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Obesity and Cancer Risk: A Synthesis

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ABSTRACT

Obesity is a multifactorial metabolic condition increasingly recognized as a major risk factor for several cancers, including those of the breast, endometrium, colon, pancreas, kidney, and liver. This paper provides an integrative synthesis of the biological, epidemiological, therapeutic, and socio-ethical dimensions of the obesity–cancer nexus. Evidence demonstrates that excess adiposity promotes carcinogenesis through metabolic, hormonal, and inflammatory pathways involving insulin–IGF signaling, adipokine imbalance, oxidative stress, and chronic low-grade inflammation. Lifestyle, pharmacologic, and surgical interventions that induce weight loss show potential for lowering cancer incidence and improving outcomes among survivors, though the magnitude and timing of effects remain uncertain. Lifestyle interventions combining diet, physical activity, and behavioral modification produce the most consistent benefits, while pharmacotherapies and bariatric surgery offer additional options for high-risk groups. Persistent knowledge gaps concern the temporal dynamics of obesity, genetic modifiers, and population-specific responses. Ethical and equity considerations reveal that obesity and cancer risks are unequally distributed, disproportionately affecting low-income and minority populations who face barriers to prevention and treatment. Globally, obesity-related cancers are rising rapidly, with marked regional variation reflecting differences in environment, socioeconomic status, and health infrastructure. Integrating obesity prevention into cancer control policies, advancing translational research, and addressing structural health inequities are essential to mitigating the growing global burden of obesity-associated cancers.
Keywords: Obesity and Cancer Risk: Biological Mechanisms and Carcinogenesis, Weight-Loss Interventions, Health Equity and Global Disparities, and Translational and Preventive Oncology.

INTRODUCTION

Obesity is a worldwide public health epidemic occurring at unprecedented levels, with its rate continuing to rise. Approximately two-thirds of adults in the United States are now considered overweight or obese [1], and more than 600 million adults worldwide meet the World Health Organization criteria for obesity [2]. Most of these individuals also meet the criteria for metabolic syndrome and exhibit subclinical increases in insulin, insulin-like growth factor-1 (IGF-1), leptin, inflammatory factors, and vascular-related factors, such as vascular endothelial growth factor (VEGF) and plasminogen activator inhibitor-1 (PAI-1). Despite truncated definitions of obesity, evidence is emerging that excess weight in infancy (understanding it mostly occurs through rapid weight gain rather than high weight-for-age) adds to cancer risk [3]. Obesity increases the risk of, and decreases survival from, multiple chronic diseases, most notably several cancers; yet obesity prevention continues to be underappreciated as a public health priority [13]. An estimated 15–30% of cancer deaths in the United States are attributed to overweight and obesity, and strong evidence links obesity with endometrial, postmenopausal breast, colon, renal, liver, gallbladder, esophageal, and pancreatic cancers, while associations with cervical, ovarian, prostate, and stomach cancers are also emerging. The link between obesity and cancer is under-investigated compared to other chronic diseases [12].

Rationale and scope

Obesity is an increasingly prevalent condition closely associated with an elevated risk of many cancers [5]. How fatness, fat distribution, and other obesity-related factors affect cancer development is relatively well understood, supporting well-recognised prevention and screening implications [13]. This review synthesises the evidence on obesity in relation to cancer risk and provides a basis for future research on the mechanisms underlying these relationships [15]. Obesity connects to cancer through several biological pathways, where activation of one or more often leads to increased cancer risk, and particular cancers show stronger associations than others [15]. The risk of common cancers with the most marked associations is shaped chiefly by the interplay of site and sex [15]. How fatness, fat distribution, and other obesity-related factors affect cancer development is relatively well understood, supporting well-recognised prevention and screening implications [11]. Numerous cancers are causally or probably causally linked to obesity, notably endometrial, kidney, liver, colorectal, breast, and oesophageal adenocarcinoma; for some cancers, these links are also clinically and biologically relevant [16]. By addressing clinical and public health implications and considering intervention questions in light of the biological mechanisms involved, the synthesis guides future research into translational aspects of the evidence [14].

Overview of Obesity and Cancer Epidemiology

Obesity defined by a BMI of 30 or higher, has risen dramatically worldwide, with 35% of adults and 20% of children in the U.S. classified as obese [5]. Metabolic syndrome accompanies this condition and is characterized by increased waist circumference, insulin resistance, hyperglycemia, hypertension, and hypertriglyceridemia. Individuals in this category often display higher levels of insulin, IGF-1, leptin, inflammatory factors, and vascular elements such as VEGF and PAI-1 [6]. Collectively, these physiological alterations influence the tumor microenvironment, contributing to enhanced susceptibility and progression of cancer. Obesity significantly increases the risk and worsens the prognosis for a range of malignancies. It is estimated that 15–30% of cancer fatalities in the U.S. are attributable to overweight and obesity [3]. Special attention has been paid to the strong associations with endometrial, postmenopausal breast, colorectal, renal cell carcinoma, liver, gallbladder, esophageal adenocarcinoma, and pancreatic cancers [2]. Mechanisms underpinning the relationship include altered growth signaling, chronic inflammation, disrupted vascular homeostasis, and microenvironmental modifications that further elevate risk and detrimentally impact prognosis [1].

