

Comprehensive Sociodemographic, Clinical and Molecular Profiling of Breast Cancer in Morocco: Insights from 833 Patients at the Mohammed VI Cancer Center

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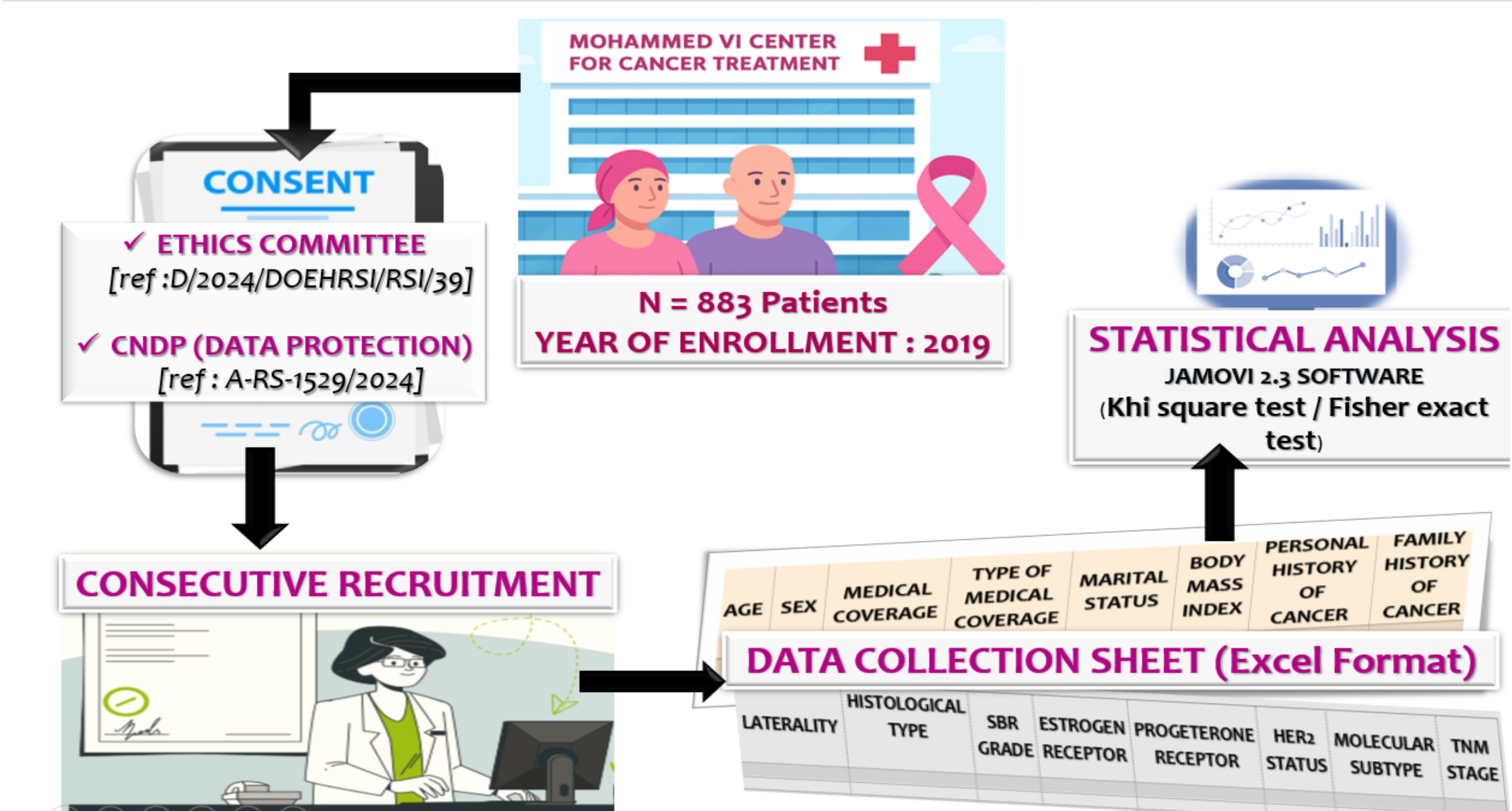
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BACKGROUND AND RATIONALE

