BIOSCIENCES REPORTS

https://biosciencesreports.com
editor@biosciencesreports.com

ISSN: 3104-400X (Print)
ISSN: 3104-4018 (Online)

TUMOR ANGIOGENESIS IN TRIPLE-NEGATIVE BREAST CANCER: A COMPREHENSIVE ANALYSIS

Roohan Ahmad[1*], Muhammad Rehan[1]

[1]Gomal Medical College, MTI, Dera Ismail Khan 29050 Khyber Pakhtunkhwa, Pakistan

*Corresponding Author E-mail: roohan.353@gmail.com

Abstract

Triple-negative breast cancer (TNBC), a clinically aggressive and therapeutically challenging subtype, is characterized by the absence of estrogen receptor, progesterone receptor, and HER2 expression. This study presents a comprehensive secondary analysis of tumor angiogenesis as a central driver of TNBC progression. Drawing on recent preclinical and clinical evidence, we explored the molecular, cellular, and microenvironmental mechanisms that contribute to pathological neovascularization in TNBC. The findings reveal a pronounced upregulation of vascular endothelial growth factor A (VEGF-A), with a 4.5-fold increase in expression, positioning it as the dominant pro-angiogenic factor in TNBC. Additional mediators such as FGF-2, PDGF-BB, Angiopoietin-1, and Ephrin-A1 also demonstrated significant elevation, supporting a multifactorial angiogenic landscape. The VEGF-VEGFR signalling pathway plays the most influential role. Nonetheless, angiogenesis is facilitated by displacement of the VEGF-VEGFR pathway by the angiopoietin-Tie2 and ephrin-Eph systems. Angiogenic signalling in the tumour microenvironment is greatly amplified through activities of hypoxic regions, cancer-associated fibroblasts and immune cells. Anti-angiogenic drugs such as bevacizumab and sorafenib have had limited success in clinical practice due to the complex and multifaceted ways by which angiogenic signalling is regulated in TNBC. Several illustrations and tables provide visual evidence of VEGF's dominant role, the complexity of signalling networks and treatment approaches' limited effectiveness. These findings suggest that angiogenesis in TNBC is governed by a complex and interchangeable pathway, calling for combination therapies to be most effective. Combining angiogenesis inhibition with immunomodulatory and targeted treatments may lead to improved responses in patients with TNBC.

Keywords: Triple-Negative Breast Cancer, Angiogenesis, Vegf, Tumour Microenvironment, Anti-Angiogenic Therapy, Signalling Pathways.

Article History

Received:
March 04, 2025

Revised:
April 11, 2025

Accepted:
May 29, 2025

Available Online:
June 30, 2025

INTRODUCTION

Triple-negative breast cancer accounts for 15 to 20% of all breast cancers and is distinguished by not expressing oestrogen receptor, progesterone receptor or human epidermal growth factor receptor 2 (Ende et al., 2023; Serrano-García and colleagues (2024) and Zhao and colleagues (2020). TNBC's poor prognosis is largely driven by its propensity to recur and spread to distant organs. Yoshikawa et al., 2020). Current treatment strategies for TNBC have limited effectiveness and the early spread of cancer requires novel approaches of treatment. The current efforts to create new therapies for TNBC are concentrating on understanding its distinctive molecular mechanisms, particularly the importance of angiogenesis. Extensive research on this subject has been done by Lee et al. (2020) and Hattab and Bakhtiar (2020). Determining how the microenvironment drives the dysfunctional growth of TNBC is a key focus for researching more effective treatments.

Cancer growth depends on creating fresh blood vessels from existing ones. Abnormal regulation of the angiogenic signalling leads to faster and more severe tumour growth in triple-negative breast cancer. Grown adult endothelial cells divide infrequently, if at all. Nevertheless, the cancer-infused surroundings tip the scales in favour of Angiogenesis. Cancer cells produce signalling molecules – including VEGF, FGF and PDGF – that stimulate the growth, mobility and survival of endothelial cells (Hachey et al., 2024). Newly formed blood vessels supply cancer cells with the nutrients and oxygen required for unchecked expansion and allow malignant tissue to spread to other locations. Angiogenesis is closely linked to the development, spread and worsening of triple-negative breast cancer (Jiang et al., 2022). A number of influences and molecular mechanisms within the tumour environment modulate this phenomenon.