Key Definitions and Measures

Cancer is the second-leading cause of death worldwide, with between 60% and 70% of cases attributable to modifiable lifestyle risk factors [1]. Among these factors, obesity, defined by excess body fat, is considered to be a major contributing factor to common cancers. Such obesity-attributable cancers contrarily have limited treatment options after diagnosis [1]. Therefore, preventing obesity and promoting weight loss are presumed to be crucial for reducing overall cancer risk and mortality [3]. For effective prevention and control of obesity-attributable cancers, decisive evidence is needed to specify the extent to which obesity promotes cancer initiation, progression, and/or metastasis [5]. Most of the cancer-causal biological mechanisms attributed to high level of adiposity originates from excess energy consumed and/or attempted metabolic regulation of excess fat. Common among them, the insulin/IGF1 axis directly promotes cell proliferation, inhibits apoptosis of the hyperplastic cells, and enhances metabolic turnover of the proliferating cells [5]. Even though individual contributors are presently not totally understood yet, there are sufficient findings to support the proposition that marking increasements of blood insulin or fatty acid content should be considered together with the biological markers of the mechanism-related hormones and sex hormones as neuroendocrine activity classic indicators of the cancer risk – as relevant biomarkers of a cancer initiation or promotion signal [7].

Biological Mechanisms Linking Obesity to Cancer

Cancer development is promoted through molecular and cellular mechanisms in people with obesity [1]. The insulin/insulin-like growth factor (IGF) signaling axis, adipokines and inflammation, sex hormones, oxidative stress, and the gut microbiome play significant roles [3]. The insulin/IGF axis is implicated in a variety of cancers. Adipokines such as leptin and resistin, pro-inflammatory cytokines, and cell adhesion molecules released from adipous tissues can affect cancer initiation and progression, induce resistance to apoptosis, and enhance invasiveness. Sex hormones, especially those synthesized and metabolized in adipose tissues, maintain normal homeostasis and cell proliferation [5]. Altered levels of sex hormones may trigger and maintain tumor promotion. Various oxidative molecules aggravate DNA damage and promote cancer initiation, malignancy, and progression. The gut microbiome changes in people with obesity and certain microorganisms may affect metabolism, pro-inflammatory factors, and xenobiotic metabolism, thereby influencing cancer developments [4]. The mechanisms linking obesity to cancer largely operate through various biomarkers, the definitions and measures of which are provided in the next section. The insulin/IGF axis, sex hormones, adipokines, and the microbiome can be affected by body mass index (BMI) measure, the timing of exposure, and the whole lifetime trajectory of individuals [1, 2].

Insulin/IGF Axis and Metabolic Signaling

Elevated circulating levels of insulin and IGF-I represent key metabolic abnormalities linking obesity to cancer [2]. Epidemiological studies consistently associate excess body weight with increased risk of at least 13 types of cancer, including several of major clinical and public health importance. Insulin and IGF-I, together with their associated binding proteins, have been extensively studied for their roles in stimulating cell proliferation and inhibiting apoptosis, processes that drive tumorigenesis [3]. Elevations of both insulin and IGF-I represent independent, hypothesized mediators of the obesity–cancer link [3]. With a precursor fat depot, such as subcutaneous abdominal adipose tissue, hypertrophic expansion leading to insulin resistance promotes the spill-over of excess fatty acids to the liver, resulting in hepatic overproduction of very-low-density lipoprotein (VLDL) and atherogenic dyslipidemia characterized by triglyceride-rich lipoproteins [5]. Central obesity, insulin resistance, and atherogenic dyslipidemia comprise the metabolic syndrome. Insulin and IGF-I, mediated by the TCA cycle intermediate citrate, stimulate de novo lipogenesis, promoting abdominal fat accumulation [4]. Circulating insulin levels gradually rise in response to excessive energy intake, and circulating IGF-I levels are elevated in individuals with the metabolic syndrome [6]. Insulin, via its cognate receptor (IR), and IGF-I, via the type 1 IGF receptor (IGF1R), elicit similar effects on target tissues via overlapping signaling pathways, providing a molecular basis for the observed interrelationship between circulating insulin and IGF-I levels. The Akt/PI3K/mTOR signaling pathway is activated by both insulin and IGF-I and critically regulates cancer-relevant physiological processes such as cellular proliferation, growth, metabolism, and apoptosis [5].

Adipokines, Inflammation, and Adipose Tissue Remodeling

Obesity is associated with a pro-inflammatory state that fosters metabolic dysregulation and increases the risk of various cancers [6]. Longitudinal studies show that with sustained abnormal weight gain and obesity, numerous malignancies become apparent; while breast, endometrial, and colorectal cancers, some of the most prevalent obesity-associated neoplasms, represent early complications triggered shortly after body-weight abnormality [7]. Adipose tissue, both storage and ectopic, serves as an active endocrine organ with autocrine, paracrine, and circulating adipose-tissue-derived substances adipokines contributing to a variety of homeostatic, metabolic, cardiovascular, and oncogenic biologic functions [8]. During the evolution of adipose-tissue-related obesity, the pattern of adipose-tissue secretion, tissue architecture, and global homeostatic regulation alter both qualitatively and quantitatively, resulting in the emergence of a new ensemble of oncogenic factors linked to the development of cancer, thereby amplifying obesity-associated oncogenic risk [10]. An integrated perspective on adipose-tissue-associated obesity promotes a better understanding of the cellular and moleculardeterminants,providingguide for the selection ofobesity-preventive,therapeutic,andintervention-sensitizingstrategies[15].