Breast cancer (BC) is the most leading cause of cancer in women worldwide and is responsible for over 2 million new cases and 666,000 deaths per year. In Morocco, it is the main cancer cause in women, representing about 39%, and it is still the second major cause of death because of its heterogeneity and the extreme variation of molecular subtypes-related prognoses. Numerous regional studies (Casablanca, Rabat, Fez, Tangier, Kenitra, Souss-Massa) have analyzed the epidemiological profile of breast cancer—sociodemographic, clinical, and anatomopathological characteristics, genetic polymorphisms, access to care and psychosocial impact—in order to guide national policies. However, these studies often suffer from limitations (small sample sizes, focus on specific subpopulations, frequent exclusion of relapses, metastases, or patients treated outside the center) and heterogeneous geographical and social coverage, which reduces their national scope. In addition, important parameters are rarely described in detail in the Moroccan literature—notably the laterality of lesions, body mass index (BMI) and personal history of cancer. The molecular profile, meanwhile, remains largely unexplored at the national level, with the few studies available often being fragmentary, limited to partial associations and conducted on exclusive populations. To address these gaps, we propose a comprehensive and inclusive approach based on data from all patients with histologically confirmed BC registered in 2019 at the Mohammed VI Cancer Treatment Center, chosen for its particularly large patient population, which guarantees the representativeness of the results, integrating all profiles (age, initial/metastatic/relapse stage, unilateral/bilateral/multifocal, public or private treatment pathway, lack of coverage) in order to provide representative and generalizable data. It thus aims to fill the current gaps by providing a comprehensive overview of BC molecular subtypes and their clinical-pathological and demographic correlations. Most importantly, the current studies do not provide sufficient mechanistic insights to fully understand the heterogeneity that has been observed and they frequently depend on entirely speculative interpretations. Our integrative approach bases observations on validated biological mechanisms (genomic instability, cellular plasticity, ferroptosis resistance, immune escape), offering a coherent and biologically sound explanation for the heterogeneity of Moroccan BC.