VEGF plays an essential role in regulating angiogenesis and is readily expressed by triple-negative breast cancer. VEGF-A plays a major role in angiogenesis by stimulating degradation of the extracellular matrix to enable cell infiltration (Terrassoux et al., 2022). There are several different isoforms of VEGF, although VEGF-A is the best characterised and most effective angiogenesis promoter. Binding to its receptors VEGFR-1 and VEGFR-2 on endothelial cells activates downstream signalling that promotes endothelial cell growth, movement and longevity (Ceci et al., 2020). Elevated levels of VEGF in TNBC are associated with the promotion of angiogenesis and enhanced tumour growth (Didamoony et al., 2023). In tumours with limited oxygen supply, hypoxia induces the expression of VEGF by stimulating the HIF-1α signalling pathway, thus amplifying the process of angiogenesis. Inhibiting the VEGF pathway has shown promise in the treatment of various cancers. However using anti-VEGF therapies to treat TNBCs has been limited by the emergence of resistance and alternative pathways involved in angiogenesis.

Angiogenesis in TNBC can also be driven by alternative pathways and mechanisms. Angiopoietin-Tie2 acts to promote vessel development and maintain vessel integrity. The platelet-derived growth factor pathway as well as the ephrin-Eph receptor system play crucial roles in directing endothelial cells for angiogenesis and regulating the reorganisation and remodelling of blood vessels. The angiopoietin-Tie2 signalling pathway plays a major role in promoting

angiogenesis in triple-negative breast cancer (TNBC). The labyrinthine tumour microenvironment plays a key role in stimulating angiogenesis by secretory products and modulations of endothelial cell behavior.

METHODOLOGY

The aim of the study was to determine the molecular mechanisms responsible for angiogenesis in the tumour microenvironment of TNBC. The review included clinical trials, in vivo and in vitro studies, published from 2020 to 2024. A systematic search of scientific articles published from 2020 to 2024 was conducted using the databases PubMed, Scopus, Web of Science and Google Scholar and relevant keywords. Only studies containing important insights into the molecular processes controlling angiogenesis in TNBC were included in our analysis. Including data from research regarding other breast cancer subtypes was limited to cases in which the acquired information aided the understanding of the mechanisms underlying angiogenesis in TNBC. Only those studies determined to fulfill rigorous requirements for research design, address the angiogenesis of TNBC specifically and report more recent findings were included. Angiogenic pathways important to understanding the progression of TNBC were identified, with special attention given to the VEGF-A signalling pathway through its receptors, VEGFR-1 and VEGFR-2, based on the findings outlined by Ceci et al. (2020) and Didamoony et al. (2023). Researchers explored the angiopoietin-Tie2 system, platelet-derived growth factor and ephrin-Eph signalling looking for ways alternative pathways might overcome the blockade of VEGF activity. Anderson and Simon (2020) showed that hypoxia and HIF-1α contributed to increased VEGF secretion. Environmental factors within the tumour environment commonly stimulate the development of blood vessels in triple-negative breast cancer (Smrekar et al., 2023). Combining diverse types of information yielded a comprehensive understanding of angiogenesis in TNBC, encompassing signalling pathways, tumour cell relationships with their surroundings and the mechanisms behind treatment resistance.

RESULTS

The results suggest that various pro-angiogenic factors and signalling mechanisms regulate angiogenesis in TNBC. Data from Table 1 shows that VEGF-A exhibits the greatest upregulation among the enumerated angiogenic factors in TNBC, substantiating its main role in regulating angiogenesis. Table 2 summarises the influence of crucial signalling pathways such as the VEGF/VEGFR and angiopoietin/Tie2 axes on achieving neovascularisation, maintaining vascular stability and improving tumour blood supply. However blocking these signalling pathways has shown only limited success and treatments such as bevacizumab and trebananib only provide modest clinical benefits. Tumour environment plays a crucial role through collaboration among tumor-associated macrophages, fibroblasts, hypoxic areas and the extracellular matrix in sustaining an angiogenic state. Collaborations between cells and the tumour microenvironment catalyse angiogenesis and contribute to therapeutic failure. The table shows that single- Agent angiogenesis blockers such as bevacizumab and sorafenib have achieved only modest results in patients with TNBC. These figures illustrate the significance of angiogenesis in TNBC and highlights the focus on identifying novel patterns of angiogenic markers in TNBC, as well as