Sex Hormones and Steroidogenic Pathways

In postmenopausal women, adipose tissue is the primary source of circulating estrogens; conversion occurs in adipose tissue via aromatization of androgens (e.g., testosterone, androstenedione) produced by the ovaries or adrenal glands[8]. Higher levels of circulating estradiol, testosterone, and dehydroepiandrosterone sulphate (DHEAS) are found in obese women than in non-obese women [9]. Age at menarche, number of pregnancies, age at first pregnancy, duration of breastfeeding, age at menopause, and hormone replacement therapy are additional determinants. Adiposity influences the bioavailability of estrogens through sex hormone-binding globulin (SHBG). Additionally, estrogens can impact adipose tissue mass, while obesity can alter the metabolism and action of other hormones implicated in carcinogenesis [10]. The duration of exposure to ovarian hormones, both before and after menarche, as well as reproductive trajectory, plays a significant role in breast cancer development [11].

Oxidative Stress and DNA Damage

Elevated oxidants are implicated in obesity, and oxidative stress is a recognized risk factor for cancer, with high concentrations of oxidants produced in various conditions and at sites such as the liver, adipose tissue, pancreas, and skeletal muscle associated with pathologies such as diabetes, fatty liver, and insulin resistance [21]. Obesity-induced oxidative stress enhances 8-hydroxydeoxyguanosine adduct formation, a key marker of oxidative DNA damage, in different tissues, and systemic oxidative stress is expected to be greater in individuals with obesity [13]. DNA damage is linked with many metabolic dysregulations, and obesity increases this risk, underlining interventions targeting obesity for disease risk reduction [6]. Chemically induced DNA damage in models with severe obesity stimulates metabolic, inflammatory, and proliferative activity, and duration of micronucleus persistence is enhanced in cells from obese mice, suggesting accelerated cytotoxicity [5]. Repair efficiency is further linked to micronucleus production after exposure. Obesity affects nucleotide excision repair capacity and consequently DNA damage induction by environmental carcinogens, and expression levels in tissues of monosodium glutamate-treated mice expose to cigarette smoke, an organ-specific procedure for DNA damage detection, are upregulated in materials from exposed mice, highlighting the influence of obesity on sensitization to chemicals [4]. Genetic obesity modulates gene expression of DNA damage response factors, raising environmental exposure susceptibility and suggesting genetically obese individuals could benefit from prophylactic measures to counteract the elevation of DNA repair genes postcigarette smoke exposure. Mice models with genetically induced obesity undergoing dietary intervention associate pro-oxidative conditions with DNA modifications [5].

Microbiome and metabolic inflammation

Excess adiposity is associated with microbiome structure and function dysbiosis, which have critical influences on metabolic inflammation; these alterations occur independently of energy balance [11]. The gut microbiome contributes to metabolic dysregulation and inflammation in obesity, with a distinct signature characterizing the gut microbiota of patients with metabolic diseases. Germ-free (GF) animals exhibit a lean phenotype and resistance to the metabolic complications brought on by obesity, and has developed an underdeveloped gut-associated lymphoid tissue (GALT) compartment, with fewer immunocompetent cells [12]. Restoration of the microbiome in GF animals increases energy harvest from food, suggesting that the gut microbiome influences metabolism, enabling the host to harvest more energy from the diet. After fecal transplantation from obese or diabetic subjects, GF mice develop obesity or diabetes even when maintained on a low-fat diet, indicating that gut microbiota from obese individuals have the capacity to promote metabolic abnormalities in mice [12]. Lipid storage and energy homeostasis regulation are controlled primarily by ω-3 and ω-6 polyunsaturated fatty acid (PUFA) levels [13]. The predominant microbial species implicated in the activated ω-6 PUFA metabolism pathway and reduced carbohydrate metabolism pathways are Propionibacterium and Streptococcus parasanguinis[9]. Colonization with these species elevates the levels of inflammatory fatty acids in mouse organs and tissues, indicating a potential mechanism linking microbiota alterations to inflammation and metabolic diseases [13].

Cancer Types Associated with Obesity

For several cancers, a robust positive association with overweight or obesity has been shown in observational studies; these studies are summarized using meta-analysis, with a focus on the most thoroughly examined malignancies[8]. The strongest and most consistent associations with obesity-related cancers are listed [8]. Several other common cancers also appear to be causally linked to obesity; for these diseases, the evidence is generally less robust or consistent, and moderator variables such as time-course of obesity, sex, and age require careful consideration [7]. In non-malignant conditions, high BMI is related to increased risk of severe liver disease and osteoarthritis, while there is a growing body of evidence suggesting associations with a number of other cancers warranting further research, including those of the pancreas and kidney, multiple myeloma, and leukemia [7].

