

Correlation Between Interleukin-33 Expression and Response to Neoadjuvant Chemotherapy in Locally Advanced Breast Cancer

Rachmad Meyliandry Putra¹, Nilam Smaradhanian¹, Indra Indra¹, Andhini Laila R. Palinrungi¹, Muhammad Fakhri Rumi¹, Arham Arsyad², Muhammad Faruk¹, Salman Ardi Syamsu¹

¹Division of Surgical Oncology, Department of Surgery, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia.

²Division of Digestive Surgery, Department of Surgery, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia.

Abstract

Introduction: Locally advanced breast cancer (LABC) is associated with high morbidity. While neoadjuvant chemotherapy (NAC) is the standard of care, clinical responses vary significantly due to tumor heterogeneity. This study evaluated pre-treatment intracellular *Interleukin-33* (*IL-33*) expression as an independent predictor of NAC response in LABC patients. **Methods:** This prospective cohort study included 54 female LABC patients at Hasanuddin University Hospital. Pre-treatment *IL-33* protein expression was evaluated semiquantitatively using immunohistochemistry. All subjects received three cycles of taxane- and anthracycline-based NAC. Post-chemotherapy clinical responses were assessed using RECIST guidelines. Data were analyzed via bivariate and multiple logistic regression models. **Results:** Most subjects presented with histologic grade 3 tumors (64.8%) and the luminal molecular subtype (57.4%). Positive intracellular *IL-33* expression was detected in 40 patients (74.1%), with 33 patients (61.1%) achieving a favorable clinical response to NAC. The *IL-33* positive cohort demonstrated a significantly higher response rate compared to the negative cohort (70.0% vs. 35.7%; $p = 0.024$; OR = 4.2; 95% CI: 1.16–15.19). Multivariate modeling confirmed that positive *IL-33* expression (Adjusted OR = 5.01; 95% CI: 1.12–22.32; $p = 0.035$) and the luminal subtype (Adjusted OR = 8.73; 95% CI: 2.29–33.28; $p = 0.002$) are robust independent predictors of clinical outcomes. **Conclusion:** Pre-treatment intracellular *IL-33* expression is a significant independent predictor of NAC response in LABC. This biomarker demonstrates substantial potential in assisting patient stratification to optimize precision oncology.

Keywords: breast cancer, *Interleukin-33*, neoadjuvant chemotherapy, clinical response, immunohistochemistry

Asian Pac J Cancer Biol, 11 (4), 1037-1042

Submission Date: 07/23/2026

Acceptance Date: 09/05/2026

Introduction

Breast cancer remains a global oncological challenge, with continually rising morbidity and mortality rates, particularly in developing nations [1, 2]. Limited coverage of early screening programs frequently results in a high proportion of patients diagnosed with locally advanced breast cancer (LABC) [3, 4]. In this clinical setting, neoadjuvant chemotherapy (NAC) has been established as the primary standard of care aimed at downstaging tumor volume, facilitating optimal surgical

resection, and evaluating in vivo sensitivity to cytotoxic agents [5, 6]. Although clinical guidelines recommend standard regimens, the clinical responses exhibited by LABC patients vary considerably ranging from complete response to disease progression which ultimately impacts long-term prognosis and survival [7, 8].

This profound variability in chemotherapy response is heavily influenced by biological heterogeneity and the complexity of the tumor microenvironment (TME)

Corresponding Author:

Dr. Nilam Smaradhanian

Division of Surgical Oncology, Department of Surgery, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia.

Email: nilam.s.adil@gmail.com

[9,10]. Therefore, the identification of tissue-based predictive biomarkers is crucial for stratifying patients and guiding precision oncology strategies [11]. Currently, the role of cytokines in modulating tumor immunity and chemotherapy resistance is garnering widespread attention. *Interleukin-33* (*IL-33*), a pro-inflammatory cytokine of the *IL-1* family that functions as a cellular alarmin, is known to have pleiotropic involvement in cancer biology [12,13]. This cytokine is expressed by various epithelial and endothelial cells, and its role in tumor progression whether facilitating immune evasion or potentiating anti-tumor responses is highly dependent on the tissue context and molecular subtype profile [14,15].

