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Editorial

Obesity, metabolism and the coming Cancer Wave: Why the oncology community must lead a whole-of-society response?

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In 2022, more than one billion people worldwide; approximately one in every eight individuals were living with obesity, a prevalence that has doubled among adults and quadrupled among adolescents since 1990 Obesity is no longer merely a lifestyle concern; it represents a global metabolic challenge that is increasingly intersecting with, and amplifying, the burden of cancer. A recent systematic analysis estimates that approximately 356,000 cancer deaths and nearly nine million cancer disability-adjusted life years (DALYs) in 2021 were attributable to high body mass index (BMI) [2,3]

Several obesity-related metabolic phenotypes have been described in the literature, often along axes of adiposity and metabolic health. These include:

1) Metabolically healthy obesity (MHO)
2) Metabolically unhealthy obesity (MUO)
3) Normal-weight obesity (NWO)
4) Metabolically obese normal weight (MONW)

While these constructs are useful heuristics, they are neither discrete nor universally accepted categories. Substantial overlap exists between phenotypes, particularly in South Asian populations, where visceral adiposity and insulin resistance may occur at lower BMI thresholds. This heterogeneity should be acknowledged when interpreting epidemiologic risk or translating findings into policy and practice.

South Asian countries such as Pakistan face a compounded challenge, where undernutrition and micronutrient deficiencies coexist with rapidly rising rates of obesity and metabolic disease. The most recent Global Nutrition Report indicates that approximately 13.4% of adult women and 7.5% of adult men in Pakistan are living with obesity, figures that exceed the regional average for women [4]. When combined with a high national prevalence of diabetes mellitus and a predominantly young population, these trends set the stage for a prolonged and resource-intensive cancer burden. Claims regarding global ranking should be interpreted cautiously, as international comparisons vary by indicator and methodology.

How excess adiposity contributes to malignancy

Obesity promotes carcinogenesis through multiple, interacting biological pathways, including hyperinsulinaemia and insulin-like growth factor-1 (IGF-1) signalling, chronic low-grade inflammation, sex-hormone dysregulation, altered adipokine profiles, and changes in gut microbiota and bile-acid metabolism. Selected examples of pathways and tumor sites with relatively strong epidemiologic or mechanistic support are summarized below. These associations should be viewed as illustrative rather than exclusive, as evidence remains heterogeneous across studies and cancer types.

Examples of obesity-related pathways and associated tumor sites:

1) Hyperinsulinaemia / IGF-1 signalling (insulin, IGF-1, PI3K–mTOR): colorectal cancer, postmenopausal breast cancer, pancreatic cancer
2) Chronic low-grade inflammation (TNF-α, IL-6, CRP): hepatocellular carcinoma arising from NAFLD, esophageal adenocarcinoma
3) Sex-hormone dysregulation (aromatase up-regulation, estrogen excess): endometrial cancer, breast cancer, ovarian cancer
4) Altered adipokine profile (increased leptin, reduced adiponectin): renal cell carcinoma, advanced prostate cancer
5) Gut dysbiosis and bile-acid alterations: colorectal cancer, gallbladder cancer

The International Agency for Research on Cancer (IARC) currently recognizes at least 13–14 cancer sites with sufficient evidence of an association with excess body fatness. Several of these, including colorectal, endometrial, and pancreatic cancers have shown rising incidence among adults under 50 years of age in multiple regions. However, the magnitude and pace of these increases vary considerably by geography and tumor type, and broad statements about cancers increasing "fastest" should be made with caution [5-7].

Non-alcoholic fatty liver disease (NAFLD) illustrates the obesity–cancer continuum. Data from Asia suggest that NAFLD prevalence has increased to nearly one-third of adults in some populations, with hepatocellular carcinoma (HCC) increasingly recognized in association with NAFLD, including a subset of patients without established cirrhosis, although cirrhosis-associated disease remains more common [8].

A dual burden for low- and middle-income countries

While high-income countries debate the cost and scalability of next-generation weight-loss pharmacotherapies, many low- and middle-income countries (LMICs) contend with the simultaneous burdens of undernutrition and overnutrition. In Pakistan, the National Nutrition Survey 2018 reports stunting in approximately 40% of children under five years of age, yet sugar-sweetened beverages remain among the cheapest sources of calories in urban settings [9]. This life-course juxtaposition of early undernutrition and later metabolic excess may further amplify long-term cancer risk.

Policy traction is possible, but context dependent

In 2023, Pakistan increased its excise duty on carbonated beverages to 20% and introduced a 10% tax on selected fruit juices and syrups [10]. Modelling studies from specific national contexts suggest that a sustained 20% sugar-sweetened beverage (SSB) tax could meaningfully reduce obesity prevalence over a decade, with proportionally larger effects among children [11]. However, these estimates are highly context specific, depending on baseline consumption patterns, price elasticities, and complementary policies, and should not be interpreted as universal or directly transferable.

Allocating SSB tax revenues toward health-promoting initiatives, such as school nutrition programs, active-transport infrastructure, or culturally appropriate public education campaigns, may enhance impact and public acceptability. Nonetheless, the feasibility of such earmarking varies across settings, and implementation challenges should be acknowledged.

New therapeutic promise is not a substitute for prevention

The oncology community has shown understandable interest in observational data presented at ASCO 2025 reporting associations between use of glucagon-like peptide-1 (GLP-1) receptor agonists and lower incidence of certain obesity-related cancers in large matched cohorts [12]. These findings are hypothesis generating and should not be interpreted as evidence of causality. Long-term randomized data on cancer-specific outcomes are still lacking, and projections from class-level associations to population-wide effects, particularly in resource-limited settings should be made cautiously.

Five priorities for an integrated response

Embed pragmatic metabolic assessment in cancer pathways. Routine oncology care should include BMI, waist circumference, and fasting plasma glucose. Glycated hemoglobin (HbA1c) may be appropriate in selected high-risk cancers. Measures such as the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) currently lack assay standardization and guideline endorsement for routine clinical use and are better framed as research tools rather than clinical screening tests [13].

Make metabolic health a core competency of cancer care teams. Dietitians, physiotherapists, and behavioral specialists are essential members of multidisciplinary oncology care. Prehabilitation programs have demonstrated benefits in functional status and perioperative outcomes; stronger claims regarding reductions in cardiovascular events should be moderated unless supported by cancer-specific evidence.

Align fiscal and urban-planning levers with health goals. Well-designed SSB taxes, agricultural subsidies for healthy foods, and investments in safe public spaces can help create environments that support healthier choices, particularly for women and girls. Leverage primary care and community health workers. In Pakistan, the Lady Health Worker program provides a scalable platform for culturally tailored counselling on breastfeeding, child nutrition, and adult dietary practices across the life course.

Prioritize region-specific research. South Asian populations often develop visceral adiposity and insulin resistance at lower BMI thresholds, a pattern sometimes described as a "thin–fat" phenotype. Locally led cohorts and biobanks are needed to refine risk prediction and guide earlier screening thresholds.

A call to action

The IARC projects that overall cancer incidence in LMICs will rise by approximately 75% by 2045, reflecting population growth, aging, and changing exposure to risk factors. The proportion of this increase attributable specifically to obesity remains uncertain, but excess adiposity is poised to become an increasingly important contributor. Oncology has mastered precision approaches to molecular targets; it must now apply comparable rigor to the metabolic determinants shaping the next cancer wave.

History will judge the cancer community not only by the therapies it develops, but by the preventable epidemics it helps avert. Combating obesity, through evidence-informed clinical practice, cautious interpretation of emerging therapies, and context-sensitive policy must be recognized as integral to comprehensive cancer control.

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