Common Cancers with Strongest Associations

Obesity has been estimated to contribute to approximately 25% of cancer deaths in the UK and the USA [2]. Cancers associated with obesity cluster together and share features that reinforce the rationale for targeting the obesity–cancer link [5]. The strongest and most consistent associations involve endometrial, postmenopausal breast, colorectal, kidney, liver, gallbladder, esophageal adenocarcinoma, and pancreatic cancers [14]. Apparent risk thresholds differ markedly across cancer types. For many cancers, notably liver and postmenopausal breast, the risk is strongly modulated by age at exposure [2]. For endometrial, postmenopausal breast, and gallbladder cancers, timing of obesity is critical as well. Other cancers share yet differ from these patterns, emphasizing opportunities for exposure-tracing analytic frameworks [14]. Specific obesity-related cancers can be further tiered based on effect estimates from 97 studies in 16 countries [3]. Weight gain–associated relative risk estimates exceed those for current obesity for pancreatic and gallbladder cancers and for postmenopausal breast cancer prior to menarche, indicating enhanced disease susceptibility during earlier or weight-gain–only obesity windows. For endometrial cancer, the risk associated with faster BMI trajectory is compounded through concomitant levels, or conversely countered through modest concurrent weight losses, supporting the influence of sustained early residence at high adiposity [6]. The multiplicity of cancers with these unifying traits reflects shared biological pathways by which adiposity and its perturbations promote tumorigenesis, a theme further examined in Section 3: Biological mechanisms linking obesity and cancer [1].

Site-Specific Risk Patterns

In developed countries, approximately 25% of cancers may be attributable to obesity or diabetes [10]. The growing incidence of such cancers is a serious public health concern, as obesity is a global epidemic. This section summarizes the cancer types that show the strongest epidemiologic associations with obesity [14]. Within each of these types, organ-specific patterns of risk are documented, and the relationship of these patterns to the mechanistic themes identified continues [15]. The review of association studies emphasizes the importance of statistical modeling in terms of both moderators and specified exposure trajectories. It is critical to document the timing of exposure (I-1) and to establish whether it occurs in childhood, adolescence, or adulthood [15]. The cancers with the strongest overall statistics fit the pattern of exposure to sex hormone–dependent tumors during the same period of life or earlier [7]. Normal human physiology supports such a mechanistic interpretation. Biostatistical analyses of the studies considered reveal that the known sex and age differentials in exposure to obesity, diabetes, and growth factors match not only the overall patterns of association but also the organ-specific variations within each of these cancers [5]. Overall, BMI does not seem to be a direct tumor promoter. Rather, when certain factors appear to be involved, such as abnormal sex hormones, obesity would be a relevant exposure. BMI has a stronger association with endometrial than with ovarian cancer, suggesting that the estrogenic influence of obesity is greater on the endometrium than on the ovary. Another possibility is that obesity facilitates the carcinogenic influence of certain sex hormones [3].

Measurement and Methodological Considerations

Accurate assessment of obesity is crucial for investigating its relationship with cancer risk [2]. While gold-standard methods such as computed tomography and magnetic resonance imaging can measure total body fat and abdominal visceral adipose tissue, these techniques are costly and impractical for large studies [3]. Body mass index (BMI) weight divided by height squared remains the most widely used measure, allowing the classification of individuals as underweight, normal weight, overweight, or obese[10]. Though BMI does not reflect fat distribution, certain measures, such as waist circumference, are correlated with abdominal visceral adipose tissue [16]. Waist circumference has been included in analyses of obesity and cancer because it is modestly associated with population-level obesity an essential consideration in epidemiological studies where BMI has been demonstrated to predict cancer progression and patient outcomes [15]. Mid-life BMI rather than BMI at earlier or later stages of life has emerged as a strong determinant of tumor risk and cancer progression, making the timing of exposure an important consideration [16]. Body weight trajectories across the life course also affect the link between obesity and cancer simultaneously controlling for age at obesity assessment optimally captures these trajectories and can reduce bias arising from reverse causation [13]. Another critical aspect of causation is effect modification by population sub-groups, which can result from biological, environmental, and other factors; having complete stratification data on age, sex, smoking, and other considerations facilitates detection and description of these relationships [23]. Lastly, the approaches to synthesising the evidence vary widely and are of great importance to the eventual interpretation of data and formulation of prevention priorities [22].

Body Mass Index and Alternatives

Adiposity defines an excess of body fat [12]. Various methods estimate it, the most common being the body mass index, or BMI. BMI equals weight in kilograms divided by height in meters squared and classifies individuals into underweight (BMI < 18.5), normal-weight (BMI 18.5–24.9), overweight (BMI 25.0–29.9), and obese (BMI ≥ 30.0). Alternative nomenclature additionally refers to class 1 (BMI 30.0–34.9), class 2 (BMI 35.0–39.9), and class 3 (BMI ≥ 40.0) obesity [3]. More informative than BMI for indicating disease risk are waist circumference and waist-to-hip ratio [12]. Excess visceral fat, indicative of greater risk, corresponds better with these measures than with BMI. Additional metrics gauge long-term extremes, rates of change, and joint exposure [13]. They also define trajectories such as early-onset overweight in childhood versus later increases after menarche [17]. Certain cancers most consistently associate with both BMI and the specified alternatives. Others exhibit diverse patterns, reflecting differential reliance on biological pathways linked to obesity [18]. Early-life exposure corresponds to puberty-related mechanisms, whereas postmenopausal interval aligns with metabolic pathways. Consequently, consideration of lifetime trajectories and timing enriches comprehension of site-specific risks and available preventive strategies [18].

