

Original article

The Effect of Smoking, Gender and Histological Type of Cancer on Body Mass Index in Lung Cancer Patients: A Cross-Sectional Study

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Abstract: *Objective* — This study aimed to investigate the association of smoking status, gender, cancer histological subtype and stage with body mass index (BMI) in patients with lung cancer.

Methods — Our cross-sectional study was conducted on 400 patients with histologically confirmed lung cancer at Tishreen University Hospital, Lattakia, Syria, between January and December 2023. Demographic data, smoking status, cancer histological subtype and stage were collected from medical records. BMI was calculated as weight (kg)/height (m²). Comparisons were performed using independent t-test, chi-squared test, and one-way ANOVA with post hoc analysis. Two-way ANOVA tested for interaction effects, while multivariate linear regression adjusted for age and gender identified independent predictors of BMI.

Results — The mean age was 60.4±9.6 years; 300 patients (75%) were men and 100 were women (25%). The mean BMI was 20.0±4.3. Men and smokers had significantly lower BMI values than women and nonsmokers ($p<0.001$). Significant differences were observed between histological subtypes of cancer. Patients with small cell lung cancer (SCLC) had the lowest mean BMI ($p<0.001$). BMI progressively decreased with increasing disease stage ($p<0.001$). Multivariate linear regression analysis confirmed that smoking, advanced stage of cancer, and histological type of SCLC were independent predictors of lower BMI, while increasing age was associated with a slightly higher BMI.

Conclusion — Lower BMI in lung cancer patients is statistically significantly associated with smoking, male gender, and advanced stage and aggressive histological subtypes of cancer, highlighting the importance of early nutritional support and further longitudinal studies.

Keywords: lung neoplasms, body mass index, smoking, histology, cachexia.

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Introduction

The increasing prevalence and high mortality of lung cancer is still one of the most significant challenges to global health [1]. The World Health Organization (WHO) estimates that more than 2 million new cases of lung cancer are diagnosed annually, with approximately 1.8 million deaths from lung cancer each year, accounting for nearly 18% of all cancer deaths [2]. Advances in early diagnosis, imaging, targeted therapy, and immunotherapy have had little impact on the overall 5-year survival rate for lung cancer, which remains alarmingly low [3]. The main factors contributing to this scenario are late diagnosis, high cancer aggressiveness, and the complex and diverse biological behavior of the disease [4]. Therefore, it is crucial to understand the individual factors influencing disease progression and prognosis for the purposes of diagnosis, risk assessment, and subsequent selection of treatment strategies [5].

Body mass index (BMI) is a common anthropometric clinical marker of nutritional deficiency [6]. It is measured by the formula: weight in kilograms divided by the square of the height in meters (kg/m²) [6]. Underweight and overweight or obesity are BMI categories that have been shown to either increase or decrease

the risk of lung cancer, its biology, and associated clinical outcomes [7]. Epidemiological studies have shown an inverse association between BMI and lung cancer among smokers with more advanced disease, as well as a disproportionately high risk of adverse outcomes among those with a lower BMI [8]. This association has been attributed to impaired immune surveillance, increased systemic inflammation, and cancer cachexia [9]. On the other hand, in the case of non-small cell lung cancer (NSCLC), breast cancer, and lung adenocarcinoma, some studies describe the so-called *obesity paradox*, which demonstrates increased survival in overweight patients vs. normal or underweight patients [10]. This paradox is due to increased physiological reserves, altered chemotherapy pharmacokinetics with preserved inflammatory responses, and weakened primary defenses [11].

Smoking is the primary etiologic factor in lung cancer, accounting for nearly 90% of cases worldwide. In addition to its carcinogenic role, smoking induces chronic systemic inflammation, metabolic dysregulation, and oxidative stress, which contribute to progressive weight loss and the development of cancer-associated cachexia. These mechanisms may partially explain the strong association between smoking and decreased BMI observed in lung cancer patients [6, 12].

Of these, the most concerning is the association between BMI and smoking, which tends to lower BMI, thereby increasing the risk of malnutrition and ultimately contributing to a shortened lifespan [6, 13]. Lung cancer has four histological subtypes of cancer: adenocarcinoma, squamous cell lung cancer, small cell lung cancer (SCLC), and large cell carcinoma [14]. Each lung cancer subtype varies in its characteristics and biological properties. This is caused partially by differences in the cancer subtypes presented by patients, as well as in BMI, cachexia, inflammation, and metabolic dysfunction [8]. For example, patients with small cell and squamous cell lung cancer tend to experience rapid weight loss, compared with patients with lung adenocarcinoma [15].

