



OPEN ACCESS

EDITED AND REVIEWED BY

Francesco Grignani,
University of Perugia, Italy

*CORRESPONDENCE

Biswarup Basu,
✉ biswarupbasu@cnci.ac.in,
✉ biswarup.basu@gmail.com
Debanjan Chakroborty,
✉ dchakroborty@southalabama.edu
Chandrani Sarkar,
✉ csarkar@southalabama.edu

RECEIVED 09 December 2024

ACCEPTED 30 January 2025

PUBLISHED 11 February 2025

CITATION

Basu B, Chakroborty D and Sarkar C (2025)

Editorial: Obesity and cancer: the possible
molecular links.*Front. Cell Dev. Biol.* 13:1542429.

doi: 10.3389/fcell.2025.1542429

COPYRIGHT

© 2025 Basu, Chakroborty and Sarkar. This is an open-access article distributed under the terms of the [Creative Commons Attribution License \(CC BY\)](https://creativecommons.org/licenses/by/4.0/). The use, distribution or reproduction in other forums is permitted, provided the original author(s) and the copyright owner(s) are credited and that the original publication in this journal is cited, in accordance with accepted academic practice. No use, distribution or reproduction is permitted which does not comply with these terms.

Editorial: Obesity and cancer: the possible molecular links

Biswarup Basu^{1*}, Debanjan Chakroborty^{2,3,4*} and
Chandrani Sarkar^{2,3,4*}

¹Department of Neuroendocrinology and Experimental Hematology, Chittaranjan National Cancer Institute, Kolkata, India, ²Department of Pathology, University of South Alabama, Mobile, AL, United States, ³Cancer Biology Program, Mitchell Cancer Institute, University of South Alabama, Mobile, AL, United States, ⁴Department of Biochemistry and Molecular Biology, University of South Alabama, Mobile, AL, United States

KEYWORDS

obesity, breast cancer, lymphoma, prostate cancer, comorbidities

Editorial on the Research Topic

Obesity and cancer: the possible molecular links

Obesity is a global public health issue that adversely affects several disease pathogenesis and prognosis, including cancer, by increasing the risk and the number of deaths associated with cancer (Wang et al., 2019). The underlying molecular mechanisms of obesity-associated cancer progression are unclear. With worldwide growing obesity, the obese tumor microenvironment, in addition to cancer patients' socio-economical or ethnic perspectives, has emerged as a new area for understanding the dynamics of cancer initiation and outcomes obtained and for developing improved approaches to cancer management. The Research Topic "Obesity and Cancer: The Possible Molecular Links" was developed to highlight novel, original research findings, and critical reviews focusing on identifying the link between obesity and cancers. With three original research studies and one review article, this Research Topic highlights the interconnecting issues regulating obesity and cancer progression. Furthermore, the Research Topic has included epidemiological findings that reinforce the role of obesity in cancer progression by assessing the effect of neighborhood obesogenic environment on cancer risk and mortalities.

Obese breast cancer (BCa) patients, particularly postmenopausal women, have an increased risk of hormone receptor (HR)-positive BCa compared to lean women and often demonstrate more aggressive forms of the disease and face numerous challenges during therapy (LeVee and Mortimer, 2023). Though in premenopausal women, obesity is reported to reduce the risk of BCa, after BCa diagnosis, obesity worsens overall survival (OS) in all BCa subtypes (LeVee and Mortimer, 2023). The interplay between adipocytes and tumor cells contributing to the adipose microenvironment significantly influences tumor growth and response to therapy (Nieman et al., 2013). Kakkat et al. reviewed the complex relationship between BCa cells and adipocytes during tumor development, progression, and therapeutic response. The review discusses how circulating adipokines influence BCa progression. While adipokines like leptin, resistin, chemerin, visfatin, osteopontin, apelin, and lipocalin 2 promote BCa pathogenesis, adiponectin confers a protective effect by suppressing breast carcinogenesis (Nehme et al., 2022). As leptin exerts an opposite effect by promoting BCa initiation, growth, and metastasis, the adiponectin-leptin ratio plays a critical role in breast tumorigenesis (Grossmann and Cleary, 2012). Cancer-associated adipocytes (CAAs) secrete factors influencing immune cell recruitment, functions, differentiation, and immune escape, thereby promoting tumor progression. Adipocytes also contribute to the formation of extracellular matrix (ECM),

which in turn promotes metastatic dissemination and cancer advancement. Furthermore, adipocytes stimulate resistance to therapy by activating multiple signaling pathways that promote angiogenesis, increase tumor cell proliferation, and decrease apoptosis. A thorough investigation of the effects of strategies in weight loss on BCa progression and therapeutic response and the molecular pathways affected is essential for the development of novel therapeutic approaches for BCa patients.