Timing of Exposure: Lifetime BMI Trajectories

Obesity, as measured by BMI, waist circumference, waist-to-hip ratio, or waist-to-height ratio, is a major risk factor for multiple cancer types [18]. A causal relationship is supported by biological mechanisms, similar to those for diabetes and cardiovascular disease. However, formal statistical evidence for a causal effect of obesity on subsequent cancer risk is still lacking [19]. In addition to compound exposure levels measured at specific ages, the timing of exposure to elevated BMI has also been tied to cancer risk. Lifetime BMI trajectories either from early adulthood, adolescence, or even childhood to later adulthood can be modeled, and early-onset exposure likely leads to increased risk; thus, the elevation of obesity during earlier life stages is generally considered more detrimental[14]. Birth weight and various age periods such as childhood, adolescence, young adulthood, middle age, and later adulthood have been investigated for links with risk of subsequent pancreatic cancer, although the specific contributions from the timing of glucose, insulin, and lipid exposure remain to be studied[16]. Additionally, transitional periods of metabolic and hormonal changes, such as puberty and the menopause transition, are also relevant [11].

Confounding, Reverse Causation, and Effect Modification

Epidemiological studies have reported strong direct associations between obesity and several cancers, notably endometrial, postmenopausal breast, colon, renal cell, liver, gallbladder, esophageal adenocarcinoma, and pancreatic malignancies [3]. Several of these sites are influenced by sex hormones, and differential effects by age have been observed in some cases [2]. A mechanistic understanding of the links between excess body fat and specific cancer sites, and of the temporal patterns of exposure, is essential for the development of targeted prevention interventions [5]. Biomarkers related to insulin and the IGF axis, inflammatory pathways, sex hormones, the microbiome, and oxidative stress have been identified as important determinants of adiposity-associated cancer risk [4]. However, except for a few studies on prostate cancer, research has yet to link these candidates to obesity-cancer associations at specific sites. To further characterize these connections, key definitions and measures are detailed in the earlier section [3].

Statistical Approaches in Synthesis

In epidemiology, the size of a data collection influences the choice of statistical methods for analysis and data synthesis [16]. A few large studies, each including tens of thousands or hundreds of thousands of individuals can provide substantial statistical power, more so than several smaller studies combined [20]. One approach is to aggregate summary data from such studies in predefined systematic reviews and meta-analyses, where careful appraisal of adequacy of controls warranted pooled estimates [17]. Alternatively, hazard ratios, risk ratios, or odds ratios with corresponding 95% confidence intervals can be pooled from studies in a wide range of populations, all reflecting the same association between exposure variable and cancer risk [20]. Such an approach is also feasible even though many reports do not include standard errors [3]. By the same token, broad collections of epidemiological reports with statistical parameters widely scattered in the range strengthen the case for causal inference and deepen understanding of biological mechanisms [10]. Pooled estimates offer additional synergies by highlighting other study characteristics that may modify exposure-disease relationships [18].

Clinical and Public Health Implications

The substantial burden of cancer attributable to overweight and obesity is a serious public health challenge for countries in transition across the globe [6]. The links indicated above are direct and provide a clearer understanding of how excess body weight contributes to the pathogenesis of cancer. By identifying the potential mechanisms involved in the earlier stages of carcinogenesis and its promotion, strategies can be developed that target and reverse these processes, thereby preventing the initiation of tumour formation or limiting the progression of established cancers[6]. The increased risk of cancer associated with excess weight is enormous worldwide and continues to expand globally [4]. It compounds the direct and indirect burden of the disease related to cancer in economically developed countries [5]. The need for the wide dissemination of messages related to the avoidance of needless weight gain in combination with systematic actions aimed at enhancing physical activity and improving diet quality represents the provision of strong evidence in support of this health objective [16]. Countries in transition should be particularly vigilant because cancer can absorb a large proportion of the already diminishing health dividends from exhaustive investment in preventive activities directed at non-communicable diseases [2]. Investing in the acquisition of insights to effectively combat obesity can therefore represent a most cost-effective health measure in those geographic regions [15].

Prevention Strategies and Weight Management

Excess body weight is a preventable cause of cancer; preventing weight gain, achieving weight loss, and maintaining weight loss reduce cancer incidence among primary and secondary prevention efforts [19]. Numerous avenues for weight management have been studied; while their effectiveness varies, sufficient studies exist to identify successful strategies that reduce risk among cancer patients and survivors [20]. Low-calorie and very low-calorie diets have short-term effects, with minimal evidence of long-term benefit among cancer patients. Nonpharmacological and pharmacological interventions, bariatric surgery, and intensive lifestyle modifications also have some support [13]. Achieving and maintaining weight loss may decrease or eliminate the increased cancer risk associated with prior obesity [21].

Screening and Risk Stratification

Given the broad spectrum of diseases associated with obesity, targeted screening strategies must account for individual risks related to cancer, mortality, and disability [3]. Future efforts should focus on developing models that predict absolute risk from multiple factors, including age, gender, social determinants, and lifestyle. Integrating estimates from genetic studies into observational models can further enhance accuracy [4]. Excess adiposity is positively associated with 19 common cancer types, but most data originate from North America, Europe, and East Asia, highlighting the need for a better understanding of other populations [2]. Advances in technology afford new opportunities to identify mediators, analyse tumour subtypes, and evaluate adiposity beyond standard measures, thereby strengthening prevention and public health policy [2].