Although the potential of *IL-33* as a biomarker is increasingly being explored, a comprehensive understanding of its clinical utility as a predictor of neoadjuvant response remains hindered by a significant research gap. The majority of previous studies have focused on quantifying *IL-33* at the systemic circulation or transcriptomic levels [16-19]. Measurements at the messenger RNA (mRNA) level often fail to represent the functional dynamics of the final protein due to post-transcriptional regulation within tumor cells. Furthermore, the direct correlation between intracellular *IL-33* protein expression in primary tissue prior to chemotherapy and clinical response outcomes based on the Response Evaluation Criteria in Solid Tumors (RECIST) guidelines within a homogeneous LABC population has not been adequately evaluated. The absence of this tissue-level proteomic data limits the functional translation of *IL-33* into routine diagnostic pathology practice. To bridge this gap, this study aimed to investigate the expression profile of intracellular *IL-33* protein in pre-treatment locally advanced breast carcinoma tissues and to comprehensively evaluate its predictive role regarding clinical response following NAC. This proteomic approach, utilizing immunohistochemical evaluation, is anticipated to provide a novel empirical foundation for optimizing personalized therapy, thereby reducing the morbidity burden in breast carcinoma patients.

Materials and Methods

Study Design

This analytical observational study utilized a prospective cohort design and was conducted at Hasanuddin University Hospital, Makassar, Indonesia, from July to December 2025. Immunohistochemical evaluation was centralized at the Hasanuddin University Medical Research Center (HUMRC) Laboratory. The study protocol was formally reviewed and approved by the Health Research Ethics Committee of the Faculty of Medicine, Hasanuddin University (Registration Number: 481/UN4.6.4.5.31/PP36/2025). All procedures involving human subjects were performed in accordance with the principles of the Declaration of Helsinki, and written informed consent was obtained from each participant prior to the acquisition of medical data and tissue specimens.

Population and Participants

The target population comprised all female patients diagnosed with locally advanced breast carcinoma via a triple diagnostic assessment at the study site. Sample selection was performed using consecutive sampling, with a minimum estimated sample size of 54 subjects determined by correlative analysis for categorical variables with a 5% margin of error. Inclusion criteria encompassed treatment-naïve patients diagnosed with invasive breast carcinoma (ductal or lobular histotypes) who were willing to undergo standard NAC regimens. Conversely, subjects were excluded if they presented with bilateral breast cancer, clinical or radiological evidence of distant metastasis, age exceeding 70 years, concomitant autoimmune diseases, carcinomatous ulceration or inflammation, fever or signs of systemic infection at the time of specimen collection, failure to complete the full chemotherapy cycle, or loss to follow-up prior to final evaluation.

Study Procedures

Data collection commenced with the registration of baseline demographic and clinicopathological parameters, including initial clinical tumor dimension measurements using precision calipers. All subjects subsequently received neoadjuvant therapy utilizing taxane- and anthracycline-based cytotoxic agents, administered at 21-day intervals for three complete cycles. Clinical response evaluation was conducted post-chemotherapy completion in accordance with the Response Evaluation Criteria in Solid Tumors (RECIST) guidelines. For statistical analysis purposes, therapeutic responses were dichotomized into a responsive cohort (encompassing partial or complete response) and a non-responsive cohort (comprising stable or progressive disease).

Breast carcinoma tissue specimens obtained from pre-treatment biopsies were processed to evaluate intracellular *IL-33* proteomic expression via immunohistochemical staining using a rabbit anti-*IL-33* polyclonal antibody. Tissues were serially sectioned at a thickness of 5 µm, transferred to electrostatic slides, and incubated at 65°C for 30 minutes. The procedure proceeded with deparaffinization in xylene, graded rehydration through an ethanol series, and inhibition of endogenous peroxidase activity using 3% hydrogen peroxide for 10 minutes. To minimize non-specific binding, 5% bovine serum albumin in Tris-buffered saline was utilized. Chromogenic detection was subsequently applied according to the manufacturer's protocol. Expression levels were quantified microscopically by a specialized pathologist utilizing the H-score system. Scores below 100 were classified as negative or low expression, whereas scores ranging from 100 to over 200 were categorized as positive expression, representing moderate to high staining intensity.

