ejpmr, 2025, 12(10), 208-213

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

SJIF Impact Factor 7.065

Research Article

ISSN (O): 2394-3211

ISSN (P): 3051-2573

THE LINK BETWEEN OBESITY AND CANCER RISK

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Article Received on 21/08/2025 **Article Revised on 11/09/2025** **Article Accepted on 01/10/2025**

ABSTRACT

Background: Obesity has become a global public health concern and is increasingly recognized as a major modifiable risk factor for cancer. Excess adiposity contributes to carcinogenesis through hormonal imbalances, metabolic dysfunction, and chronic inflammation. This study aimed to examine the association between obesity and cancer risk in adults. **Methods:** A cross-sectional analytical study was conducted over six months among 1,200 adults recruited using stratified random sampling. Data were collected through structured questionnaires and anthropometric measurements. Body mass index (BMI) was calculated and categorized according to World Health Organization criteria. Cancer status was determined by self-reported physician diagnoses confirmed by medical documentation when available. Logistic regression was used to estimate odds ratios (OR) and 95% confidence intervals (CI), adjusting for age, sex, smoking, physical activity, and comorbid conditions. **Results:** The prevalence of obesity was 34.8%, while 29.6% were overweight and 32.1% had normal weight. A total of 96 participants (8.0%) reported a confirmed cancer diagnosis. Cancer prevalence increased progressively with BMI, from 4.2% in normal-weight individuals to 12.7% in obese participants. Multivariable logistic regression showed that obesity was associated with a significantly higher cancer risk (OR = 3.28, 95% CI: 1.92–5.60, $p < 0.001$), while overweight showed a non-significant trend (OR = 1.74, 95% CI: 0.98–3.08, $p = 0.058$). Age was also an independent predictor of cancer (OR = 1.05 per year, $p < 0.001$). **Conclusion:** Obesity was strongly associated with increased cancer risk, independent of age, sex, and smoking. The findings emphasize the importance of obesity prevention and management as integral components of cancer control strategies. Targeted interventions promoting healthy lifestyles and weight reduction are essential to mitigate the dual burden of obesity and cancer.

KEYWORDS: Obesity, Cancer risk, Cross-sectional study, Body mass index, Public health.

BACKGROUND

Obesity has emerged as one of the most pressing public health concerns worldwide, with its prevalence steadily increasing over the past few decades. It is no longer considered a problem restricted to developed nations but has become a global epidemic affecting populations across different age groups, socioeconomic statuses, and regions. This growing burden has been strongly associated with a range of chronic conditions, including diabetes, cardiovascular diseases, and more recently, cancer (Pati et al., 2023).

The association between obesity and cancer risk is an area of intense research, as evidence indicates that excess body fat may contribute to both the initiation and progression of malignant tumors. Obesity is characterized by abnormal or excessive fat accumulation that impairs health, and this state can create a biological environment conducive to carcinogenesis. This includes hormonal imbalances, metabolic dysfunction, and chronic inflammation, all of which may play a role in increasing cancer susceptibility (Ingram et al., 2025).

One of the key pathways linking obesity to cancer is the alteration of hormone levels. Adipose tissue functions as an active endocrine organ, producing hormones such as estrogen, insulin, and leptin, which can promote cellular proliferation and inhibit apoptosis. These hormonal changes are particularly relevant in cancers such as breast, endometrial, and prostate cancer, where hormone regulation significantly influences tumor development (Shi et al., 2024).

Chronic inflammation associated with obesity is another critical factor. Excess adipose tissue often triggers the release of inflammatory cytokines, creating a state of low-grade systemic inflammation. This persistent inflammatory response can cause DNA damage, promote angiogenesis, and support the tumor microenvironment,

thereby increasing the likelihood of cancer development (Avgerinos et al., 2019).

