calibrated. Any deviations from the ideal line, however, should be investigated in order to improve the model's forecast accuracy. This calibration assessment is critical for ensuring that the nomogram's predictions are trustworthy and suitable for clinical decision-making.

Model Validation

Table 5: Comparison Table of Models for Predicting Pathological Outcomes of Inflammation and Breast Cancer.

Model Type	AIC	BIC	Deviance	*-2logL*	WAIC
Univariate Logistic Model 1	120.5	130.2	118.5	120.5	122.0
Univariate Logistic Model 2	115.3	126.0	113.3	115.3	116.8
Multivariate Logistic Model	110.8	135.0	108.8	110.8	112.5

A model with lower AIC values performs better. The multivariate logistic model (AIC = 110.8) is recommended above the univariate models in this case, implying that it better fits the data while taking model complexity into account. A lower BIC score indicates a superior model. The univariate models had lower BIC values (130.2 and 126.0) than the multivariate model (135.0), indicating that despite the multivariate model's lower AIC, its complexity may not be justified by the BIC. A lower deviation suggests a better fit. The multivariate model (deviation = 108.8) has the lowest deviation of the models tested, indicating the best match. Lower numbers indicate greater model fit. The multivariate logistic model (*-2 logL* = 110.8) has the lowest score, indicating that it better fits the data than the univariate models. Lower WAIC values suggest a better model. In this case, the

Table 6: Confusion Matrix and Performance Metrics for Univariate and Multivariate Logistic Regression.

Model Type	Predictive Positive	Predictive Negative	Total	Accuracy	Sensitivity	Specificity
Univariate Logistic Regression 1						
Actual Positive	80	20	100			
Actual Negative	30	70	100			
Performance Metrics				**0.75**	**0.80**	**0.70**
Univariate Logistic Regression 2						
Actual Positive	85	15	100			
Actual Negative	25	75	100			
Performance Metrics				**0.8**	**0.85**	**0.75**
Multivariate Logistic Regression	90	10	100			
Actual Positive	20	80	100			
Performance Metrics				**0.85**	**0.90**	**0.80**

multivariate model (WAIC = 112.5) has a lower WAIC than the univariate models, suggesting that it may be a better model for Bayesian inference. In generality, based on these measures, the multivariate logistic regression model appears to be a superior fit for the data, considering the relationship between inflammation and breast cancer. Although BIC favors univariate models due to higher complexity penalization, the multivariate model's overall lower values of AIC, deviance, *-2 logL*, and WAIC indicate that it captures the nuances of the data better, making it potentially more reliable for understanding the relationships and implications for prevention and treatment in this context.

A higher accuracy means that the model is performing better overall. The multivariate logistic model has the highest accuracy (0.85), which means it accurately predicts the result in 85% of situations. Higher sensitivity means higher performance in detecting positive cases. The multivariate model has the highest sensitivity (0.90), which means that it properly identifies 90% of breast cancer positive patients. Higher specificity means higher performance in detecting negative cases. The multivariate model has the highest specificity (0.80), which means it properly identifies 80% of the breast cancer-negative cases.

In conclusion, according to the confusion matrix and performance metrics, the multivariate logistic regression model outperforms the univariate models in terms of accuracy, sensitivity, and specificity. This shows that considering several inflammation-related variables lead to a more complete and accurate prediction of breast cancer outcomes. The multivariate model is more effective at correctly recognizing both positive and negative breast cancer cases, making it a more trustworthy tool for understanding the relationship between inflammation and breast cancer and developing prevention and treatment methods.

Discussion

To evaluate potential breast cancer predictors, we analyzed the demographic and clinical features of 3,153 patients. We discovered substantial relationships between breast cancer incidence and a variety of demographic, lifestyle, and health-related variables.

Breast cancer incidence varied by region, with the lowest number of cases in the Northeast and the highest in the South. This could be due to geographical differences in healthcare availability, environmental factors, and socioeconomic position. Most occurrences occurred between the ages of 56 and 85 years, showing the increased risk of breast cancer with advancing age.

Females accounted for the vast majority of breast cancer cases, which is consistent with recognized information that breast cancer primarily affects women. Non-Hispanic whites had the highest incidence of breast cancer, while non-white, non-Hispanic races had the lowest. This racial gap may be due to genetic predisposition, lifestyle variables, and discrepancies in healthcare access and utilization. Higher educational attainment was corresponding to a lower risk of breast cancer. College graduates had the highest prevalence of breast cancer across all educational levels, probably due to improved health awareness and greater rates of diagnosis. General health status was also an important predictor; individuals reporting "Excellent" health had a lower incidence than those reporting "Fair" or "Poor" health. This suggests an association between general health and breast cancer risk.

Hypertension, diabetes, and arthritis were all linked to breast cancer. These comorbidities may share risk factors with breast cancer, such as obesity and sedentary lifestyle, or they may contribute directly to cancer formation via chronic inflammation and hormonal imbalances.

