

## LETTER to the EDITOR

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# Mitigating Chemotherapy Toxicities in Breast Cancer: The Potential of Arglabin Therapy

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### Dear Editor

#### *Ethical consideration*

IRB approval was not required because no data was collected for this article.

Breast cancer, a serious malignancy in women, is highly heterogeneous and categorized into subtypes based on the immune-histochemical markers of hormone receptors. These subtypes are divided into four categories: estrogen receptor-positive (ER+), progesterone receptor-positive (PR+), human epidermal growth factor receptor 2-positive (HER2+), and triple-negative breast cancer (TNBC) [1]. The incidence of breast cancer is increasing by approximately 1% annually. This increase is more pronounced in women under 50 years old (1.4% per year) compared to those aged 50 and older (0.7% per year). Among racial groups, White women exhibit a significant upward trend; however, the highest annual increases are observed in Asian American/Pacific Islander women, with rates of 2.7% for those under 50 and 2.5% for those aged 50 and older [2]. Around 15–20% of breast cancer cases are categorized as triple-negative breast cancer (TNBC), a category marked by tumors that lack the display estrogen receptor, progesterone receptor, or epidermal growth factor receptor 2 [3]. Treatment for TNBC includes immunotherapy, deoxyribonucleic acid (DNA)-interfering agents, chemotherapy and targeted therapies.

Chemotherapy is a primary approach in cancer treatment, aimed at both extending the patient's life and upgrading its quality [4]. The most often temporary toxic reactions linked to nearly all chemotherapeutic drugs include myelosuppression (e.g., neutropenia nadir, anemia, leukopenia, and thrombocytopenia), febrile neutropenia, hair loss (alopecia), and gastrointestinal (GI) side effects. These hematological (38.6%) and GI toxicities (12.9%) occur in 40% to 80% of breast cancer patients undergoing neo-adjuvant or adjuvant chemotherapy [5].

Given the high prevalence of chemotherapy-induced toxicities, researchers have looked into possible adjuvant medicines to lessen these side effects. Arglabin is one agent that shows promise. A Recent study by Valentina B. Sirota clearly indicates that Arglabin, when used as an adjuvant in breast cancer chemotherapy reduces toxicity related to erythrocytosis, leukocytosis, and granulocytosis. The study of Arglabin's antitumor mechanism revealed Arglabin blocks the mitogenic signals from oncogenic H-Ras and K-Ras proteins, which carry a farnesyl group, and induces the restoration of transformed cells by restricting

these signals. Arglabin has an immunomodulatory effect, exhibits low toxicity, and has therapeutic endurance [4].

Furthermore, Zhumakayeva Sabina Sakenkyzy's study indicates the advantages of coupling Arglabin with adjuvant polychemotherapy using the AS regimen in breast cancer treatment. This combination proves to be practically feasible as it helps to alleviate the hematological toxicities typically caused by chemotherapy, decreases oxidative stress, and restores normal purine metabolism. Additionally, it enhances the 3-year relapse-free survival rate by 9.5%. This adjuvant polychemotherapy approach is thus recommended for adoption in clinical oncology settings [6].

While chemotherapy remains the foundation of breast cancer treatment, its hematological toxicities vary across molecular subtypes, stressing further investigation. Identifying targeted strategies, such as Arglabin-based therapies, could help mitigate these effects and improve patient outcomes. This knowledge could enable more individualized treatment approaches, leading to better results. Additionally, clinical trials are essential to assess the safety and effectiveness of strategies for managing hematological toxicities, including optimal timing, dosage, and treatment combinations. Healthcare providers should consider adopting this approach to improve patient outcomes and minimize toxic effects. Investigating the mechanisms behind chemotherapy-induced anemia is crucial for improving patient care, enhancing treatment adherence, and developing more effective, tailored therapies.

### Author Contribution Statement

U.A, M.U.A, contributed significantly to the culmination, editing, revision and compilation of the entire manuscript.

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#### *Conflicts of interest*

None to declare.

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