METHODOLOGY

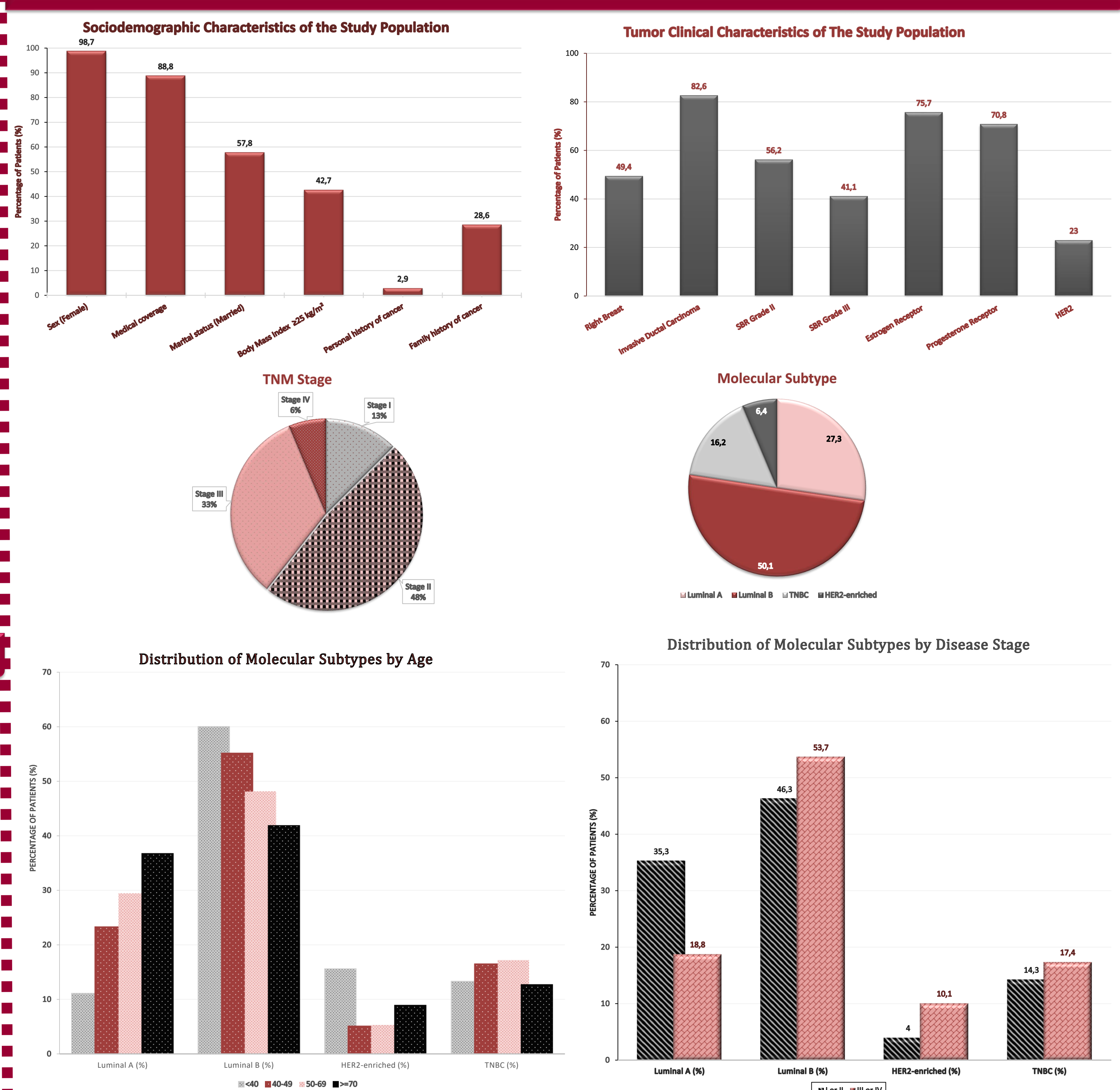


OBJECTIVE

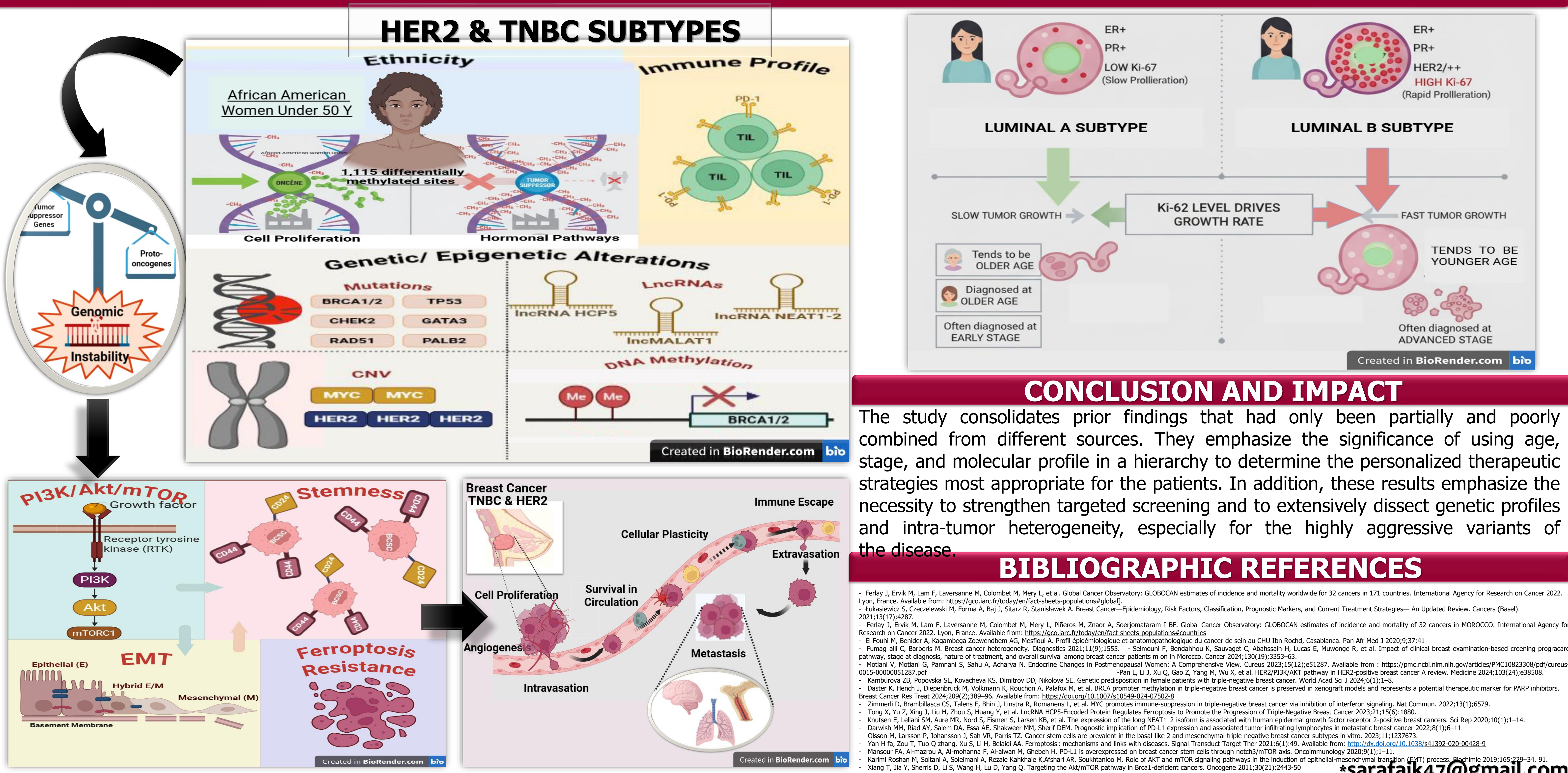
In the context of expanding our epidemiological and molecular understanding of BC, this study has been designed as a retrospective study, with the following: aims:

- 1- To describe the demographic, clinical, and histopathological profile of BC patients in Morocco;
- 2- To Analyze the associations between BC molecular subtypes and patients' socio-demographic and clinical features in Morocco, along with mechanistic explanations based on confirmed biological processes.

RESULTS



PHYSIOPATHOLOGICAL INTERPRETATION: MOLECULAR PROFILE DISTRIBUTION BY AGE AND STAGE



CONCLUSION AND IMPACT

The study consolidates prior findings that had only been partially and poorly combined from different sources. They emphasize the significance of using age, stage, and molecular profile in a hierarchy to determine the personalized therapeutic strategies most appropriate for the patients. In addition, these results emphasize the necessity to strengthen targeted screening and to extensively dissect genetic profiles and intra-tumor heterogeneity, especially for the highly aggressive variants of the disease.