Metabolic alterations seen in obese individuals also contribute to cancer risk. Insulin resistance and hyperinsulinemia, commonly present in obesity, can activate signaling pathways that stimulate cell growth and survival. Such metabolic disturbances not only enhance tumor progression but may also reduce the body's ability to suppress abnormal cell growth effectively (Malik et al., 2024).

The link between obesity and cancer risk is also influenced by lifestyle and behavioral factors. Diets high in calories, sedentary lifestyles, and lack of physical activity not only contribute to obesity but independently increase the likelihood of developing certain cancers. Conversely, maintaining a healthy weight and adopting an active lifestyle have been shown to reduce the risk of cancer, emphasizing the importance of prevention strategies (Jang et al., 2024).

From an epidemiological perspective, research has demonstrated associations between obesity and several cancer types, including colorectal, pancreatic, kidney, esophageal, and liver cancers. The strength of these associations varies, but the evidence consistently points toward a heightened risk in obese individuals compared to those with a healthy body weight. This highlights obesity as a modifiable risk factor that can significantly impact cancer prevention (Vick et al., 2024).

Beyond cancer initiation, obesity has also been linked to worse outcomes and prognosis in patients diagnosed with cancer. Obese patients often experience higher rates of cancer recurrence, reduced treatment effectiveness, and poorer overall survival. Factors such as altered pharmacokinetics, treatment complications, and the impact of obesity-related comorbidities contribute to these unfavorable outcomes (Krupa-Kotara & Dakowska, 2021).

Addressing obesity as part of cancer prevention strategies requires a multifaceted approach that includes public health policies, education, and healthcare interventions. Promoting balanced nutrition, increasing physical activity, and creating supportive environments for healthy lifestyle choices are essential steps in reducing obesity prevalence and, consequently, cancer risk (Watts et al., 2024).

Given the rising global burden of both obesity and cancer, understanding the mechanisms linking the two has become increasingly important. Research in this area not only helps identify at-risk populations but also provides valuable insights for targeted interventions and treatments. By exploring this link, healthcare systems can develop more effective strategies for prevention, early detection, and management, ultimately reducing the impact of obesity-related cancers on individuals and societies (Hoehn, 2021).

METHODOLOGY

Study design

The study was designed as a cross-sectional analytical survey to examine the association between obesity and cancer risk in adults. Data were collected over a six-month period. The cross-sectional approach enabled the simultaneous assessment of obesity status, cancer history, and related sociodemographic and lifestyle variables.

Study population and eligibility criteria

The study population consisted of adult men and women attending outpatient clinics. Participants were eligible if they were between 18 and 70 years of age and consented to take part in the study.

Exclusion criteria included pregnancy, severe cognitive or psychiatric illness that limited the ability to respond, and missing or incomplete anthropometric or cancer history data.

Sampling and sample size

A stratified random sampling technique was applied to recruit participants across different age groups and both sexes. The minimum required sample size was calculated using the formula for cross-sectional studies, assuming a prevalence of obesity of 30%, an estimated cancer prevalence of 8% among adults, a 95% confidence level, and 5% margin of error. The calculated sample size was 1,050 participants, and to account for potential incomplete data, a total of 1,200 individuals were included in the final analysis.

Data collection procedures

Data were collected using a structured, interviewer-administered questionnaire along with physical measurements. The questionnaire captured sociodemographic characteristics (age, sex, marital status, educational level, and income), lifestyle factors (smoking, alcohol use, and physical activity), and self-reported cancer history confirmed by medical records when available. Height and weight were measured using calibrated equipment, and BMI was calculated as weight in kilograms divided by height in meters squared (kg/m^2).

Variables and measurements

The primary exposure variable was obesity, defined according to World Health Organization (WHO) criteria: underweight (<18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), overweight (25–29.9 kg/m^2), and obese (≥ 30 kg/m^2). The main outcome variable was cancer status, defined as self-reported physician-diagnosed cancer, confirmed by medical documentation when possible. Additional variables included smoking, physical activity, comorbidities (diabetes, hypertension), and family history of cancer.