Statistical Analysis

Comprehensive sequential data analysis was performed using statistical software. Bivariate analysis was initially applied to evaluate the association between independent variables including the *IL-33* immunohistochemical profile and standard clinicopathological characteristics

and chemotherapy response outcomes. This was conducted using the Chi-square test, or Fisher's exact test when expected frequency assumptions were not met. Subsequently, multiple logistic regression modeling was applied to determine the independence and magnitude of the predictive role of variables that demonstrated significance in the bivariate phase. Statistical significance was universally defined as $p < 0.05$. Odds ratios (OR) and 95% confidence intervals (CI) were reported to quantify the strength of association for each predictive variable.

Results

The clinicopathological characteristics of the 54 patients with LABC included in this study were comprehensively evaluated (Table 1). The majority of patients presented with histologic grade 3 carcinoma (64.8%), the luminal molecular subtype (57.4%), premenopausal status (59.3%), and clinical lymph node involvement (68.5%). Evaluation of the clinical response following NAC, based on RECIST criteria, revealed that 33 patients (61.1%) achieved a favorable response, whereas 21 patients (38.9%) were classified as non-responsive.

Immunohistochemical evaluation confirmed positive intracellular *IL-33* protein expression in 40 patients (74.1%). The staining pattern exhibited varying intensities, ranging from low (negative) to moderate and high expression levels within the intracellular compartment of the tumor cells (Figure 1).

Bivariate analysis revealed a significant association between pre-treatment molecular profiles and post-chemotherapy clinical outcomes. Patients with positive *IL-33* expression demonstrated a substantially higher clinical response rate compared to the *IL-33* negative cohort (70.0% vs. 35.7%). This proportional difference was statistically significant ($p = 0.024$), yielding an odds ratio (OR) of 4.2 (Table 2).

Furthermore, the luminal molecular subtype exhibited a robust predictive correlation with chemotherapy response compared to the non-luminal subtype (80.6%

Table 1. Clinical and Clinicopathological Characteristics of the Study Participants

Variable	n	%
Histologic Grade		
2	19	35.2
3	35	64.8
Molecular Subtype		
Luminal	31	57.4
Non-luminal	23	42.6
Menopausal Status		
Premenopausal	32	59.3
Postmenopausal	22	40.7
Lymph Node Involvement		
Positive	37	68.5
Negative	17	31.5
<i>IL-33</i> Expression		
Positive	40	74.1
Negative	14	25.9
RECIST Response		
Responsive	33	61.1
Non-responsive	21	38.9

vs. 34.8%; $p = 0.001$). Conversely, other conventional clinicopathological parameters, including histologic grade, menopausal status, and clinical lymph node status, did not demonstrate statistically significant associations with clinical response (Table 3).

To evaluate the independence and predictive strength of the significant variables, a multiple logistic regression model was applied, incorporating molecular subtype and *IL-33* expression (Table 4). Multivariate analysis confirmed that the luminal subtype remained a strong predictor (Adjusted OR = 8.73; $p = 0.002$). Crucially, following simultaneous adjustment within the regression model, *IL-33* expression maintained its significance as an independent predictor. Patients with positive intracellular *IL-33* expression had a five-fold greater probability of

Table 2. Association between *IL-33* Expression and Clinical Response to Neoadjuvant Chemotherapy

<i>IL-33</i> Expression	Responsive n (%)	Non-responsive n (%)	p-value	OR	95% CI
Positive	28 (70.0)	12 (30.0)	0.024	4.2	1.16 – 15.19
Negative	5 (35.7)	9 (64.3)			