BIBLIOGRAPHIC REFERENCES

1. Ferlay J, Ervik M, Lam F, Laversanne M, Colombet M, Mery L, et al. Global Cancer Observatory: GLOBOCAN estimates of incidence and mortality worldwide for 32 cancers in 171 countries. International Agency for Research on Cancer 2022. Lyon, France. Available from: <https://gco.iarc.fr/today/en/tac-sheets-populations#global>.
2. Lukaszewicz S, Czelewska M, Forma A, Baj J, Sierz S, Stanislawska A. Breast Cancer—Epidemiology, Risk Factors, Classification, Prognostic Markers, and Current Treatment Strategies—An Updated Review. Cancers (Basel). 2021;13(17):4287.
3. Ferlay J, Ervik M, Lam F, Laversanne M, Colombet M, Mery L, Pillerose M, Znaor A, Soerjomataram I, Bray F. Global Cancer Observatory: GLOBOCAN estimates of incidence and mortality of 32 cancers in MOROCCO. International Agency for Research on Cancer 2022. Lyon, France. Available from: <https://gco.iarc.fr/today/en/tac-sheets-populations#global>.
4. El Fouhi M, Bender A, Karambaga Zouwendarm AG, Mesfoui A. Profil épidémiologique et anatomopathologique du cancer de sein au CHU Ibn Rochd, Casablanca. Pan Afr Med J 2020;9:3741.
5. Fumagalli C, Barberis M. Breast cancer heterogeneity. Diagnostics 2021;11(9):1555.
6. Selimouni F, Bendahhou K, Sauvageat C, Abahssain H, Lucas E, Muwonge R, et al. Impact of clinical breast examination-based screening programme pathway, stage at diagnosis, nature of treatment, and overall survival among breast cancer patients in Morocco. Cancer 2024;130(19):3353–63.
7. Motiani V, Motiani G, Parniani S, Sahu A, Acharya N. Endocrine Changes in Postmenopausal Women: A Comprehensive Review. Cureus 2023;15(12):e51287. Available from: <https://pmc.ncbi.nlm.nih.gov/articles/PMC10823308/pdf/cureus-0015-0000051287.pdf>.
8. Kamburova ZB, Poposka SL, Kovacheva KS, Dimitrova DD, Nikolova SE. Genetic predisposition in female patients with triple-negative breast cancer. World Acad Sci J 2024;6(1):1–8.
9. Dastar K, Hench J, Diepenbruck M, Volkmann K, Rouillon A, Palafox M, et al. BRCA promoter methylation in triple-negative breast cancer is preserved in xenograft models and represents a potential therapeutic marker for PARP inhibitors. Breast Cancer Res Treat 2024;209(2):389–96. Available from: <https://doi.org/10.1007/s10549-024-07502-8>.
10. Zimmer D, Brambilla CS, Talens F, Bhan J, Linde R, Romanens L, et al. MYC promotes immune-suppression in triple-negative breast cancer via inhibition of interferon signaling. Nat Commun. 2022;13(1):6579.
11. Tong X, Yu Z, Xing J, Liu H, Zhou S, Huang Y, et al. LncRNA HCP5-Encoded Protein Regulates Ferroptosis to Promote the Progression of Triple-Negative Breast Cancer 2023;21:15(6):1880.
12. Knutsen E, Lilliehöj SM, Aure KR, Nord S, Fløien S, Larsen KB, et al. The expression of the long NEAT1_2 isoform is associated with human epidermal growth factor receptor 2-positive breast cancers. Sci Rep 2020;10(1):1–14.
13. Darwish MM, Rad AY, Salem DA, Eise AE, Shaker MM, Sherif DE. Prognostic implication of PD-L1 expression and associated tumor-infiltrating lymphocytes in metastatic breast cancer 2022;8(1):6–11.
14. Olsson M, Larsson P, Johansson J, Sah VR, Paris TZ. Cancer stem cells are prevalent in the basal-like 2 and mesenchymal triple-negative breast cancer subtypes in vitro. 2023;11:1237673.
15. Yin H, Zou T, Tuo Q, Zhang X, Li H, Belaid AA. Ferroptosis: mechanisms and links with diseases. Signal Transduct Target Ther 2021;6(1):49. Available from: <http://dx.doi.org/10.1038/s41392-020-00428-9>.
16. Mansour FA, Al-mazrou A, Al-mohanna F, Al-salwan M, Chebbi H, PD-L1 is overexpressed on breast cancer stem cells through notch3/MTOR axis. Oncolimmunology 2020;9(1):1–11.
17. Karimi Roshan M, Soltani A, Soleimani A, Rezaei Kakhkhaie K, Ashari AR, Soukhtanloo M. Role of AKT and mTOR signaling pathways in the induction of epithelial-mesenchymal transition (EMT) process. Biochimie 2019;165:29–34. 91.
18. Xiang T, Jia Y, Sherris DJ, Wang H, Lu D, Yang Q. Targeting the Akt/mTOR pathway in BRCA1-deficient cancers. Oncogene 2011;30(21):2443–50.