Data quality assurance

All interviewers were trained in data collection and measurement techniques. The questionnaire was piloted on 50 participants not included in the study, and adjustments were made to improve clarity. Anthropometric equipment was calibrated daily, and 10% of questionnaires were rechecked by a supervisor for consistency and accuracy.

Statistical analysis

Data were entered into a secure database and analyzed using SPSS version 26. Descriptive statistics were calculated for all variables, with means and standard deviations for continuous variables and frequencies and percentages for categorical variables. The prevalence of obesity and cancer was reported with 95% confidence intervals. Associations between obesity categories and cancer risk were examined using chi-square tests and logistic regression analysis.

Both crude and adjusted odds ratios (ORs) with 95% confidence intervals were reported. Multivariable models adjusted for age, sex, smoking status, physical activity, and comorbid conditions. A p-value <0.05 was considered statistically significant.

Ethical considerations

The study protocol was reviewed and approved by an institutional ethics review committee. Participation was voluntary, and written informed consent was obtained from all participants. Data confidentiality was maintained, and all personal identifiers were removed prior to analysis.

RESULTS

A total of 1,200 adults participated in the study. The mean age of participants was 44.7 ± 12.6 years, and 52.1% were female. The overall prevalence of obesity was 34.8%, while overweight accounted for 29.6%, normal weight 32.1%, and underweight 3.5%. Among the total participants, 96 individuals (8.0%) reported a physician-confirmed diagnosis of cancer. The distribution of cancer cases was higher in obese participants compared with those of normal weight.

Table 1. Sociodemographic characteristics of participants (n = 1,200).

Variable	Category	Frequency (n)	Percentage (%)
Age group (years)	18–29	210	17.5
	30–49	480	40.0
	50–70	510	42.5
Sex	Male	575	47.9
	Female	625	52.1
Marital status	Single	340	28.3
	Married	790	65.8
	Widowed/Divorced	70	5.9
Education	Below secondary	260	21.7
	Secondary	430	35.8
	University+	510	42.5

The largest age group was 50–70 years (42.5%), reflecting a relatively older sample. Females constituted slightly more than half of the participants (52.1%). Most participants were married (65.8%) and had at least a secondary education (78.3%).

Table 2: Distribution of BMI categories among participants.

BMI Category	Frequency (n)	Percentage (%)
Underweight (<18.5)	42	3.5
Normal weight (18.5–24.9)	385	32.1
Overweight (25–29.9)	355	29.6
Obese (≥30)	418	34.8

More than one-third of participants were classified as obese (34.8%), and almost 30% were overweight. Together, 64.4% of the sample had excess body weight, while only 32.1% were in the normal range.

Table 3: Prevalence of cancer by BMI category.

BMI Category	Total (n)	Cancer cases (n)	Cancer prevalence (%)
Underweight (<18.5)	42	2	4.8
Normal weight (18.5–24.9)	385	16	4.2
Overweight (25–29.9)	355	25	7.0
Obese (≥30)	418	53	12.7
Total	1,200	96	8.0

Cancer prevalence increased progressively with higher BMI. The highest prevalence was observed in obese participants (12.7%), nearly three times that of normal-weight individuals (4.2%). Overweight individuals also showed elevated prevalence (7.0%).

Table 4: Association between obesity and cancer risk (Logistic regression analysis).

Variable	Odds Ratio (OR)	95% CI	p-value
Overweight vs. Normal	1.74	0.98–3.08	0.058
Obese vs. Normal	3.28	1.92–5.60	<0.001
Age (per year)	1.05	1.03–1.07	<0.001
Female vs. Male	1.22	0.78–1.91	0.378
Smoking (Yes vs. No)	1.41	0.87–2.27	0.162

After adjusting for age, sex, and smoking, obesity remained significantly associated with cancer risk. Obese individuals had more than a threefold higher odd of cancer compared with normal-weight participants (OR 3.28, 95% CI 1.92–5.60, $p < 0.001$). Overweight participants showed a trend toward higher risk (OR 1.74, $p = 0.058$), though it did not reach statistical significance. Increasing age was also an independent predictor of cancer (OR 1.05 per year, $p < 0.001$).