Despite understanding of these individual elements, there are very few studies examining the combined effects of BMI, smoking, gender, cancer subtype, and cancer stage within the same study population [16]. Advancing understanding of the important interactions among all of these factors could significantly improve patient prognosis and develop personalized support strategies, such as nutritional optimization, weight management, and intensification therapy [17, 18].

This study examines multiple relationships between BMI and associated factors, such as gender, age, and smoking, in a population of 400 lung cancer patients. We further explore the potential impact of smoking and histological type of cancer on BMI. The goal of this study was to develop a nutrition-based lung cancer therapy model for prognosis, assessment, and treatment planning based on an understanding of these critical, clinically relevant relationships.

Material and Methods

Study setting

This cross-sectional study was conducted at Tishreen University Hospital, Lattakia, Syria, from January through December 2023.

Study design and participants

The study included 400 adult patients (≥ 18 years of age) with histologically confirmed primary lung cancer. Patients met inclusion criteria if they had a complete physical examination and a documented active diagnosis of lung cancer in their medical records. Exclusion criteria included a history of other malignancies, severe comorbidities affecting nutritional status, incomplete clinical information, or missing anthropometric data.

Study variables

- Demographic data: age, gender;
- Smoking status: smokers vs. nonsmokers;
- Anthropometric data: height and weight were measured at admission using a calibrated stadiometer and digital scale. BMI was calculated as weight (kg)/height (m²) and classified according to WHO categories: moderate thinness (16.0-16.99), mild thinness (17.0-18.49), normal weight (18.5-24.9), overweight (25.0-29.9), and Class I obesity (30.0-34.9);
- Cancer histology: classified into SCLC and NSCLC, including adenocarcinoma, squamous cell lung cancer, and large cell carcinoma;
- Cancer stage: determined according to the TNM classification (stages I-IV).

Table 1. Baseline characteristics of the study population

Variable	N	%
Gender:		
Male	300	75%
Female	100	25%
Cancer type:		
Adenocarcinoma	147	36.75%
Large cell carcinoma	28	7%
Small cell lung cancer	120	30%
Squamous cell lung cancer	105	26.25%
Smoking:		
Smoker	317	79.25%
Nonsmoker	83	20.75%
BMI category:		
Mild thinness	196	49%
Moderate thinness	54	13.5%
Normal weight	95	23.75%
Overweight	37	9.25%
Class I obesity	18	4.5%
Cancer stage		
1	2	
2	76	20.1%
3	218	57.67%
4	82	21.69%

Mean age: 60.38 $\pm$ 9.55 years; mean BMI: 20 $\pm$ 4.3.

Table 2. Distribution of BMI categories by smoking status

BMI Category	Smokers N (%)	Nonsmokers N (%)
Mild thinness	184 (93.88%)	12 (6.12%)
Moderate thinness	52 (96.3%)	2 (3.7%)
Normal weight	52 (55.92%)	41 (44.08%)
Overweight	20 (51.28%)	19 (48.72%)
Class I obesity	9 (50%)	9 (50%)

Chi-squared test $p < 0.001$, indicating strong association between smoking and lower BMI values.

Table 3. Distribution of BMI categories by gender

BMI Category	Male N (%)	Female N (%)
Mild thinness	172 (87.75%)	24 (12.25%)
Moderate thinness	51 (94.44%)	3 (5.55%)
Normal weight	49 (52.7%)	44 (47.3%)
Overweight	19 (48.7%)	20 (51.3%)
Class I obesity	9 (50%)	9 (50%)

Chi-squared test $p < 0.001$.

Table 4. Mean BMI by histological subtype of lung cancer

Cancer type	Mean BMI $\pm$ SD
Adenocarcinoma	20.73 $\pm$ 4.29
Large cell carcinoma	22.74 $\pm$ 5.20
Small cell lung cancer	19.12 $\pm$ 3.56
Squamous cell lung cancer	19.5 $\pm$ 4.56

One-way ANOVA, $p < 0.0001$; Holm-Bonferroni post hoc test: significant pairwise differences for adenocarcinoma vs. SCLC ($p = 0.008$), large cell carcinoma vs. SCLC ($p = 0.00032$), and large cell carcinoma vs. squamous cell lung cancer ($p = 0.00187$).

