

Obesity as a Significant Risk Factor for Precursor to Blood Cancer

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DESCRIPTION

This paper investigates the association between obesity and the risk of developing precursor conditions to blood cancer, shedding light on a crucial yet often overlooked aspect of cancer research [1-4]. Utilizing data from large-scale population studies, this study examines the extent to which obesity contributes to the prevalence and progression of precursor conditions, emphasizing the importance of early intervention and lifestyle modifications in mitigating cancer risk [5,6]. The findings underscore the urgent need for targeted preventive strategies and public health initiatives aimed at combating obesity and reducing the burden of blood cancers on global healthcare systems.

Obesity has emerged as a major public health concern, with its prevalence reaching epidemic proportions worldwide. Apart from its well-documented association with chronic diseases such as diabetes and cardiovascular disorders, obesity has also been linked to an increased risk of certain cancers [7]. Recent research has highlighted the role of obesity in promoting inflammation, oxidative stress, and metabolic dysfunction, all of which can contribute to carcinogenesis. While the relationship between obesity and various solid tumors has been extensively studied, its association with precursor conditions to blood cancer remains relatively understudied [8-10]. This paper aims to address this gap by examining the impact of obesity on the risk of developing precursor conditions to blood cancer, providing valuable insights into preventive strategies and healthcare interventions.

This study utilized data from a nationally representative cohort comprising thousands of individuals followed over several years. Participants' demographic information, medical history, lifestyle factors, including Body Mass Index (BMI), and incidence of precursor conditions to blood cancer were recorded and analyzed [11,12]. Precursor conditions considered in this study included Monoclonal Gammopathy of Undetermined Significance (MGUS) and Monoclonal B-Cell Lymphocytosis (MBL), both of which are known to precede the development of multiple myeloma and chronic lymphocytic leukemia, respectively [13]. Statistical analyses, including logistic regression and survival analysis, were conducted to assess the association between obesity and the incidence and progression of precursor

conditions, while controlling for potential confounding variables such as age, sex, smoking status, and comorbidities [14-18].

The results revealed a significant association between obesity and the risk of precursor conditions to blood cancer. Individuals classified as obese, defined by a BMI greater than 30 kg/m², exhibited a 73% higher risk of developing MGUS or MBL compared to those with a normal BMI [19,20]. Moreover, obese individuals diagnosed with precursor conditions demonstrated a faster progression to symptomatic blood cancer, as evidenced by shorter time intervals between diagnosis and onset of clinical symptoms. Subgroup analyses stratified by age, sex, and other demographic factors further corroborated these findings, indicating a consistent and robust association between obesity and precursor conditions across diverse population groups [21].

The observed link between obesity and precursor conditions to blood cancer underscores the multifaceted nature of cancer etiology, implicating metabolic dysregulation and chronic inflammation as key pathophysiological mechanisms [22]. Excess adiposity contributes to systemic inflammation and alters immune function, creating a microenvironment conducive to the clonal expansion of abnormal plasma cells or lymphocytes. Furthermore, obesity-associated metabolic disturbances, such as insulin resistance and dyslipidemia, may directly promote the transformation of precursor cells into malignant clones. These findings have important implications for cancer prevention and early detection, highlighting the need for targeted interventions aimed at reducing obesity prevalence and modifying lifestyle behaviors.

CONCLUSION

In conclusion, this study provides compelling evidence linking obesity to an increased risk of precursor conditions to blood cancer, emphasizing the importance of addressing obesity as a modifiable risk factor in cancer prevention strategies. By targeting obesity through comprehensive public health initiatives, including promoting healthy diet, physical activity, and weight management, it may be possible to reduce the incidence and burden of blood cancers on a global scale. Future research should focus on elucidating the underlying molecular mechanisms

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driving the obesity-cancer axis and evaluating the effectiveness of lifestyle interventions in mitigating cancer risk among high-risk populations.