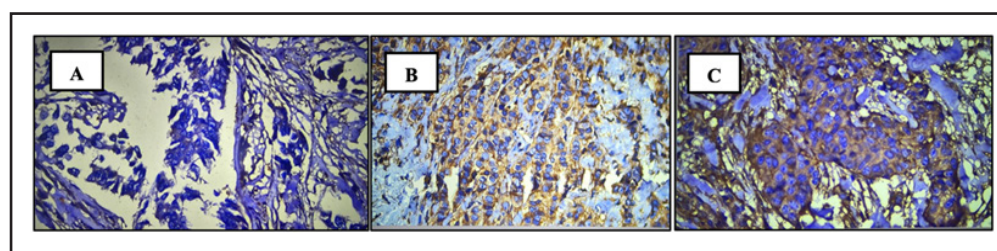


Figure 1. Representative Immunohistochemical Staining of *IL-33* Expression in Locally Advanced Breast Carcinoma Tissue. (A) Negative expression (Low Expression); (B) Moderate positive expression; (C) Strong positive expression (High Expression).

Table 3. Correlation of Patient Clinicopathological Features with Neoadjuvant Chemotherapy Outcomes

Variable		Responsive n (%)	Non-responsiven (%)	p-value	OR	95% CI
Histologic Grade						
	2	14 (73.7)	5 (26.3)	0.163	—	—
	3	19 (54.3)	16 (45.7)			
Molecular Subtype						
	Luminal	25 (80.6)	6 (19.4)	0.001	7.8	2.27 – 26.92
	Non-luminal	8 (34.8)	15 (65.2)			
Menopausal Status						
	Postmenopausal	15 (68.2)	7 (31.8)	0.377	—	—
	Premenopausal	18 (56.3)	14 (43.7)			
Lymph Node						
	Positive	20 (54.1)	17 (45.9)	0.117	—	—
	Negative	13 (76.5)	4 (23.5)			

Table 4. Multivariate Analysis of Predictive Variables for Favorable Clinical Outcomes

Variable	B	SE	p-value	Adjusted OR	95% CI
Luminal subtype	2.166	0.683	0.002	8.73	2.29 – 33.28
Positive <i>IL-33</i>	1.611	0.762	0.035	5.01	1.12 – 22.32
Constant	-5.705	1.622	<0.001	—	—

achieving a clinical response compared to the negative expression cohort (Adjusted OR = 5.01; $p = 0.035$).

Discussion

The results of this study indicate that pre-treatment intracellular *IL-33* protein expression is significantly correlated with clinical response to NAC in LABC. Within this study cohort, the *IL-33* positive expression group demonstrated a substantially higher proportion of favorable clinical responses compared to the negative expression group. Furthermore, multivariate analysis confirmed that intracellular *IL-33* status provides significant independent predictive information beyond established molecular subtype classifications, such as the luminal subtype. This pattern suggests that *IL-33* evaluation has the potential to complement current predictive biomarker panels for stratifying the probability of patient response to cytotoxic agents.

Conceptually, these empirical findings align with the role of *IL-33* as an *IL-1* family cytokine that functions as an alarmin and an immune regulator with pleiotropic properties. Current literature emphasizes that the *IL-33* and its receptor (ST2) axis does not possess a singular function in cancer pathogenesis; rather, it demonstrates a broad spectrum of effects ranging from the potentiation of anti-tumor immunity to the promotion of an immunosuppressive TME [13]. This biological variability is largely dictated by the cellular source of *IL-33*, its compartment of expression, and the composition of surrounding resident immune cells, which collectively modulate the balance of the local immune response [15].

In the specific context of breast carcinoma, evidence indicates that *IL-33* expressed by the stromal compartment, particularly by cancer-associated fibroblasts, can facilitate progression and metastasis by orchestrating a

type-2 immune microenvironment [20]. Other studies have also highlighted the involvement of the *IL-33*/ST2 axis in stemness pathways and Wnt activation, which drive cellular aggressiveness [21]. Nevertheless, the pro-tumorigenic effects reported in these studies do not contradict the findings of the present cohort, which demonstrate an association between intracellular *IL-33* and improved short-term clinical outcomes. This divergence in outcomes is highly likely influenced by the compartment of measurement namely, intracellular *IL-33* within primary tumor cells versus stromal or circulating *IL-33* which may reflect the biological susceptibility of the tumor cells themselves to chemotherapy-induced apoptosis.