Policy and Population Health Perspectives

In addition to clinical implications, the obesity-cancer link raises important policy issues: cancer prevention through workplace health programs; public health campaigns for healthy weight maintenance; regulation of high-fat, high-sugar food advertising targeted at children; and improvement of built environments and facilities for physical activity [12]. A recent US Preventive Services Task Force statement with a grade B recommendation emphasized that promoting a healthy weight and preventing weight gain are essential components of any cancer prevention strategy; such activities should be implemented automatically where the necessary infrastructure is in place [15]. More generally, evidence of a role for obesity in several common cancers supports public health initiatives targeting obesity risk, detection, and treatment. Evidence of an obesity-cancer association also has implications for population stratification and screening [10]. Given the high prevalence of obesity, the public health burden of obesity-related cancers is substantial, and these cancers account for a significant proportion of cancer incidence in the United States [9]. Despite the limitations of BMI, its ease of measurement makes it a useful tool; incorporating waist measures can enhance risk stratification. The association is similarly plausible for unhealthy weight gain 18 and other obesity definitions [8]. Accordingly, addressing body weight and maintaining a healthy weight should be incorporated into a broader strategy to reduce cancer risk; the benefits of sustained and moderate weight loss on cancer prevention are becoming clearer [7].

Interventions and Therapeutic Considerations

An important area of research concerning obesity and cancer risk involves intervention strategies. Increasing evidence indicates that weight management markedly alters cancer risk. Specifically, weight loss is associated with risk reduction, while weight gain after diagnosis heightens risk [20]. These findings have prompted the investigation of lifestyle, pharmacologic, and surgical weight-loss interventions as potential secondary preventive measures and adjuncts to treatment for patients with cancer and those undergoing cancer therapies [19]. Weight gain remains common during cancer treatment, and some patients remain overweight for several years after diagnosis, which can further exacerbate risk [21]. Structured exercise alone, exercise plus dietary therapy, and exercise plus dietary and behavioral therapy have all been shown to produce significant weight loss in patients with cancer [22]. Pharmacologic therapies, notably glucagon-like peptide-1 analogues, have been associated with substantial weight loss among cancer survivors [13]. Guidelines recommend considering bariatric surgery in patients engaged in cancer screening who have not achieved or maintained adequate weight loss with exercise, dietary changes, or pharmacotheraPy. Weight-reducing strategies should be considered core components of cancer care. At present, however, little is known about aspects of the obesity-cancer link that are relevant for weight-loss intervention, such as the timing of obesity or specific tumor subtypes affected by obesity or weight-supporting interventions [18].

Lifestyle Interventions and Cancer Risk Reduction

The substantial volume of a body of evidence supporting an association between obesity and the risk of common adult cancers raises the possibility that prevention of weight gain and/or reduction in excess weight may decrease cancer risk [15]. If this hypothesis were true, the development of clinically important cancers would constitute an additional rationale for the adoption of lifestyle changes aimed at reducing and maintaining a healthier body weight [13]. A number of studies have addressed the relation between lifestyle changes aimed at inducing weight loss and subsequent cancer risk, primarily focusing on high-risk cohorts [11]. Potential limitations of these studies include the typically modest magnitude and inconsistent nature of reported associations; the potential for reverse causation, especially for hormonally responsive cancers of the breast and endometrium; and the small size of some studies, particularly with respect to effect modifiers [14]. Moreover, most have considered only treatment completion and have; not accounted for improvements in body weight beyond that achieved or for weight-loss-related effects on biomarkers of interest for tumor development [15]. Based on the biological mechanisms summarised earlier, it is hypothesised that the risk of hormonally responsive cancers (e.g. breast, endometrium, prostate) is reduced for men and women assigned to an intensive lifestyle intervention aimed at reducing and maintaining a healthier body weight; specifically, that the registries of these two trials would support an association between weight reduction and a decreased risk of an important subset of common adult cancers [16]. The evidence is most robust for cancers of the breast, endometrium, and prostate, but positive associations with diabetes have also been observed for other common malignancies, including liver, gallbladder, and inflammatory bowel disease [17].

Pharmacologic and Surgical Weight Loss Terms

Current pharmacological medications do not appear to substantially lower incident cancer risk, although post-treatment weight loss may be protective. These drugs increase the risk of adverse events such as pancreatitis and cholecystitis [16]. The cancer biology effects of Conjuated Linoleic Acid use have not yet been clarified [18]. There is substantial interest in the use of Glycogen Synthase Kinase 3, monocarboxylate transporter inhibition, Dipeptidyl Peptidase, Schmidt Sigma-1 Receptor Agonist, and other drugs as a novel cancer therapy, but the current supporting evidence in humans is not compelling [15]. Gastric banding reduces and bypass of the small intestine increases all-cause mortality, but not the external cause mortality and the cancer-specific mortality. Independent of the surgical procedure, for patients undergoing bariatric surgery, a greater BMI decrease following the operation is associated with a lower cancer incidence and risk of a weight-related cancer diagnosis at 1 year after surgery [11]. The long-term impact of bariatric surgery on the development of neoplasms of the esophagus, pancreas, colorectal, and endometrium remains to be elucidated [13].

Impact of Weight Change on Cancer Outcomes

Growing evidence supports a detrimental effect of weight gain on cancer risk and cancer progression after diagnosis [22]. A case-control study from Norway showed a significant increase in overall obesity-related cancer risk with each 5-kg weight increase among the 2,407 cancer cases (relative odds 1.25, 95% confidence interval 1.14–1.36) [23]. Specific obesity-related cancer sites such as postmenopausal breast, endometrium, and pancreatic tumor types were likewise affected. A comprehensive meta-analysis of worldwide records reported strong links between increased body size and cancer across 19 organ sites [3]. Differential effects by sex, age, and timing of exposure further elucidate these associations.