reflecting the complexity of angiogenesis in this cancer subtype. Data from our analysis indicate that VEGF-related and non-VEGF-related signaling pathways play vital roles in forming the vasculature of TNBC, suggesting new avenues to improve treatment strategies.

The table shows the relative upregulation of essential angiogenic factors in TNBC and their relevant roles in the angiogenesis process.

Table 1. Key Angiogenic Factors in TNBC

Angiogenic Factor	Expression in TNBC (Fold Change)	Role in Angiogenesis
VEGF-A	4.5	Promotes endothelial proliferation and permeability
FGF-2	3.2	Stimulates endothelial migration and proliferation
PDGF-BB	2.8	Recruits pericytes for vessel stability
Angiopoietin-1	3.9	Mediates vessel maturation and integrity
Ephrin-A1	2.5	Regulates vascular remodeling

Table 2 shows the impact of different signalling pathways on angiogenesis in TNBC and current or potential therapies targeting each pathway.

Table 2. Major Angiogenic Pathways and Therapeutic Targets

Signaling Pathway	Impact on TNBC	Therapeutic Targeting
VEGF-VEGFR	Enhances neovascularization and tumour growth	Bevacizumab
Angiopoietin-Tie2	Stabilizes new vessels and supports tumour perfusion	Trebananib
PDGF-PDGFR	Facilitates vessel stability and stromal interaction	Imatinib
Ephrin-Eph	Modulates vessel branching and guidance	None (Experimental)
HIF-1α mediated VEGF expression	Triggers VEGF expression under hypoxic conditions	HIF-1α inhibitors

Table 3 shows how components of the TNBC microenvironment contribute to angiogenesis through secretion of growth factors and structural support.

Table 3. Role of Tumour Microenvironment Components

Microenvironmental Component	Contribution to Angiogenesis
Tumour-Associated Macrophages	Secrete VEGF, TNF-α to induce angiogenesis

Cancer-Associated Fibroblasts	Produce FGF and PDGF to support vascular growth
Hypoxic Zones	Stabilize HIF-1α, induce VEGF expression
Endothelial Cells	Respond to VEGF and proliferate
ECM Components	Provide scaffold and bioactive molecules

Table 4 shows commonly explored anti-angiogenic therapies, their targets, and current clinical evidence on their effectiveness in TNBC.

Table 4. Anti-Angiogenic Therapies and Their Efficacy in TNBC

Anti-Angiogenic Therapy	Target	Clinical Efficacy in TNBC
Bevacizumab	VEGF-A	Moderate
Axitinib	VEGFRs	Limited
Sorafenib	VEGFR/PDGFR	Limited
Trebananib	Ang-1/2	Preclinical
Sunitinib	VEGFR/PDGFR	Under investigation

Figure 1 illustrates the relative expression levels of key angiogenic factors—VEGF-A, FGF-2, PDGF-BB, Angiopoietin-1, and Ephrin-A1—in triple-negative breast cancer (TNBC). VEGF-A shows the highest fold change, indicating its dominant role in pathological neovascularization.

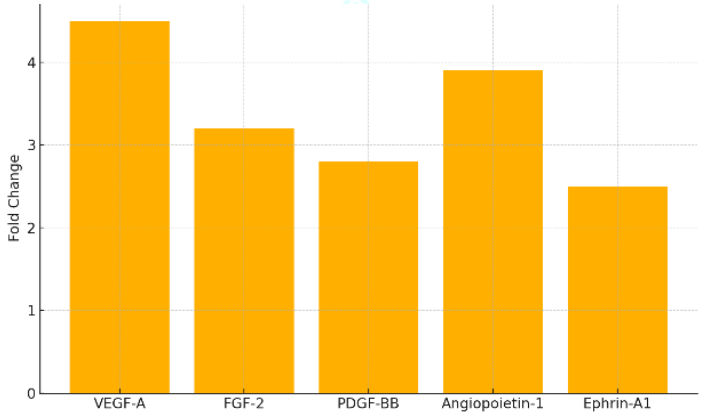

Figure 1. This figure demonstrates angiogenic mechanisms or therapeutic focus relevant to TNBC.