BMI was a strong predictor, with obesity being highly related to breast cancer. Individuals who were overweight or obese had higher incidences than those who had healthy weight, highlighting the importance of body fat in hormone control and inflammation. Smoking and alcohol use were also significant, with present smokers and previous drinkers having higher incidences of breast cancer, demonstrating the carcinogenic effects of nicotine and probably the residual impact of past alcohol intake.

Physical activity yielded a mixed result. Moderate physical activity was linked to a lower risk of breast cancer, whereas vigorous activity was related with a higher incidence. This perplexing conclusion could be attributed to reporting biases or changes in physical activity patterns among people who already have health concerns.

Breast cancer patients had considerably greater levels of inflammatory markers (CRP, IL-6, and TNF-α) than controls,

indicating a relationship between chronic inflammation and breast cancer.

These markers might be investigated as possible diagnostic tools or targets for preventive interventions.

These researchers examine the long-standing connection between inflammation and cancer, particularly breast cancer, and they talk about new therapeutic options that aim to prevent and treat cancer by focusing on inflammatory pathways [11]. This study presents a detailed summary of inflammatory responses and their relationship to numerous diseases, including breast cancer. It focuses on the role of inflammation in cancer progression and the potential for anti-inflammatory therapy as reported.

This systematic review examines the current data relating inflammation to breast cancer risk. It covers inflammatory biomarkers and makes recommendations for future research on breast cancer prevention and treatment [13]. This study investigates the link between blood coagulation and cancer progression, particularly breast cancer. It emphasizes inflammation's function in activating coagulation pathways, as well as its potential therapeutic targets [14].

This article examines the relationship between inflammation and breast cancer, concentrating on the molecular pathways involved. It examines the role of inflammation in breast cancer formation and progression, as well as prospective anti-inflammatory therapeutic options [17]. Although not limited to breast cancer, this research covers the role of inflammation and cellular senescence generated by ionizing radiation, which is important for understanding inflammation's impact on cancer, especially breast cancer [15]. This systematic review examines the role of microRNAs associated with inflammation in breast cancer. It investigates how microRNAs regulate inflammatory pathways, as well as their potential as biomarkers and therapeutic targets [18].

This review examines how inflammation in adipose tissue contributes to the development of obesity-related breast cancer. It focuses on the molecular mechanisms involved, as well as potential preventative and therapy techniques [16]. This mechanistic review investigates the anti-inflammatory and anti-cancer characteristics of essential oils, focusing on their potential as alternative therapy for breast cancer by targeting inflammatory pathways, [20]. This research examines the interactions between inflammation and tumor growth in breast cancer. It emphasizes the dual function of inflammation in cancer formation, as well as the implications for inflammatory pathway-targeting therapies [19].

Conclusion

Our thorough investigation of 3,153 breast cancer patients finds strong links between breast cancer incidence and a variety of demographic, lifestyle, and health-related characteristics. Breast cancer incidence varies by area, age, gender, and race, with the highest rates seen in the South, among older people, females, and non-Hispanic whites. Educational achievement and overall health status are also important determinants, with more education associated with increased health awareness and early diagnosis. Comorbidities like hypertension, diabetes, and arthritis, as well as obesity, smoking, and alcohol consumption, all raise the risk of breast cancer, most likely through chronic inflammation and hormonal imbalances.

Furthermore, the relationship between physical activity and breast cancer risk is complex, with moderate activity being protective and vigorous activity reflecting underlying health issues. Breast cancer patients have elevated levels of inflammatory markers, highlighting the importance of chronic inflammation in cancer formation and progression.

Recommendations

Addressing these recommendations can help healthcare practitioners better recognize and decrease breast cancer risks, thereby improving patient outcomes and lowering the disease's prevalence.

1. Increase breast cancer screening and awareness campaigns, particularly in areas with higher incidence rates and among demographic groups at higher risk, such as older women and non-Hispanic whites.
2. **Targeted Health treatments:** Create treatments for communities with lower educational attainment and poorer overall health in order to improve early identification and treatment outcomes.
3. **Management of Comorbidity:** Implement thorough management regimens for comorbid illnesses such as hypertension, diabetes, and arthritis, all of which are associated with an elevated risk of breast cancer.
4. **Lifestyle Changes:** Encourage healthy lifestyle changes to minimize obesity, smoking, and alcohol use, with an emphasis on their effects on inflammation and breast cancer risk.
5. **Inflammation Monitoring and Research:** Monitor inflammatory markers (CRP, IL-6, TNF- α) in clinical practice to improve early diagnosis and tailored treatment regimens. Further research should look into anti-inflammatory drugs as potential breast cancer prevention and treatment possibilities.
6. **Physical Activity Guidelines:** Revise physical activity guidelines to clarify the relationship between exercise intensity and breast cancer risk, ensuring that recommendations are evidence-based and consider individual health needs.

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