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(RESEARCH ARTICLE)

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Associations between obesity, smoking and colorectal pathology in a clinical cohort

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Abstract

Background: Obesity and smoking are known risk factors for colorectal cancer (CRC) and other gastrointestinal pathologies. However, their combined effects in Middle Eastern clinical cohorts remain incompletely understood.

Objective: To determine associations between obesity (BMI) and smoking with various colorectal diseases in patients attending a major tertiary care centre in Jordan.

Methods: A retrospective cohort analysis included 1,207 patients who underwent colonoscopy at King Hussein Medical Centre (January 2021–December 2023). Data extracted included smoking history, BMI, colonoscopy findings, and histopathology. Multivariate logistic regression adjusted for age, sex, and family history.

Results: Among 1,207 patients, 32.4% were current smokers and 39.2% were obese (BMI ≥30 kg/m^2). Overall, 46.3% had clinically significant colorectal pathology. Obesity was associated with adenoma (aOR=1.68, 95% CI: 1.24–2.28) and CRC (aOR=2.15, 95% CI: 1.42–3.25). Current smoking was associated with adenoma (aOR=1.92, 95% CI: 1.40–2.64) and CRC (aOR=2.45, 95% CI: 1.58–3.80). The highest risk was observed in obese current smokers (aOR=3.85, 95% CI: 2.40–6.18 for CRC), with significant synergy beyond additive effects.

Conclusion: Obesity and smoking were independently and synergistically associated with higher rates of colorectal pathology, especially neoplastic lesions. Targeted lifestyle modification programmes and incorporation of these risk factors into screening decisions are warranted.

Keywords: Obesity; Smoking; Colorectal cancer; Adenoma; Colonoscopy; Risk factors; Jordan

1. Introduction

Colorectal cancer (CRC) is a major global health problem, with approximately three million people affected worldwide (Sung et al., 2021). CRC ranks among the top five cancers in both men and women in Jordan according to the Jordan Cancer Registry (Jordan Cancer Registry, 2020). Non-modifiable risk factors include age and genetic predisposition; however, modifiable lifestyle factors such as obesity and smoking have emerged as major contributors to CRC and benign colorectal pathology (Bardou et al., 2013; Botteri et al., 2020).

Obesity (BMI ≥30 kg/m^2) promotes chronic low-grade inflammation, insulin resistance, and altered adipokine secretion, which may facilitate colorectal neoplasia (Renchan et al., 2008). A 5 kg/m^2 increase in BMI is associated with an 18% higher CRC risk in men and 9% in women (Ma et al., 2013). Tobacco smoke contains carcinogens such as polycyclic aromatic hydrocarbons and N-nitrosamines, which damage the colonic mucosa and promote adenoma

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progression (Liang et al., 2009). Current smokers have a 20–60% increased risk of CRC compared with never smokers (Botteri et al., 2020).

Obesity and smoking may synergistically increase colorectal disease risk through overlapping mechanisms involving inflammation, oxidative stress, and insulin resistance (Bardou et al., 2013). However, limited data exist regarding their combined effects in Middle Eastern populations. In Jordan, over 35% of adults are overweight or obese, and smoking prevalence remains high (Jordan Department of Statistics, 2021). Current CRC screening guidelines in Jordan emphasize age and family history, with less attention to lifestyle factors (Al-Jaberi et al., 2019).

This study aimed to examine associations between obesity, smoking, and various colorectal diseases in a large clinical cohort from Jordan.

2. Materials and methods

2.1. Study Design and Setting

A retrospective cohort study was conducted at the Gastroenterology Department of King Hussein Medical Centre, Amman, Jordan. The study was approved by the IRB (No. 51_17/2025, 29 February 2026) and the Educational & Technical Directorate (7 April 2026). Informed consent was waived per retrospective, anonymized design.

2.2. Participants

Included: patients aged ≥18 years who underwent colonoscopy between January 2021 and December 2023 with complete bowel preparation (Boston Bowel Preparation Scale ≥6), histopathological results, documented BMI and smoking status.

Excluded: incomplete colonoscopy, inadequate bowel preparation, history of IBD, FAP, Lynch syndrome, previous colorectal resection, pregnancy, incomplete data.

Final analysis: 1,207 patients.