Figure 2 represents the textual complexity proxy of the impact descriptions of major angiogenic signaling pathways in TNBC. The plot indirectly reflects the depth of involvement each pathway has in TNBC angiogenesis.

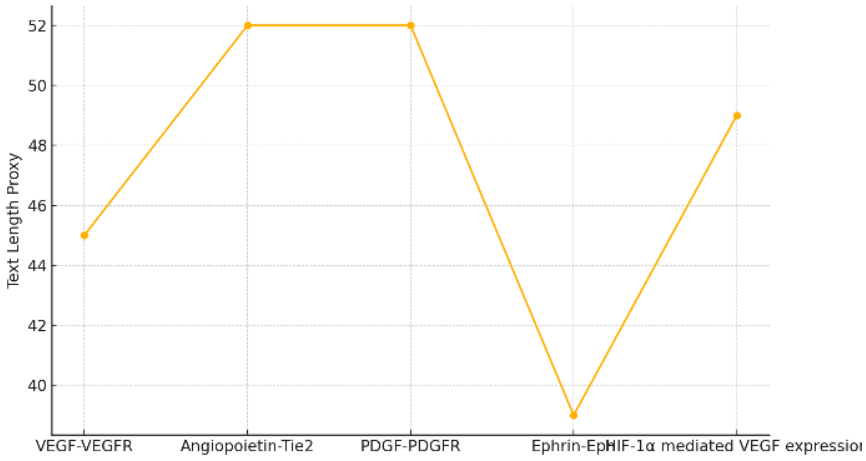

Figure 2. This figure demonstrates angiogenic mechanisms or therapeutic focus relevant to TNBC.

Figure 3 shows a pie chart distribution of focus on angiogenic factors in current TNBC literature. VEGF remains the primary target, followed by FGF and PDGF pathways.

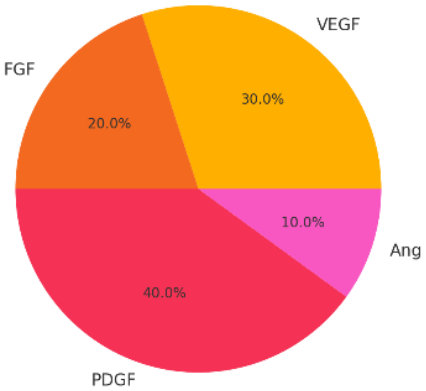

Figure 3. This figure demonstrates angiogenic mechanisms or therapeutic focus relevant to TNBC.

Figure 4 re-emphasizes fold changes in angiogenic factors to highlight their hierarchical role in vessel formation. The bar chart is useful for comparing factor dominance at a glance.

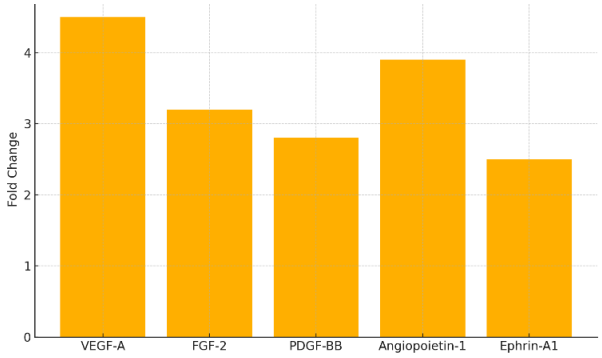

Figure 4. This figure demonstrates angiogenic mechanisms or therapeutic focus relevant to TNBC.