Table 5. Mean BMI by lung cancer stage

Cancer stage	Mean BMI $\pm$ SD
2	23.07 $\pm$ 4.35
3	19.56 $\pm$ 3.96
4	17.44 $\pm$ 1.46

One-way ANOVA showed $p < 0.0001$; all pairwise differences are statistically significant.

Table 6. Multivariable linear regression analysis predicting BMI

Variable	β	95% CI	p-value
Smoking (smoker vs. nonsmoker)	-3.50	-5.45 to -1.55	<0.001
Male gender (male vs. female)	-0.07	-1.88 to 1.74	0.94
Advanced stage (III-IV vs. I-II)	-2.95	-3.87 to -2.03	<0.001
SCLC histology (vs. NSCLC)	-1.34	-2.11 to -0.57	0.001
Age (years)	+0.05	0.01 to 0.08	0.016

$R^2=0.309$; Adjusted $R^2=0.299$.

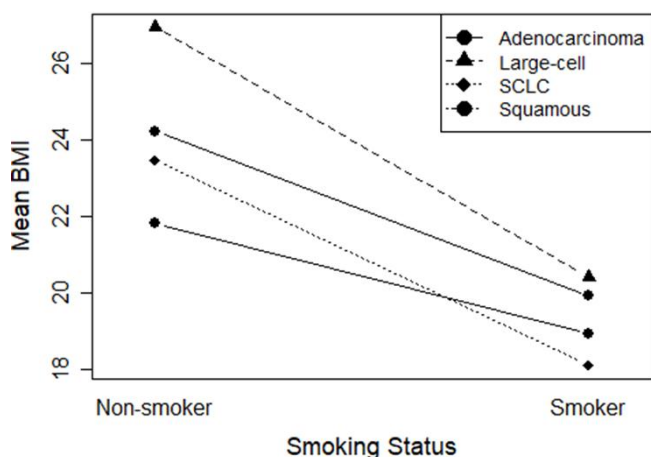


Figure 1. Interaction between smoking status and lung cancer histology depending on mean BMI.

Statistical analysis

Continuous variables were expressed as mean $\pm$ standard deviation (SD), and categorical variables as frequencies and percentages. Independent t-test was employed to compare mean BMI values by gender and smoking groups. Chi-squared test was used for BMI categories. One-way analysis of variance (ANOVA) with Holm-Bonferroni correction was applied when comparing BMI by cancer type and stage. Two-way ANOVA tested the independent and combined effects of smoking and cancer type on BMI. Multivariate linear regression was employed to identify independent predictors of BMI after adjusting for age and gender. Patients with Stage I disease ($n=2$) were excluded from the ANOVA analysis due to insufficient sample size, which precludes reliable variance estimation or meaningful statistical comparisons. A p-value less than 0.05 was considered statistically significant. All analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA).

Results

Demographics and baseline characteristics

The demographic, clinical, and lifestyle characteristics of the 400 lung cancer patients included in this study are summarized in [Table 1](#).

BMI and smoking

The distribution of BMI categories among smokers and nonsmokers is presented in [Table 2](#), highlighting the higher prevalence of underweight and thinness among smokers vs. nonsmokers.

BMI and gender

[Table 3](#) shows the distribution of BMI categories among men and women with lung cancer, demonstrating that underweight and thinness were more common among men, while women were more likely to fall into the normal and elevated BMI categories.

Cancer type and BMI

[Table 4](#) presents the mean BMI and standard deviation for each histological subtype of lung cancer, indicating that patients with large cell carcinoma had the highest mean BMI, while patients with SCLC had the lowest mean BMI.

The distribution of BMI categories varied by cancer type, with thinness being highest in SCLC and squamous cell lung cancer.

Cancer stage and BMI

Patients with Stage I lung cancer were not included in the comparative analysis because only two patients were classified as such, thus making statistical comparisons impractical. [Table 5](#) shows the mean BMI and standard deviation by lung cancer stage, demonstrating a progressive decrease in BMI with increasing disease stage.