In the NAC setting, *IL-33* dynamics are reported to be significantly modulated in response to cytotoxic agent exposure. Previous translational studies in breast cancer patients have shown that *IL-33* expression correlates with therapeutic outcome parameters and possesses heterogeneous prognostic value across subtypes, indicating that *IL-33* modulation reflects massive alterations in tumor biology during intervention [18]. Within this framework, the sustained significance of the luminal subtype determinant in our analysis further underscores the internal heterogeneity of luminal responses to chemotherapy, wherein a subset of luminal tumors exhibits cytotoxic sensitivity that necessitates additional complementary markers for clinical stratification [22].

This study possesses several fundamental strengths. First, the prospective cohort design enables clear temporal observation between pre-treatment biomarker expression and clinical response outcomes. Second, *IL-33* quantification was performed at the proteomic level (intracellular immunohistochemistry), representing

the functional end-product within tumor cells, thereby overcoming the limitations of prior studies that predominantly relied on transcriptomic (mRNA) profiling. Third, the stringent inclusion criteria, strictly involving locally advanced stages, minimizes confounding variables related to disparate disease burdens.

Despite these strengths, the present study is not without limitations that warrant careful consideration. The most notable constraint is the relatively small sample size ($n = 54$), which inherently limits the overall statistical power of the analysis. This restricted cohort size is directly reflected by the wide 95% confidence intervals observed in the multivariate logistic regression model particularly for the predictive values of *IL-33* expression and the luminal subtype. Such wide intervals indicate a degree of mathematical estimation instability; therefore, while the predictive association remains statistically significant, the precise magnitude of the odds ratios must be interpreted with caution. Consequently, these findings may not be broadly generalizable to more heterogeneous clinical populations without further validation. Furthermore, therapeutic evaluation in this study relies on macroscopic, lesion size-based clinical response criteria (RECIST) rather than absolute microscopic disease eradication, such as pathological Complete Response (pCR). While RECIST provides valuable early clinical assessment, its primary limitation in the neoadjuvant setting is the inability to reliably distinguish between viable residual tumor tissue and chemotherapy-induced necrosis, fibrosis, or scarring. Consequently, macroscopic clinical or radiological measurements may either underestimate or overestimate the true extent of residual disease. In contrast, pCR which evaluates the complete absence of invasive carcinoma microscopically in both the breast and axillary lymph nodes serves as the definitive gold standard and a superior surrogate endpoint for long-term disease-free and overall survival. The reliance on RECIST in our cohort may therefore not capture the full spectrum of biological efficacy and microscopic tumor clearance achieved by the chemotherapy.

In conclusion, pre-treatment intracellular *IL-33* protein expression serves as a robust independent predictor of clinical response to NAC in patients with LABC. Patients exhibiting positive *IL-33* expression demonstrate a significantly higher probability of achieving a favorable clinical response; notably, this finding retains statistical significance irrespective of the underlying tumor molecular subtype. These results underscore the substantial promise of intracellular *IL-33* assessment as a complementary biomarker, facilitating clinical patient stratification and the optimization of precision oncology strategies.

To address the limitations associated with the current sample size and to externally validate these empirical findings, future investigations warrant large-scale, multicenter cohort studies. Subsequent research should concurrently evaluate ST2 receptor status and profile the surrounding tumor immune microenvironment. Furthermore, the incorporation of definitive oncological endpoints such as pCR and long-term survival metrics is

highly recommended to comprehensively establish the clinical utility of this biomarker.

Ethical Considerations

This study received formal ethical approval from the Medical Research Ethics Board of Hasanuddin University (Approval No: 481/UN4.6.4.5.31/PP36/2025). The research was conducted in compliance with the Declaration of Helsinki. Informed consent was obtained from all participants, and all personal data were anonymized to ensure strict patient confidentiality.