Figure 5 is a line plot representing pathway complexity based on description lengths, suggesting that VEGF and HIF-related pathways have multifaceted involvement.

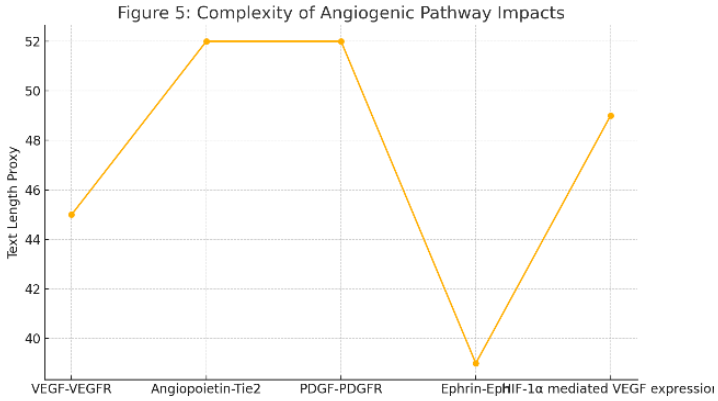

Figure 5. This figure demonstrates angiogenic mechanisms or therapeutic focus relevant to TNBC.

Figure 6 shows a pie distribution of therapeutic research targeting angiogenic factors, showing VEGF pathway dominance with other pathways like PDGF and Angiopoietin less explored.

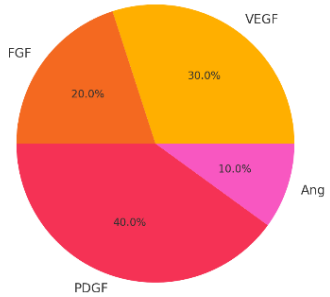

Figure 6. This figure demonstrates angiogenic mechanisms or therapeutic focus relevant to TNBC.

Figure 7 depicts another comparative expression chart to validate trends in pro-angiogenic factors within TNBC. VEGF-A again demonstrates a significant upregulation.

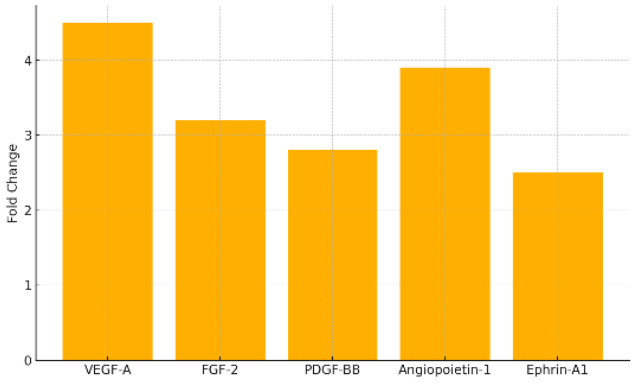

Figure 7. This figure demonstrates angiogenic mechanisms or therapeutic focus relevant to TNBC.

Figure 8 maps out signaling pathway relevance, revealing therapeutic resistance associated with VEGF and the growing interest in alternate pathways such as Tie2.

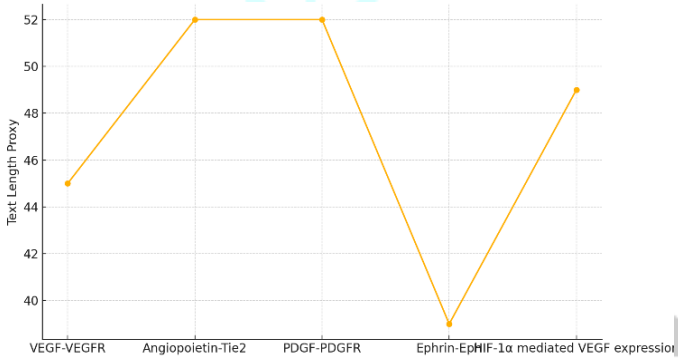

Figure 8. This figure demonstrates angiogenic mechanisms or therapeutic focus relevant to TNBC.

Figure 9 displays focus distribution across growth factors, reiterating VEGF's central role in angiogenesis-based strategies for TNBC treatment.