In their original research article, [Wu et al.](#) studied the impact of body mass index (BMI) on malignant lymphoma. Numerous prior reports indicate a positive link between high BMI and both B cell derived Hodgkin's lymphoma (HL) and non-Hodgkin's lymphoma (NHL), derived from B cells and T cells, indicating that obesity is a potential risk towards malignant lymphoma ([Larsson and Wolk, 2011](#); [Murphy et al., 2013](#)). Interestingly, some studies also indicate that obese or overweight patients have a better favorable prognosis in malignant lymphoma, making the role of obesity in lymphoma controversial ([Landgren et al., 2005](#); [Ho et al., 2014](#)). In their study, [Wu et al.](#) determined the potential link between malignant lymphoma and BMI through two-sample Mendelian randomization. The study took into account 369 cases of HL, 209 cases of diffused large B-cell lymphoma, 522 cases of follicular lymphoma, 150 cases of mature T/NK- cell lymphomas, and 533 cases of other and unspecified types of NHL. The study findings indicate that adipose tissue is protective against HL, and lower BMI could be a significant risk factor for HL. The study results highlight the need for further research to understand the underlying molecular mechanism for this potential correlation thoroughly.

Studies showing the effect of comorbidities on cancer management and treatment are mostly reported from developed countries ([Edwards et al., 2014](#); [Panigrahi and Ambs, 2021](#)) because of improved event reporting and disease management systems. However, studies from less developed countries are scarce, leading to a substantial gap in the understanding of comorbidities like obesity on cancer progression among diverse populations. [Birhanu et al.](#), in their original research work, assessed the prevalence of comorbidities and their associated factors in Eastern Ethiopia. The cross-sectional study took into consideration 422 cancer cases selected by a simple random sampling technique. Medical records extracted data was entered into the Epi-Data statistical software and analyzed using STATA. The study identified a lower overall prevalence of chronic comorbidities in cancer patients compared to previously reported numbers from other regions worldwide. However, a strong association between obesity and other comorbidities was identified, which influenced cancer progression. The study findings indicate that the chances of having chronic comorbidities were higher among obese cancer patients compared to lean patients. The findings further suggest that weight reduction is a measure to limit added complications and reduce cancer progression. It also provides interesting information about the lower incidence of comorbidities in cancer patients in Eastern Ethiopia, which suggests that different lifestyles and differences in genetic background can affect cancer incidence and progression differently. The observation underscores the critical need for more studies in underdeveloped and developing countries to help better understand the role of obesity and other comorbidities in cancer and other diseases.

Obesity has been reported to be associated with aggressive and high-grade prostate cancer (PCa) and increased PCa-associated

deaths ([Saha et al., 2023](#)). The original research article by [Kumsa et al.](#) examined the association between neighborhood obesogenic attributes such as neighborhood socioeconomic status (nSES) with environment indices like retail-food environment index, restaurant environments index, parks, businesses, recreational facilities, and disease-associated mortality as well as incidences in prostate cancer in the Southern Community Cohort Study (SCCS). Risk analysis of PCa was further stratified by race and BMI. The study included 28,356 prostate cancer patients. Study results indicate that lower nSES, like reduced walkable areas and recreational facilities, lead to an increased prostate cancer risk and associated mortality, particularly in the African American population. The study outcomes indicate the role of obesity-associated factors in regulating PCa and highlight the importance of investigating the effect of the local environment on PCa screening and management.

Taken together, the findings from these studies clearly indicate the complex relationship between body weight and cancer progression. Further research is needed to comprehend the molecular pathways underlying obesity-associated cancer progression, identify novel biomarkers in different groups at risk, and identify new targets that can lead to the design of improved therapeutic approaches for successful clinical management of cancer. We believe that this Research Topic will serve as a valuable resource to drive further advancement in this field.