2.3. Data Collection

Standardized case report form extracted: age, sex, family history of CRC, height/weight (BMI categories: underweight <18.5, normal 18.5–24.9, overweight 25.0–29.9, obese ≥30.0), smoking status (current, former, never; pack-years calculated), colonoscopy findings, histopathological diagnosis (malignant, premalignant, inflammatory, other).

2.4. Outcome Definition

Primary outcome: Clinically significant colorectal pathology (malignant lesions, premalignant lesions [adenomas ≥10 mm, villous histology, high-grade dysplasia, or any serrated lesion], or IBD).

Secondary outcomes: Individual pathology categories.

2.5. Statistical Analysis

SPSS v26. Descriptive statistics: means±SD or frequencies (%). Group comparisons: chi-square or ANOVA. Multivariate logistic regression examined associations between obesity (BMI ≥30 vs. <30) and smoking (current vs. never) with outcomes, adjusting for age, sex, and family history. Interaction assessed by including interaction term. RERI, AP, and synergy index (S) calculated. Significance: $p<0.05$ (two-tailed).

3. Results

3.1. Participant Characteristics (Table 1, Figure 1)

Of 1,650 patients undergoing colonoscopy, 1,207 were included. Mean age 55.8±12.4 years, 54.6% male. Overall, 39.2% (n=473) were obese, 32.4% (n=391) current smokers. Family history of CRC in 8.7%.

Table 1 Baseline Characteristics by Obesity and Smoking Status (N=1,207)

Characteristic	Total (N=1,207)	Obese (n=473)	Non-Obese (n=734)	p-value	Current Smoker (n=391)	Never Smoker (n=604)	p-value
Age (years), Mean ± SD	55.8 ± 12.4	57.2 ± 11.8	54.9 ± 12.7	<0.001	52.4 ± 11.5	57.6 ± 12.6	<0.001
Male, n (%)	659 (54.6)	283 (59.8)	376 (51.2)	0.003	312 (79.8)	199 (32.9)	<0.001
BMI (kg/m^2), Mean ± SD	28.7 ± 5.2	33.8 ± 3.4	25.6 ± 2.8	<0.001	29.2 ± 5.4	28.3 ± 5.0	0.021
Obese (BMI ≥30), n (%)	473 (39.2)	473 (100)	0 (0)	—	162 (41.4)	233 (38.6)	0.045
Pack-years*, Mean ± SD	—	22.4 ± 15.8	18.6 ± 13.2	<0.001	25.3 ± 16.4	—	—
Family history of CRC, n (%)	105 (8.7)	46 (9.7)	59 (8.0)	0.312	28 (7.2)	55 (9.1)	0.368

*Pack-years calculated for current and former smokers only.

Table 2 Adjusted Associations of Obesity with Colorectal Pathology

Pathology Category	Obese (n=473) n (%)	Non-Obese (n=734) n (%)	Adjusted OR* (95% CI)	p-value
Any Significant Pathology	261 (55.2)	298 (40.6)	1.58 (1.23–2.03)	<0.001
Colorectal Cancer	48 (10.1)	34 (4.6)	2.15 (1.42–3.25)	<0.001
Adenoma	158 (33.4)	185 (25.2)	1.68 (1.24–2.28)	0.001
Advanced Adenoma	68 (14.4)	58 (7.9)	1.85 (1.28–2.67)	0.001
Serrated Lesion	22 (4.7)	33 (4.5)	1.12 (0.63–1.99)	0.697
Inflammatory Bowel Disease	18 (3.8)	29 (4.0)	0.88 (0.47–1.64)	0.680
Diverticular Disease	92 (19.5)	128 (17.4)	1.08 (0.79–1.48)	0.618

*Adjusted for age, sex, smoking status, and family history of CRC.