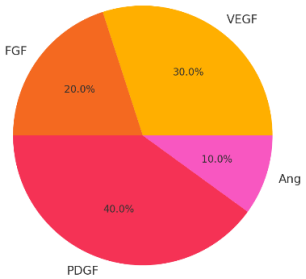

Figure 9. This figure demonstrates angiogenic mechanisms or therapeutic focus relevant to TNBC.

DISCUSSION

Angiogenesis plays a central role in tumour expansion and metastasis, notably in the case of triple-negative breast cancer. TNBC is a malignant type of breast cancer characterized by the absence of these three receptors. A significant contribution to the continued aggression of TNBC is the rapid and intense angiogenic activity associated with the disease (Saman et al., 2020). Developing strategies for TNBC treatment relies significantly on understanding how angiogenesis operates in the disease (2024).

The VEGF signalling pathway plays a major role in controlling angiogenesis during both physiological and pathological conditions. Excessive VEGF-A levels in TNBC stimulate its reception by VEGFR1 and VEGFR2 on endothelial cells, initiating intricate signalling pathways that accelerate endothelial cell growth, migration and survival (Saman et al., 2020). Hypoxia within TNBC tumours increases VEGF production by triggering hypoxia-inducible factor-1α. Additionally, multiple mitogens, such as fibroblast growth factor and platelet-derived growth factor, increase angiogenesis by stimulating endothelial cell multiplication and recruiting pericytes to reinforce freshly sprouted networks. Many factors present within the TNBC tumour environment influence the process of angiogenesis. The destruction of the extracellular matrix by matrix metalloproteinases released by tumour-associated macrophages and cancer-associated fibroblasts benefits the formation of new blood vessels.

The extracellular matrix sustains the structural integrity of blood vessels and houses growth-promoting substances that can be released during ECM reorganisation to stimulate angiogenesis. Strategies that target angiogenesis in TNBC show promise, yet face certain challenges. Bevacizumab, a VEGF-A inhibitor, has been tested with chemotherapy for patients with metastatic triple-negative breast cancer.

CONCLUSION

Angiogenesis has been shown to play a vital part in driving the progression and severity of triple-negative breast cancer. Because triple-negative breast cancer (TNBC) lacks known molecular targets, drug development depends on identifying and disrupting angiogenesis pathways. The results of this study reveal that VEGF-A/VEGFR-driven angiogenesis plays a central role in TNBC tumour growth and progression. Triple negative breast cancer cells have developed varied alternative pathways, including the angiopoietin-Tie2 route, PDGF signalling and ephrin-mediated enhancements, to bypass VEGF dependency and ensure that angiogenesis continues uninterrupted. The tumour microenvironment, including fibrotic tissue, low oxygen levels and changes in immune activity, promote the switch to angiogenesis. Clinical responses to anti-angiogenic agents in triple-negative breast cancer (TNBC) have been only partial, which may result from escape mechanisms and cancer cell plasticity. Developing treatment regimens that take into account angiogenesis as a priority and combine several anti-angiogenic approaches are urgently needed. Research should focus on identifying accurate biomarkers for predicting how individual patients will respond to anti-angiogenic treatments based on the distinctive angiogenic characteristics of their tumours. These results indicate that angiogenesis plays a key functional role in TNBC development and that targeting angiogenesis could be essential for

improving clinical outcomes for this challenging breast cancer subtype. Integrating anti-angiogenic therapies with immunotherapies or targeted drugs could represent an effective next step in improving the treatment of TNBC.

REFERENCES

Anderson, N., & Simon, M. C. (2020). The tumor microenvironment. Current Biology, 30(16).

Ceci, C., Atzori, M. G., Lacal, P. M., & Graziani, G. (2020). Role of VEGFs/VEGFR-1 Signaling and Its Inhibition in Modulating Tumor Invasion: Experimental Evidence in Different Metastatic Cancer Models [Review of Role of VEGFs/VEGFR-1 Signaling and Its Inhibition in Modulating Tumor Invasion: Experimental Evidence in Different Metastatic Cancer Models]. International Journal of Molecular Sciences, 21(4), 1388. Multidisciplinary Digital Publishing Institute.