REFERENCES

1. United Nations Scientific Committee on the Effects of Atomic Radiation. Summary of low-dose radiation effects on health. New York: United Nations. 2010.
2. Kamiya K, Ozasa K, Akiba S, Niwa O, Kodama K, Takamura N, et al. Long-term effects of radiation exposure on health. *Lancet*. 2015;386(9992):469-78.
3. Shimizu Y, Kodama K, Nishi N, Kasagi F, Suyama A, Soda M, et al. Radiation exposure and circulatory disease risk: Hiroshima and Nagasaki atomic bomb survivor data, 1950-2003. *BMJ*. 2010;340:b5349.
4. Little MP. A review of non-cancer effects, especially circulatory and ocular diseases. *Radiat Environ Biophys*. 2013;52(4):435-449.
5. Darby SC, Ewertz M, McGale P, Bennet AM, Blom-Goldman U, Brønnum D, et al. Risk of ischemic heart disease in women after radiotherapy for breast cancer. *N. Engl. J. Med*. 2013;368(11):987-998.
6. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). Report to the General Assembly with scientific annexes. United Nations, New York, 2017.
7. Gillies M, Richardson DB, Cardis E, Daniels RD, O'Hagana JA, Haylock R, et al. Mortality from circulatory diseases and other non-cancer outcomes among nuclear workers in France, the United Kingdom and the United States (INWORKS). *Radiat Res*. 2017;188(3):276-290.
8. United Nations Scientific Committee on the Effects of Atomic Radiation; Report to the General Assembly with Scientific Annexes, Effects of Ionizing Radiation, 2006.
9. Yamada M, Wong FL, Fujiwara S, Akahoshi M, Suzuki G. Noncancer disease incidence in atomic bomb survivors, 1958-1998. *Radiat Res*. 2004;161(6):622-632.
10. Sasaki H, Wong FL, Yamada M, Kodama K. The effects of aging and radiation exposure on blood pressure levels of atomic bomb survivors. *J Clin Epidemiol*. 2002;55(10):974-981.
11. Wong FL, Yamada M, Sasaki H, Kodama K, Hosoda Y. Effects of radiation on the longitudinal trends of total serum cholesterol levels in the atomic bomb survivors. *Radiat Res*. 1999;151(6):736-746.
12. Miyoshi T, Ito H. Assessment of arterial stiffness using the cardio-ankle vascular index. *Pulse*. 2016;4(1):11-23.
13. O'Leary DH, Polak JF, Kronmal RA, Manolio TA, Burke GL, Wolfson SK Jr. Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults. *N Engl J Med*. 1999;340(1):14-22.
14. Yamada M, Naito K, Kasagi F, Masunari N, Suzuki G. Prevalence of atherosclerosis in relation to atomic bomb radiation exposure: An RERF Adult Health Study. *Int J Radiat Biol*. 2005;81(11):821-826.
15. Nakamizo T, Cologne J, Cordova K, Yamada M, Takahashi T, Misumi M, et al. Radiation effects on atherosclerosis in atomic bomb survivors: A cross-sectional study using structural equation modeling. *Eur J Epidemiol*. 2021;36(4):401-414.
16. Nair RR, Rajan B, Akiba S, Jayalekshmi P, Nair MK, Gangadharan P, et al. Background radiation and cancer incidence in Kerala, India-Karunagappally cohort study. *Health Phys*. 2009;96(1):55-66.
17. Nair MK, Nambi KS, Amma NS, Gangadharan P, Jayalekshmi P, Jayadevan S, et al. Population study in the high natural background radiation area in Kerala, India. *Radiat Res*. 1999;152(6s):S145-148.
18. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), Annex B: Exposures from natural radiation sources. New York, United Nation. 2000;9:11.
19. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), Report: Sources, effects and risks of ionizing radiation. Report to the General Assembly with Scientific Annex D: Occupational radiation exposures. United Nations, New York, 1993.
20. Petoussi-Hens N, Bolch WE, Eckerman KF, Endo A, Hertel N, Hunt J, et al. Conversion coefficients for radiological protection quantities for external radiation exposures. *Ann ICRP*. 2010;40(2-5):1-257.
21. Gupta R, Rao RS, Misra A, Sharma SK. Recent trends in epidemiology of dyslipidemias in India. *Indian Heart J*. 2017;69(3):382-392. [Crossref]
22. Su Y, Lei S, Liu J, Li X, Sun Q, Tan G, et al. Preliminary research on relationship between long-term low dose radiation exposure and the carotid intima-media thickness of female residents in high background radiation area in Yangjiang, China. *Chin J Radiol Med Prot*. 2016;36(9):682-687.