DISCUSSION

The findings of this cross-sectional study demonstrated a clear and significant association between obesity and cancer risk. Among the 1,200 participants, the prevalence of cancer was notably higher in obese individuals (12.7%) compared to those with normal weight (4.2%). Logistic regression analysis revealed that obesity was associated with more than a threefold increase in cancer odds after adjusting for potential confounders. These results are consistent with previous evidence suggesting that excess adiposity plays a key role in cancer development and progression (Pati et al., 2023).

Our results highlight that the relationship between obesity and cancer is dose-dependent, as prevalence increased progressively from normal weight to overweight and obesity. This trend aligns with the biological plausibility that greater adiposity contributes to cumulative carcinogenic effects through hormonal and metabolic dysregulation (Ingram et al., 2025). While overweight participants showed a higher cancer prevalence (7.0%), the association was not statistically significant in multivariable models, indicating that the most substantial risk increase occurs once obesity thresholds are reached.

One possible explanation for these findings is the role of adipose tissue as an endocrine organ. Obese individuals have elevated circulating estrogen, insulin, and leptin levels, which can promote tumor growth and impair apoptosis. This mechanism is particularly relevant in hormone-sensitive cancers such as breast and endometrial cancer (Shi et al., 2024). Our study, although not site-specific, supports the general hypothesis that obesity-driven hormonal imbalance contributes significantly to cancer risk.

Chronic inflammation represents another important pathway linking obesity and cancer. Adipose tissue expansion promotes the release of pro-inflammatory cytokines, leading to DNA damage and an environment conducive to tumor initiation (Avgerinos et al., 2019). The higher cancer prevalence observed among obese participants in our study could, in part, reflect the cumulative impact of persistent low-grade inflammation, which has been strongly implicated in carcinogenesis.

In addition to hormonal and inflammatory factors, obesity is commonly associated with metabolic dysfunctions such as insulin resistance and hyperinsulinemia. These metabolic disturbances stimulate pathways that support uncontrolled cellular proliferation and inhibit apoptosis (Malik et al., 2024). The elevated risk observed in obese individuals in this study is consistent with these metabolic explanations, highlighting the importance of addressing metabolic health in obesity management strategies to mitigate cancer risk.

Lifestyle behaviors further contribute to the observed associations. Diets high in energy-dense foods, combined with physical inactivity, exacerbate obesity and independently increase cancer risk (Jang et al., 2024). Although this study did not directly measure dietary intake or physical activity, the strong link between obesity and cancer risk in our findings underscores the need for comprehensive lifestyle interventions targeting multiple risk factors simultaneously.

Interestingly, our results showed that age was an independent predictor of cancer, with each additional year of life slightly increasing the odds of diagnosis. This is consistent with epidemiological evidence that cancer incidence rises with age due to the accumulation of genetic mutations and prolonged exposure to risk factors (Watts et al., 2024). The combined effect of aging and obesity may explain why the prevalence of cancer was highest in the older, obese subgroup in our study.

The findings also revealed that sex and smoking status were not significantly associated with cancer risk after adjustment. This suggests that the effect of obesity on cancer risk was robust across both genders and not substantially confounded by smoking in this sample. However, other studies have reported sex-specific

differences, particularly in cancers influenced by hormonal pathways (Vick et al., 2024). Future studies should therefore stratify analyses by cancer type and sex to explore these nuances more thoroughly.

Our results align closely with the work of Krupa-Kotara and Dakowska (2021), who found that obesity was strongly associated with increased cancer risk across multiple populations. Similarly, a large body of epidemiological evidence supports obesity as a modifiable risk factor for cancer incidence and mortality (Pati et al., 2023; Malik et al., 2024). By demonstrating consistent associations in a cross-sectional sample, our study adds further weight to the global evidence base linking obesity with cancer.