Author contributions

BB: Writing–original draft, Writing–review and editing. DC: Writing–original draft, Writing–review and editing. CS: Writing–original draft, Writing–review and editing.

Funding

The author(s) declare that financial support was received for the research, authorship, and/or publication of this article. Authors are supported by funding from the Department of Biotechnology, Government of India (Sanction no. BT/PR38493/TRM/120/465/2020) (BB) and the Department of Pathology, USA Health Mitchell Cancer Institute, University of South Alabama, and Breast Cancer Research Foundation of Alabama (DC and CS).

Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

The author(s) declared that they were an editorial board member of *Frontiers*, at the time of submission. This had no impact on the peer review process and the final decision.

Generative AI statement

The author(s) declare that no Generative AI was used in the creation of this manuscript.

Publisher's note

All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated

organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher.

References

- Edwards, B. K., Noone, A. M., Mariotto, A. B., Simard, E. P., Boscoe, F. P., Henley, S. J., et al. (2014). Annual Report to the Nation on the status of cancer, 1975-2010, featuring prevalence of comorbidity and impact on survival among persons with lung, colorectal, breast, or prostate cancer. *Cancer*. 120 (9), 1290–1314. doi:10.1002/cncr.28509
- Grossmann, M. E., and Cleary, M. P. (2012). The balance between leptin and adiponectin in the control of carcinogenesis - focus on mammary tumorigenesis. *Biochimie* 94 (10), 2164–2171. doi:10.1016/j.biochi.2012.06.013
- Hong, F., Habermann, T. M., Gordon, L. I., Hochster, H., Gascoyne, R. D., Morrison, V. A., et al. (2014). The role of body mass index in survival outcome for lymphoma patients: US intergroup experience. *Ann. Oncol.* 25 (3), 669–674. doi:10.1093/annonc/mdt594
- Landgren, O., Andrén, H., Nilsson, B., Ekblom, A., and Björkholm, M. (2005). Risk profile and outcome in Hodgkin's lymphoma: is obesity beneficial? *Ann. Oncol.* 16 (5), 838–840. doi:10.1093/annonc/mdl145
- Larsson, S. C., and Wolk, A. (2011). Body mass index and risk of non-Hodgkin's and Hodgkin's lymphoma: a meta-analysis of prospective studies. *Eur. J. Cancer* 47 (16), 2422–2430. doi:10.1016/j.ejca.2011.06.029
- LeVee, A., and Mortimer, J. (2023). The challenges of treating patients with breast cancer and obesity. *Cancers (Basel)* 15 (9), 2526. doi:10.3390/cancers15092526
- Murphy, F., Kroll, M. E., Pirie, K., Reeves, G., Green, J., and Beral, V. (2013). Body size in relation to incidence of subtypes of haematological malignancy in the prospective Million Women Study. *Br. J. Cancer* 108 (11), 2390–2398. doi:10.1038/bjc.2013.159
- Nehme, R., Diab-Assaf, M., Decombat, C., Delort, L., and Caldefie-Chezet, F. (2022). Targeting adiponectin in breast cancer. *Biomedicines* 10 (11), 2958. doi:10.3390/biomedicines10112958
- Nieman, K. M., Romero, I. L., Van Houten, B., and Lengyel, E. (2013). Adipose tissue and adipocytes support tumorigenesis and metastasis. *Biochim. Biophys. Acta* 1831 (10), 1533–1541. doi:10.1016/j.bbali.2013.02.010
- Panigrahi, G., and Ambs, S. (2021). How comorbidities shape cancer biology and survival. *Trends Cancer* 7 (6), 488–495. doi:10.1016/j.trecan.2020.12.010
- Saha, A., Kolonin, M. G., and DiGiovanni, J. (2023). Obesity and prostate cancer - microenvironmental roles of adipose tissue. *Nat. Rev. Urol.* 20 (10), 579–596. doi:10.1038/s41585-023-00764-9
- Wang, Y. X., Zhu, N., Zhang, C. J., Wang, Y. K., Wu, H. T., Li, Q., et al. (2019). Friend or foe: multiple roles of adipose tissue in cancer formation and progression. *J. Cell Physiol.* 234 (12), 21436–21449. doi:10.1002/jcp.28776