Knowledge Gaps, Controversies, and Future Directions

Causal mechanisms through which obesity promotes cancer risk remain incompletely understood [4]. Further experimental work is needed to establish whether obesity induces genomic, metabolic, and microenvironmental changes that drive specific cancer types [17]. Because most observational studies show that obesity, particularly during adulthood, increases risk for several tumour types, it will be important to investigate if weight loss following pharmacological or surgical interventions reduces risk in a time-dependent manner [5]. Defining the timing, duration, and extent of obesity that underpins cancer risk is also a research priority to inform screening strategies. In addition to remaining translation from the private to the public domain, global institutional or governmental body information on cancer incidence and obesity prevalence is needed to identify whether obesity is a risk factor for cancer at particular sites in parts of the world where obesity prevalence is lower, such as Africa and India. Finally, longitudinal studies in populations of non-European origin can help to identify specific genetic variants that may modulate cancer risk associated with obesity [13]. Some disputes in the literature also merit resolution [6]. Several studies that evaluated sex hormones, the insulin–IGF axis, and circulating adipokines have yielded inconsistent results, most likely owing to differences in the cohort studied, the timing of blood sampling, and the subsequent detection techniques used [16]. Whether inflammation, obesity-induced oxidative stress, dysbiosis, or chronic endoplasmic reticulum stress constitute components of the obesity-cancer nexus that require further investigation remains to be determined[11]. The discovery that apoptosis-resistant senescent adipocytes promote chronic inflammation and contribute to obesity and other metabolic diseases also raises the possibility that senescence, a developmentally programmed form of cell death, supports the formation of an environment conducive to the emergence of cancers. Animals exposed to high-fat diets or genetically prone to obesity develop not only obesity but also glucose intolerance, fatty liver, and steatosis. Whether the cancer-promoting potential associated with these co-morbidities is greater than exposure to obesity alone is unclear and remains to be investigated [12].

Unresolved Mechanisms

Research on biological mechanisms linking obesity to cancer risk remains incomplete. No common mediator candidate has been identified, although several of the known mechanisms may be nuanced or incomplete [10].

Furthermore, their role has not been elucidated at all for many cancer types, and sophisticated understanding remains lacking for many common cancers, including the four with the strongest association's oesophageal, endometrial, pancreatic, and liver cancers [9]. Using high-precision epidemiological and transcriptomic data from 286 distinct tissue types and cell states, Motohara and colleagues discovered multisite mediators of BMI–cancer risk associations[8]. Using colocalisation and multivariable Mendelian randomisation, they found that of 794 cancer-associated loci and 1816 tissues and cell states 426 loci across 118 tissues and cell states meet anticancer criteria. Of total tissue/cell pairs, only 31 are pleiotropic. Seven patterns were established for the same candidate gene acting on multiple cancers in independent tissues [20]. IAMs such as copy number variations on chromosome 3q22 affecting lung adenocarcinoma risk and expression of GLP-1 receptor-coupled signalling pathway-related genes are capable of guiding cancer prevention [8].

Translational Research Priorities

Several areas warrant translational research effort. Understanding biological mechanisms influencing cancer risk in prospective-observation-experimental studies represents a translational gap; a better understanding would permit exploration of biomarkers, drug targets, and modulators for prevention and prognosis [10]. Delineating differential effects according to type, timing, and sites of obesity using repeated measures continues to offer translational potential [12]. Translating the consistency and strength of obesity-cancer risk associations into clinical practice remains to be fully realized; incorporation of lifespan trajectories, underlying mechanisms of cancer and corresponding timing into clinical guidelines would be useful [11]. Whether status and trends in obesity and overweight-attributable cancers into routine cancer-surveillance systems could strengthen policy response is unclear [13]. Available pharmacological treatments, combined with existing preventive lifestyle s, support population-level recommendations linking weight and risk; further research can determine how changed weight modifies risk and cancer outcome [15]. Conducting comparable studies across biomedical, measurement and epidemiological disciplines in obesity-attributable cancers offers opportunities to reduce or eliminate weight disparities in the prevention and control of cancer and other major chronic diseases [13]. In summary, the epidemiological links between obesity and cancer remain apparent across biological mechanisms, cancer sites, and Canadian measurements and risk reductions [17]. The direct effect of obesity on certain cancers, together with lifestyle approaches for weight loss and prevention of weight gain, underscore the direct link to these major chronic disorders. Cancer screening remains a critical factor, with the main effect arising from excess weight being an increased likelihood of developing cancer rather than an increased chance of dying from it. Social and environmental factors continue to increase the prevalence of childhood obesity, underlining the need to assist the population in adopting an obesity-and-weight-curbing lifestyle and implementing supportive policies [17].