Beyond incidence, obesity has been shown to negatively affect cancer prognosis, with obese patients often experiencing reduced treatment effectiveness and higher recurrence rates (Krupa-Kotara & Dakowska, 2021). While our cross-sectional design did not allow assessment of prognosis, the higher prevalence of cancer among obese participants in this study underscores the need for proactive obesity management in both prevention and post-diagnosis care.

From a public health perspective, these findings emphasize the urgent need for strategies targeting obesity reduction as a means of cancer prevention. Interventions that promote balanced nutrition, regular physical activity, and weight management are essential components of comprehensive cancer control programs (Watts et al., 2024). Incorporating obesity prevention into cancer policy frameworks could help reduce the burden of both conditions simultaneously.

It is important to note that while the association between obesity and cancer risk was significant, causality cannot be inferred due to the cross-sectional design. Longitudinal studies are required to confirm temporal relationships and better understand the progression from obesity to cancer (Hoehn, 2021). Nonetheless, the strong associations observed in our study align with mechanistic and prospective evidence from prior research, lending confidence to the findings.

Another limitation was reliance on self-reported cancer diagnoses, though these were confirmed with medical documentation when available. This may have led to underreporting of some cancer cases. Similarly, unmeasured lifestyle factors such as dietary intake and alcohol use may have introduced residual confounding. Despite these limitations, the study benefited from a large sample size, rigorous sampling, and standardized anthropometric measurements, strengthening the reliability of the results.

The results of this study also highlight the potential for obesity-targeted interventions to reduce cancer disparities. Given that obesity prevalence is higher in certain populations, addressing obesity could contribute to narrowing inequalities in cancer incidence (Ingram et al., 2025). Future research should therefore prioritize high-risk groups and evaluate the effectiveness of integrated obesity and cancer prevention strategies.

In conclusion, the study confirmed that obesity was significantly associated with increased cancer risk, independent of age, sex, and smoking. These findings reinforce the growing body of evidence that obesity is a major modifiable risk factor for cancer. Addressing obesity at both individual and population levels is crucial to reducing the overall burden of cancer and improving public health outcomes (Pati et al., 2023; Watts et al., 2024).

CONCLUSION

This cross-sectional study demonstrated a significant association between obesity and cancer risk. Obese individuals were more than three times as likely to report a cancer diagnosis compared to their normal-weight counterparts, even after adjusting for confounding variables. The results confirmed a dose–response relationship, with cancer prevalence progressively increasing from normal weight to overweight and obesity. These findings align with the broader body of evidence that implicates excess adiposity as a critical modifiable risk factor for cancer development. While causality cannot be established due to the study design, the observed associations strongly suggest that obesity prevention and management should be integral components of cancer control strategies. Addressing obesity not only has implications for reducing cancer incidence but also contributes to improving overall population health outcomes.

RECOMMENDATIONS

1. **Integrate obesity prevention into cancer control programs**: National and regional cancer strategies should explicitly include obesity reduction through lifestyle modification as a key component of prevention.
2. **Promote healthy lifestyles at the population level**: Public health campaigns should emphasize balanced nutrition, physical activity, and weight management to reduce the dual burden of obesity and cancer.
3. **Target high-risk groups with tailored interventions**: Populations with higher obesity prevalence, such as older adults and those with comorbidities, should be prioritized for preventive programs and early cancer screening.
4. **Enhance healthcare provider training**: Clinicians should be equipped with skills and resources to counsel patients on weight management and its link to cancer risk, ensuring obesity is addressed as part of routine care.
5. **Support longitudinal and mechanistic research**: Future studies should adopt prospective designs to establish causal pathways, examine site-specific

cancers, and assess the impact of obesity interventions on cancer incidence and prognosis.

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