Acknowledgements

None.

Competing Interest

The author declares no competing interests in this study.

References

- Bray F, Laversanne M, Sung H, Ferlay J, Siegel RL, Soerjomataram I, Jemal A. Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA: A Cancer Journal for Clinicians*. 2024 05;74(3):229-263. <https://doi.org/10.3322/caac.21834>
- Giaquinto AN, Sung H, Miller KD, Kramer JL, Newman LA, Minihan A, Jemal A, Siegel RL. Breast Cancer Statistics, 2022. *CA: A Cancer Journal for Clinicians*. 2022 Nov;72(6):524-541. <https://doi.org/10.3322/caac.21754>
- Wahidin M, Febrianti R, Susanty F, Hasanah S. Twelve Years Implementation of Cervical and Breast Cancer Screening Program in Indonesia. *Asian Pacific Journal of Cancer Prevention*. 2022 03 01;23(3):829-837. <https://doi.org/10.31557/APJCP.2022.23.3.829>
- Benitez Fuentes JD, Morgan E, De Luna Aguilar A, Mafra A, Shah R, Giusti F, Vignat J, et al. Global Stage Distribution of Breast Cancer at Diagnosis: A Systematic Review and Meta-Analysis. *JAMA Oncology*. 2024 01 01;10(1):71. <https://doi.org/10.1001/jamaoncol.2023.4837>
- Korde LA, Somerfield MR, Carey LA, Crews JR, Denduluri N, Hwang ES, Khan SA, et al. Neoadjuvant Chemotherapy, Endocrine Therapy, and Targeted Therapy for Breast Cancer: ASCO Guideline. *Journal of Clinical Oncology*. 2021 05 01;39(13):1485-1505. <https://doi.org/10.1200/JCO.20.03399>
- Aebi S, Karlsson P, Wapnir IL. Locally advanced breast cancer. *The Breast*. 2022 03;62:S58-S62. <https://doi.org/10.1016/j.breast.2021.12.011>
- Pandurangappa V, Paruthy SB, Jamwal R, Singh A, Tanwar S, Kumar D, Pal S, et al. Assessment of Response to Neoadjuvant Chemotherapy in Locally Advanced Breast Carcinoma Using Image-Guided Clip Placement. *Cureus*. 2023 Oct 26;. <https://doi.org/10.7759/cureus.47763>
- Yılmaz C, Zengel B, Üreyen O, Adıbelli ZH, Taşlı F, Yılmaz HT, Özdemir Ö, et al. A Comprehensive Analysis of Neoadjuvant Chemotherapy in Breast Cancer: Adverse Events, Clinical Response Rates, and Surgical and Pathological Outcomes—Bozyaka Experience. *Cancers*. 2025 01 07;17(2):163. <https://doi.org/10.3390/cancers17020163>
- Lüönd F, Tiede S, Christofori G. Breast cancer as an example of tumour heterogeneity and tumour cell plasticity during