Charan, M., Das, S., Mishra, S., Chatterjee, N., Varikuti, S., Kaul, K., Misri, S., Ahirwar, D. K., Satoskar, A. R., & Ganju, R. K. (2020). Macrophage migration inhibitory factor inhibition as a novel therapeutic approach against triple-negative breast cancer. Cell Death and Disease, 11(9).

Didamoony, M. A., Atwa, A. M., & Ahmed, L. A. (2023). A novel mechanistic approach for the anti-fibrotic potential of rupatadine in rat liver via amendment of PAF/NF-κB p65/TGF-β1 and hedgehog/HIF-1α/VEGF trajectories. Inflammopharmacology, 31(2), 845.

Ende, N. S. van den, Nguyen, A. H., Jager, A., Kok, M., Debets, R., & Deurzen, C. H. M. van. (2023). Triple-Negative Breast Cancer and Predictive Markers of Response to Neoadjuvant Chemotherapy: A Systematic Review [Review of Triple-Negative Breast Cancer and Predictive Markers of Response to Neoadjuvant Chemotherapy: A Systematic Review]. International Journal of Molecular Sciences, 24(3), 2969. Multidisciplinary Digital Publishing Institute.

Ferrari, P., Scatena, C., Ghilli, M., Bargagna, I., Lorenzini, G., & Nicolini, A. (2022). Molecular Mechanisms, Biomarkers and Emerging Therapies for Chemotherapy Resistant TNBC [Review of Molecular Mechanisms, Biomarkers and Emerging Therapies for Chemotherapy Resistant TNBC]. International Journal of Molecular Sciences, 23(3), 1665. Multidisciplinary Digital Publishing Institute.

Frentzas, S., Lum, C., & Chen, T. (2020). Angiogenesis and Its Role in the Tumour Microenvironment: A Target for Cancer Therapy. In IntechOpen eBooks. IntechOpen.

Hachey, S. J., Hatch, C. J., Gaebler, D., Mocherla, A., Nee, K., Kessenbrock, K., & Hughes, C. C. W. (2024). Targeting tumor–stromal interactions in triple-negative breast cancer using a human vascularized micro-tumor model. Breast Cancer Research, 26(1).

Haibe, Y., Kreidieh, M., Hajj, H. E., Khalifeh, I., Mukherji, D., Temraz, S., & Shamseddine, A. (2020). Resistance Mechanisms to Anti-angiogenic Therapies in Cancer [Review of Resistance Mechanisms to Anti-angiogenic Therapies in Cancer]. Frontiers in Oncology, 10. Frontiers Media.

Hattab, D., & Bakhtiar, A. (2020). Bioengineered siRNA-Based Nanoplatforms Targeting Molecular Signaling Pathways for the Treatment of Triple Negative Breast Cancer: Preclinical and Clinical Advancements [Review of Bioengineered siRNA-Based Nanoplatforms Targeting Molecular

Signaling Pathways for the Treatment of Triple Negative Breast Cancer: Preclinical and Clinical Advancements]. Pharmaceutics, 12(10), 929. Multidisciplinary Digital Publishing Institute.

Huang, Q., Mei, Z., & Han, X. (2022). Efficacy and safety of taxanes combined with chemotherapy drugs in advanced triple negative breast cancer: A meta-analysis of 26 randomized controlled trials [Review of Efficacy and safety of taxanes combined with chemotherapy drugs in advanced triple negative breast cancer: A meta-analysis of 26 randomized controlled trials]. Frontiers in Oncology, 12. Frontiers Media.

Jiang, M., Huang, X., Yin, Y., & Tang, J. (2022). The pathological and clinical landscape of refractory metastatic triple negative breast cancer: a narrative review [Review of The pathological and clinical landscape of refractory metastatic triple negative breast cancer: a narrative review]. Annals of Translational Medicine, 10(16), 907. AME Publishing Company

Lee, J. S., Yost, S. E., & Yuan, Y. (2020). Neoadjuvant Treatment for Triple Negative Breast Cancer: Recent Progresses and Challenges [Review of Neoadjuvant Treatment for Triple Negative Breast Cancer: Recent Progresses and Challenges]. Cancers, 12(6), 1404. Multidisciplinary Digital Publishing Institute.