Table 3 Adjusted Associations of Smoking with Colorectal Pathology

Pathology Category	Current Smoker (n=391) n (%)	Never Smoker (n=604) n (%)	Adjusted OR* (95% CI)	p-value
Any Significant Pathology	206 (52.7)	241 (39.9)	1.96 (1.48–2.60)	<0.001
Colorectal Cancer	36 (9.2)	28 (4.6)	2.45 (1.58–3.80)	<0.001
Adenoma	127 (32.5)	148 (24.5)	1.92 (1.40–2.64)	<0.001
Advanced Adenoma	50 (12.8)	48 (7.9)	2.08 (1.35–3.21)	0.001
Serrated Lesion	20 (5.1)	23 (3.8)	1.52 (0.81–2.84)	0.194
Inflammatory Bowel Disease	13 (3.3)	28 (4.6)	0.69 (0.35–1.38)	0.294
Diverticular Disease	66 (16.9)	116 (19.2)	0.82 (0.58–1.16)	0.260

*Adjusted for age, sex, BMI category, and family history of CRC.

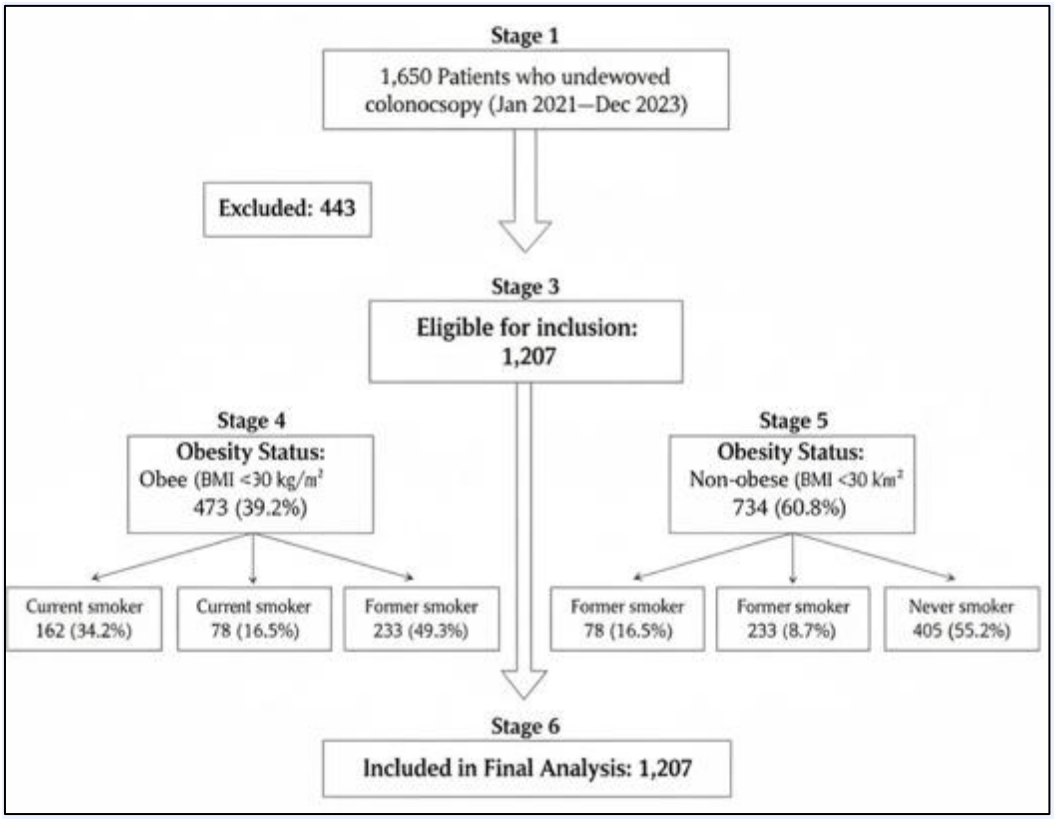
Table 4 Combined Effects of Obesity and Smoking on Colorectal Pathology

Risk Factor Combination	n	Any Significant Pathology aOR* (95% CI)	CRC aOR* (95% CI)	Adenoma aOR* (95% CI)
Normal Weight Never Smoker (Ref)	233	1.00	1.00	1.00
Obese Never Smoker	233	1.62 (1.10–2.38)	2.05 (1.10–3.82)	1.58 (1.02–2.45)
Normal Weight Current Smoker	149	1.84 (1.20–2.82)	2.52 (1.30–4.88)	1.75 (1.09–2.81)
Obese Current Smoker	162	3.12 (2.18–4.46)	3.85 (2.40–6.18)	3.05 (2.02–4.60)

Interaction Metrics (Obese × Current Smoker):

Outcome	RERI (95% CI)	AP (95% CI)	S (95% CI)
Any Significant Pathology	0.89 (0.32–1.46)	0.29 (0.11–0.47)	2.14 (1.32–3.46)
Colorectal Cancer	1.15 (0.41–1.89)	0.30 (0.12–0.48)	2.25 (1.38–3.67)
Adenoma	0.72 (0.18–1.26)	0.24 (0.06–0.42)	1.92 (1.18–3.12)

*Adjusted for age, sex, and family history of CRC.