- malignant progression. *British Journal of Cancer*. 2021 07 20;125(2):164-175. <https://doi.org/10.1038/s41416-021-01328-7>
10. Guo L, Kong D, Liu J, Zhan L, Luo L, Zheng W, Zheng Q, Chen C, Sun S. Breast cancer heterogeneity and its implication in personalized precision therapy. *Experimental Hematology & Oncology*. 2023 01 09;12(1):3. <https://doi.org/10.1186/s40164-022-00363-1>
 11. Derouane F, Van Marcke C, Berlière M, Gerday A, Fellah L, Leconte I, Van Bockstal MR, et al. Predictive Biomarkers of Response to Neoadjuvant Chemotherapy in Breast Cancer: Current and Future Perspectives for Precision Medicine. *Cancers*. 2022 08 11;14(16):3876. <https://doi.org/10.3390/cancers14163876>
 12. Cayrol C, Girard J. *Interleukin-33 (IL-33)*: A critical review of its biology and the mechanisms involved in its release as a potent extracellular cytokine. *Cytokine*. 2022 08;156:155891. <https://doi.org/10.1016/j.cyto.2022.155891>
 13. Stojanovic B, Gajovic N, Jurisevic M, Stojanovic MD, Jovanovic M, Jovanovic I, Stojanovic BS, Milosevic B. Decoding the *IL-33*/ST2 Axis: Its Impact on the Immune Landscape of Breast Cancer. *International Journal of Molecular Sciences*. 2023 09 13;24(18):14026. <https://doi.org/10.3390/ijms241814026>
 14. Zhang X, Chen W, Zeng P, Xu J, Diao H. The Contradictory Role of *Interleukin-33* in Immune Cells and Tumor Immunity. *Cancer Management and Research*. 2020 08;Volume 12:7527-7537. <https://doi.org/10.2147/CMAR.S262745>
 15. Yeoh WJ, Vu VP, Krebs P. *IL-33* biology in cancer: An update and future perspectives. *Cytokine*. 2022 09;157:155961. <https://doi.org/10.1016/j.cyto.2022.155961>
 16. Kuncman W, Orzechowska M, Kuncman L, Kordek R, Taran K. Intertumoral Heterogeneity of Primary Breast Tumors and Synchronous Axillary Lymph Node Metastases Reflected in IHC-Assessed Expression of Routine and Nonstandard Biomarkers. *Frontiers in Oncology*. 2021 Nov 03;11:660318. <https://doi.org/10.3389/fonc.2021.660318>
 17. Goda N, Nakashima C, Nagamine I, Otagaki S. The Effect of Intratumoral Interrelation among FOXP3+ Regulatory T Cells on Treatment Response and Survival in Triple-Negative Breast Cancer. *Cancers*. 2022 04 25;14(9):2138. <https://doi.org/10.3390/cancers14092138>
 18. Albuquerque RB, Borba MASM, Fernandes MSS, Filgueira TO, Martins DBG, Filho JLL, Castoldi A, Souto FO. *Interleukin-33* Expression on Treatment Outcomes and Prognosis in Brazilian Breast Cancer Patients Undergoing Neoadjuvant Chemotherapy. *International Journal of Molecular Sciences*. 2023 Nov 15;24(22):16326. <https://doi.org/10.3390/ijms242216326>
 19. Haghbini M, Sotoodeh Jahromi A, Ranjbaran R, Abbasi M, Hashemi Tayer A. Comparison of *Interleukin-33* Serum Levels in Patients with Breast Cancer and Idiopathic Granulomatous Mastitis. *Asian Pacific Journal of Cancer Prevention*. 2023 05 01;24(5):1629-1634. <https://doi.org/10.31557/APJCP.2023.24.5.1629>
 20. Shani O, Vorobyov T, Monteran L, Lavie D, Cohen N, Raz Y, Tsarfaty G, et al. Fibroblast-Derived IL33 Facilitates Breast Cancer Metastasis by Modifying the Immune Microenvironment and Driving Type 2 Immunity. *Cancer Research*. 2020 Dec 01;80(23):5317-5329. <https://doi.org/10.1158/0008-5472.CAN-20-2116>
 21. Fan G, Zuo S, Wang Z, Zhang S, Liu L, Luo H, Xie Y, Zhang Y, Li D. Targeting of the *IL-33*/Wnt axis restricts breast cancer stemness and metastasis. *Scientific Reports*. 2025 05 25;15(1):18172. <https://doi.org/10.1038/s41598-025-03260-9>
 22. Collins PM, Brennan MJ, Elliott JA, Abd Elwahab S, Barry K, Sweeney K, Malone C, et al. Neoadjuvant chemotherapy for luminal a breast cancer: Factors predictive of histopathologic response and oncologic outcome. *The American Journal of Surgery*. 2021 08;222(2):368-376. <https://doi.org/10.1016/j.amjsurg.2020.11.053>



This work is licensed under a Creative Commons Attribution-Non Commercial 4.0 International License.