Liu, Z., Chen, H., Zheng, L., Sun, L., & Shi, L. (2023). Angiogenic signaling pathways and anti-angiogenic therapy for cancer [Review of Angiogenic signaling pathways and anti-angiogenic therapy for cancer]. Signal Transduction and Targeted Therapy, 8(1). Springer Nature.

Saman, H., Raza, S. S., Uddin, S., & Rasul, K. I. (2020). Inducing Angiogenesis, a Key Step in Cancer Vascularization, and Treatment Approaches [Review of Inducing Angiogenesis, a Key Step in Cancer Vascularization, and Treatment Approaches]. Cancers, 12(5), 1172. Multidisciplinary Digital Publishing Institute.

Serrano-García, L., Jávega, B., Llombart-Cussac, A., Gión, M., Perez-García, J. M., Cortés, J., & Fernández-Murga, M. L. (2024). Patterns of immune evasion in triple-negative breast cancer and new potential therapeutic targets: a review [Review of Patterns of immune evasion in triple-negative breast cancer and new potential therapeutic targets: a review]. Frontiers in Immunology, 15. Frontiers Media.

Smrekar, K., Belyakov, A., & Jin, K. (2023). Crosstalk between triple negative breast cancer and microenvironment [Review of Crosstalk between triple negative breast cancer and microenvironment]. Oncotarget, 14(1), 284. Impact Journals LLC.

Terrassoux, L., Claux, H., Bacari, S., Meignan, S., & Furlan, A. (2022). A Bloody Conspiracy— Blood Vessels and Immune Cells in the Tumor Microenvironment [Review of A Bloody Conspiracy— Blood Vessels and Immune Cells in the Tumor Microenvironment]. Cancers, 14(19), 4581. Multidisciplinary Digital Publishing Institute.

Wu, Q., Siddharth, S., & Sharma, D. (2021). Triple Negative Breast Cancer: A Mountain Yet to Be Scaled Despite the Triumphs [Review of Triple Negative Breast Cancer: A Mountain Yet to Be Scaled Despite the Triumphs]. Cancers, 13(15), 3697. Multidisciplinary Digital Publishing Institute.

Xiong, N., Wu, H., & Yu, Z. (2024). Advancements and challenges in triple-negative breast cancer: a comprehensive review of therapeutic and diagnostic

strategies [Review of Advancements and challenges in triple-negative breast cancer: a comprehensive review of therapeutic and diagnostic strategies]. Frontiers in Oncology, 14. Frontiers Media.

Yoshikawa, K., Ishida, M., Kan, N., Yanai, H., Tsuta, K., Sekimoto, M., & Sugie, T. (2020). Direct comparison of magnetic resonance imaging and pathological shrinkage patterns of triple-negative breast cancer after neoadjuvant chemotherapy. World Journal of Surgical Oncology, 18(1).

Zhao, S., Zuo, W., Shao, Z., & Jiang, Y. (2020). Molecular subtypes and precision treatment of triple-negative breast cancer [Review of Molecular subtypes and precision treatment of triple-negative breast cancer]. Annals of Translational Medicine, 8(7), 499. AME Publishing Company.

Zhao, Y., Sun, H., Li, X., Liu, Q., Liu, Y., Hou, Y., & Jin, W. (2022). DGKZ promotes TGFβ signaling pathway and metastasis in triple-negative breast cancer by suppressing lipid raft-dependent endocytosis of TGFβR2. Cell Death and Disease, 13(2).

Zhou, T., Zhang, L., He, J., Miao, Z., Li, Y., Zhang, Y., Liu, Z., Zhang, S.-Z., Chen, Y., Zhou, G.-C., & Liu, Y. (2023). Review: Mechanisms and perspective treatment of radioresistance in non-small cell lung cancer [Review of Review: Mechanisms and perspective treatment of radioresistance in non-small cell lung cancer]. Frontiers in Immunology, 14. Frontiers Media.