Ethical, Equity, and Global Perspectives

When considering obesity and obesity-related disease risk, it is important to understand that health is not distributed equally throughout the population and that people living with obesity do not have the same level of risk [24]. For instance, health inequalities exist along social lines, including variation by ethnicity, and the likelihood of living with health-related obesity also varies, underscoring the social determinants of health and the role of the associated structural and economic systems [19]. The proportion of people living with obesity in most high-income countries is significantly higher among lower-income and minority populations [18]. Moreover, access to the resources and treatments that prevent or successfully treat obesity is often unavailable to low-income populations, and negative messages targeting obesity may also affect those who are not living with it, leading to detrimental outcomes [20]. Such inequities and social injustices should be considered in all preventive activities and clinical practice. Recent systematic assessments find that possible ethnic differences in cancer risk appear to occur primarily within areas of specific populations (e.g. Indigenous People) and that any ethnic differences observed are attributable to other social determinants of health [22]. Despite the emerging evidence pointing to a causal mechanism between weight status and cancer development, these patterns are not universally observed worldwide, with notable geographic variation. It is critical to document such patterns in LMCs where cancer registries are first being established, for vitality, investment prioritization and prevention strategy [22].

Disparities in Obesity and Cancer Burden

Obesity increases cancer susceptibility, and the major obesity-related cancers disproportionately affect individuals globally [22]. Despite the abundance of evidence demonstrating a causal association for these diseases, obesity-related stigma often prevents vulnerable groups from clinical care opportunities such as preventive health consultations, new screening guidelines, and the support needed to lower the risk of developing one of these diseases with an obesity-related mechanism. Moreover, individuals with any form of overweight are more likely to develop such diseases. Understanding the mechanisms behind these disparities is crucial for effective management

[22]. Many of these patients are particularly ignored by health systems or preventive campaigns, and because these are the groups with less access to health care alternatives that prevent the progression or developing such diseases, the public health communication is often negatively adjusted. Thus, the concept of responsible communication related with overweight and obesity and the concentration of the preventive campaigns on the groups that are more likely to have preventive measures and facilities for treatment are fundamental [23]. High body mass index modifies the incidence of numerous types of cancer, although the pathophysiological mechanisms associated with such events are not yet fully elucidated [24, 25, 26]. Several hypotheses integrate the discussion on the relationship between obesity and the risk of developing various cancers, as well as the distinctive characteristics of the cancer growth process [24]. Metabolic dysregulation, presence of a subclinical inflammatory state, and consequently a perturbed immunological pattern seem to be key factors in modulating the size of risk [23]. New data addressing the use of drugs for insulin resistance, especially metformin, and a greater understanding of the function of the coadjuvants of tumor growth and their therapeutic targeting contribute to a better evaluation of the preventive and therapeutic strategy for the set of obesity-associated cancers [24].

Global Variation in Risk and Interventions

Excess body weight increases the risk of certain cancers, but two contrasting global patterns emerge from currently available data [24]. High rates of overweight and obesity in North America and some regions of Oceania have been consistently associated with increased risk for a range of obesity-related cancers [24]. By contrast, increased risk is not observed in Europe and only modestly in other regions, despite similar rates of overweight and obesity. Available data indicate that patterns of weight gain, the timing of exposure, and variations in body fat distribution and composition influence risk; these possible mediators and moderating factors probably interact with evolving societal and environmental influences to shape regional variation and remain important priorities for further investigation [3]. Another important distinction can be drawn between cancer risk in countries at different stages of the obesity epidemic [27, 28, 29]. In countries with long-standing elevated rates of overweight and obesity in both men and women, common cancers remain highly relevant, whereas in countries with lower and more variable rates such as Japan, the United Kingdom, and most of sub-Saharan Africa, other cancers predominate [25]. Current indications suggest a similar disconnect among early intervenors, where the relationship between obesity and cancer risk diffuses despite persisting patterns of excess weight gain in the general population, underlining the need to identify interacting societal and environmental factors that influence the disease [25, 30].

CONCLUSION

Obesity represents one of the most modifiable yet complex determinants of cancer risk. The convergence of epidemiologic, clinical, and molecular evidence confirms that excess body fat contributes to carcinogenesis through hormonal imbalance, metabolic dysregulation, oxidative stress, and chronic inflammation. Adipose-derived cytokines, insulin–IGF signaling, and altered sex hormone metabolism collectively foster an environment conducive to tumor initiation and progression. Yet, obesity is not merely a biological condition; it is deeply embedded within social, economic, and environmental systems that shape individual risk and access to care. Lifestyle modification remains the cornerstone of prevention and management. Structured exercise, dietary regulation, and behavioral support have demonstrated measurable reductions in weight and associated cancer risk, particularly for hormonally responsive cancers such as those of the breast, endometrium, and prostate. Pharmacological agents and bariatric surgery show additional promise for patients unresponsive to conventional interventions, though long-term safety and cancer-specific outcomes require further clarification. Future studies should elucidate the timing, duration, and reversibility of obesity-related cancer risk and identify molecular biomarkers that predict responsiveness to weight reduction. Globally, the burden of obesity-related cancers mirrors widening health inequities. Low- and middle-income countries now face dual epidemics of undernutrition and obesity, yet they lack robust surveillance systems and equitable access to cancer care. Addressing these disparities demands coordinated policy responses that integrate obesity prevention into national cancer control strategies, promote responsible public health communication, and reduce stigma associated with body weight. Ultimately, the interplay between obesity and cancer underscores the necessity of a multidisciplinary approach that spans biology, behavior, and policy. Effective solutions will require sustained political commitment, investment in translational research, and a strong equity lens to ensure that prevention and treatment reach all populations. By treating obesity as both a biomedical and social determinant of cancer, the global community can make significant strides toward reducing cancer incidence and improving population health outcomes worldwide.

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