BMI decreased significantly with increasing disease stage, consistent with cancer cachexia.

Multivariate linear regression analysis revealed that smoking, advanced disease stage (III-IV), and histological type of SCLC were independently associated with lower BMI. Increasing age was associated with a moderate but statistically significant increase in BMI, while gender was not an independent predictor after adjustment ([Table 6](#)).

Interaction between smoking and cancer type

The [Figure 1](#) below illustrates the mean BMI for different histological subtypes of lung cancer depending on smoking status. A two-way ANOVA revealed that both smoking ($p<0.0001$) and cancer type ($p<0.001$) independently affected BMI. The interaction (Smoking $\times$ Cancer type) was not statistically significant ($p=0.116$), implying that the effect of smoking on BMI was similar across all histological subtypes.

Discussion

Our study revealed a strong correlation between lower BMI and the following clinical factors: smoking, male gender, more aggressive histological subtypes such as small cell lung cancer, and advanced-stage disease [16]. These findings further complement the existing publications and provide new clinical insights that can be applied in both the prognostic assessment and clinical management of patients with lung cancer [19]. Several reports indicate that BMI is lower in smokers than in nonsmokers, emphasizing the association of lifestyle factors with nutritional status in cancer patients [19]. This is also supported by the findings of Jin et al. (2023), who showed that male gender, underweight, and pretreatment weight loss negatively impact the efficacy of immune checkpoint inhibitors in patients with NSCLC [20]. It is also well known that smoking can decrease appetite, increase basal metabolic rate, and induce systemic inflammation, which may be associated with weight loss. Furthermore, as noted by Tseng et al. (2022), weight loss was observed before lung cancer diagnosis, especially at later stages, indicating clinical differences between

smokers and nonsmokers with SCLC. These observations suggest that monitoring BMI in high-risk groups, particularly among smokers, may serve as an early warning of disease progression or the presence of aggressive pathology [21].

In our study, we observed a lower body mass index (BMI) in men than in women, which is consistent with previous studies [22]. This difference may be due to several biological and hormonal factors. For example, Georgakopoulou et al. (2024) examined the role of gender, smoking, and ethnicity in the association between BMI and overall survival in NSCLC and how they influence the prognostic role of BMI in cancer outcomes. Furthermore, biological differences between women and men may also result in variations in fat distribution and hormone levels, which may influence metabolic rate and, consequently, BMI [23]. These findings highlight the importance of considering gender-based differences when interpreting BMI as a prognostic marker and planning individualized treatment for patients [24].

The association between lower BMI and aggressive histological subtypes, such as SCLC and squamous cell lung cancer, may be explained by various biological and metabolic mechanisms rather than tumor aggressiveness alone. SCLC is often associated with pronounced systemic inflammation and paraneoplastic neurological syndromes that contribute to increased energy expenditure and metabolic dysregulation. Elevated levels of proinflammatory cytokines, including interleukin 6 (IL-6) and tumor necrosis factor alpha (TNF- α), are implicated in the pathogenesis of cancer cachexia, promoting muscle proteolysis, lipolysis, and anorexia [24].

Similarly, squamous cell lung cancer is associated with a higher inflammatory burden and chronic tissue damage caused by smoking, which may exacerbate systemic catabolic states. These processes lead to hypermetabolism, loss of skeletal muscle mass, and adipose tissue depletion, ultimately manifesting as a decrease in BMI. Collectively, these data suggest that tumor-specific inflammatory and paraneoplastic pathways play a critical role in shaping the nutritional status of patients with lung cancer and may partially explain the pronounced decrease in BMI observed in these histological subtypes [25, 26].

In our study, a continuous decrease in BMI was observed with disease progression, reflecting cachexia and generalized inflammation. This is consistent with the findings of Kwon et al. (2023), who analyzed the prognostic value of BMI in SCLC and whether skeletal muscle has prognostic significance, highlighting how body composition influences cancer outcomes [27]. These data suggest that weight loss and muscle atrophy are not only markers of disease severity but may also actively contribute to poor outcomes, supporting the potential benefit of early nutritional support [28]. Outcomes may be improved through targeted interventions that address weight loss and muscle atrophy in later stages of cancer, potentially improving treatment tolerability and overall survival [29].

Although our cohort predominantly had a low BMI (particularly among smokers, patients with advanced-stage disease, and aggressive histological subtypes), the concept of the obesity paradox highlights the complex and nonlinear relationship between obesity and cancer outcomes. While several studies suggested a survival advantage for overweight or obese patients with certain lung cancer subtypes or treatment modalities, these findings do not contradict our data but rather highlight the heterogeneity of the impact of body composition on cancer. In this

context, low BMI in our population likely reflects active catabolic processes and cancer cachexia, whereas the obesity paradox may be due to preserved metabolic reserves, treatment-related factors, and patient selection. These observations highlight the need for individualized nutritional assessment that goes beyond BMI alone [10, 30].

This study accentuates the potential value of BMI as a clinical marker in lung cancer. Although low BMI is associated with worse outcomes, the relationship between BMI and cancer prognosis is multifaceted and depends on several factors, including histological subtype, disease stage, and treatment approaches. Incorporating BMI assessment into routine clinical evaluation, along with other relevant factors such as smoking status and tumor biology, may aid in risk stratification and support early interventions [10]. Further well-designed observational and prospective studies are needed to clarify the relationship of BMI with lung cancer prognosis and to develop targeted strategies to improve patient outcomes, including nutritional interventions, pharmacological treatment, and supportive care.

Key findings

- Smoking was strongly associated with lower BMI among lung cancer patients.
- Patients with SCLC and squamous cell lung cancer exhibited the lowest BMI values.
- BMI decreased significantly with cancer stage progression.
- Multivariate analysis confirmed that smoking, advanced stage of cancer, and SCLC histological type were independent predictors of lower BMI.

Conclusion

This study demonstrates that lower BMI in lung cancer patients is significantly associated with smoking, advanced-stage cancer, and aggressive histological subtypes, particularly SCLC. Multivariate analysis confirmed that these factors are independent predictors of lower BMI, highlighting the influence of tumor biology and lifestyle factors on nutritional status. These results emphasize the importance of early assessment of nutritional status and targeted supportive therapy, particularly in high-risk patient groups. Further prospective studies are needed to examine the prognostic and therapeutic implications of BMI changes in lung cancer.

Clinical Implications

This study demonstrated that BMI is a simple, inexpensive, and rapid clinical marker for identifying lung cancer patients who are more likely to have an aggressive disease course or a worse prognosis. Regular monitoring of weight and BMI during treatment and at diagnosis can help clinicians identify patients at risk for developing cachexia and significant weight loss. Underweight patients should prioritize timely nutritional and supportive therapy to mitigate the effects of malnutrition and systemic inflammation, which can negatively impact treatment tolerance and outcomes. Furthermore, integrating BMI with other clinical variables, such as smoking status and histological subtype, allows for more individualized patient management, including nutritional support, weight gain strategies, pharmacological treatment, and adjuvant therapy.

Limitations of the study

This study has several limitations that should be considered when interpreting the results. The retrospective, cross-sectional design limits the ability to establish causal relationships between clinical factors and BMI. Furthermore, BMI is a crude measure of body composition that does not distinguish between fat and muscle mass, meaning that more detailed body composition measures could provide better prognostic and treatment-related information. The lack of survival or treatment outcome data over time also limits this study, as it precludes an assessment of the BMI impact on overall survival. Finally, selection bias may be present due to the hospital-based cohort, which may reduce the generalizability of the results to broader populations.

Smoking status was recorded as a binary variable (smoker vs. nonsmoker), which may obscure potential dose-response relationships between smoking intensity/duration and BMI. Future studies using quantitative measures of smoking, such as pack-years, may provide a more accurate assessment of the relationship between smoking and nutritional status in patients with lung cancer.

BMI was measured at hospitalization, which may not fully reflect weight changes prior to diagnosis or unintentional weight loss that occurred before lung cancer diagnosis.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

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Ethical approval

All procedures performed in studies involving human participants complied with the ethical standards of the institutional research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

AI use statement

In preparing this manuscript, the authors used AI-enabled tools to support the literature search and improve the linguistic clarity and readability of the text. AI tools were not used to generate scientific content, analyze data, or formulate conclusions. All content was reviewed, edited, and approved by the authors, who take full responsibility for the accuracy, originality, and integrity of the manuscript.

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