Abbreviations: BBPS, Boston Bowel Preparation Scale; IBD, Inflammatory Bowel Disease; FAP, Familial Adenomatous Polyposis; BMI, Body Mass Index.; Legend: CONSORT-style flow diagram illustrating the screening, exclusion, and inclusion process of patients undergoing colonoscopy at King Hussein Medical Center (January 2021–December 2023). All included patients had complete BMI data, smoking status documentation, adequate bowel preparation (Boston Bowel Preparation Scale ≥6), and definitive histopathological diagnosis.

Figure 1 Participant Flow Diagram

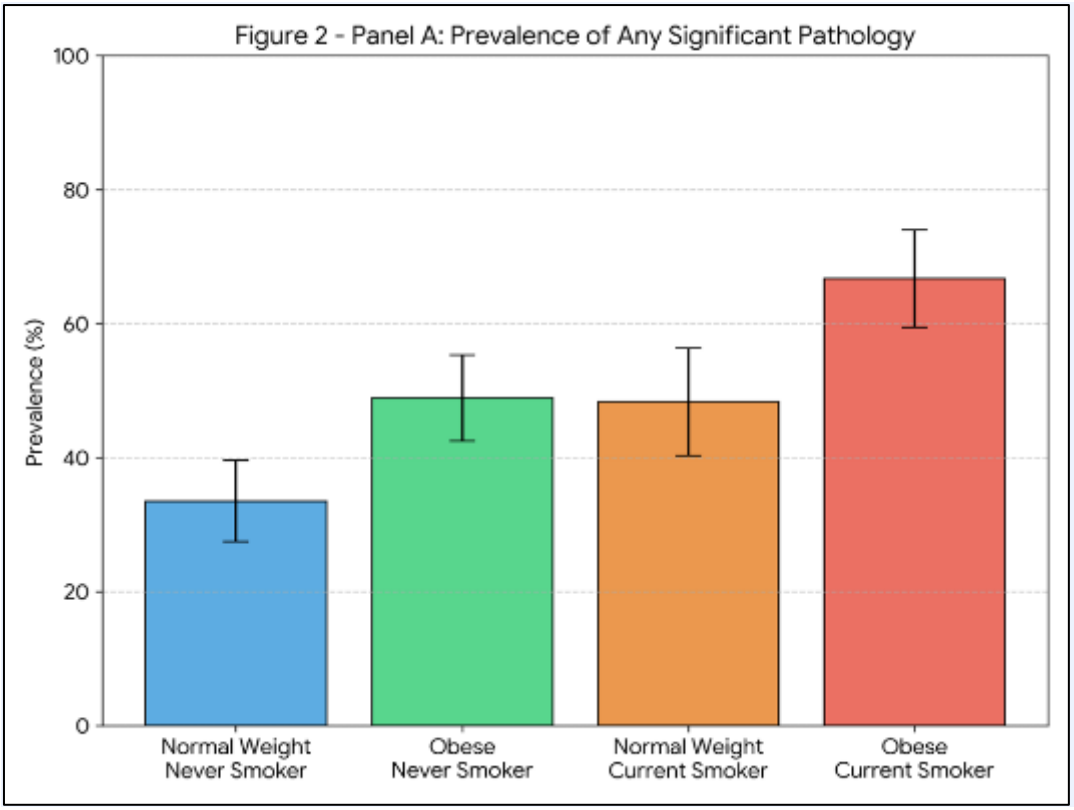


Figure 2 - Panel A: Prevalence of Any Significant Pathology
Prevalence (%)
100
80
60
40
20
0
Normal Weight Never Smoker
Obese Never Smoker
Normal Weight Current Smoker
Obese Current Smoker

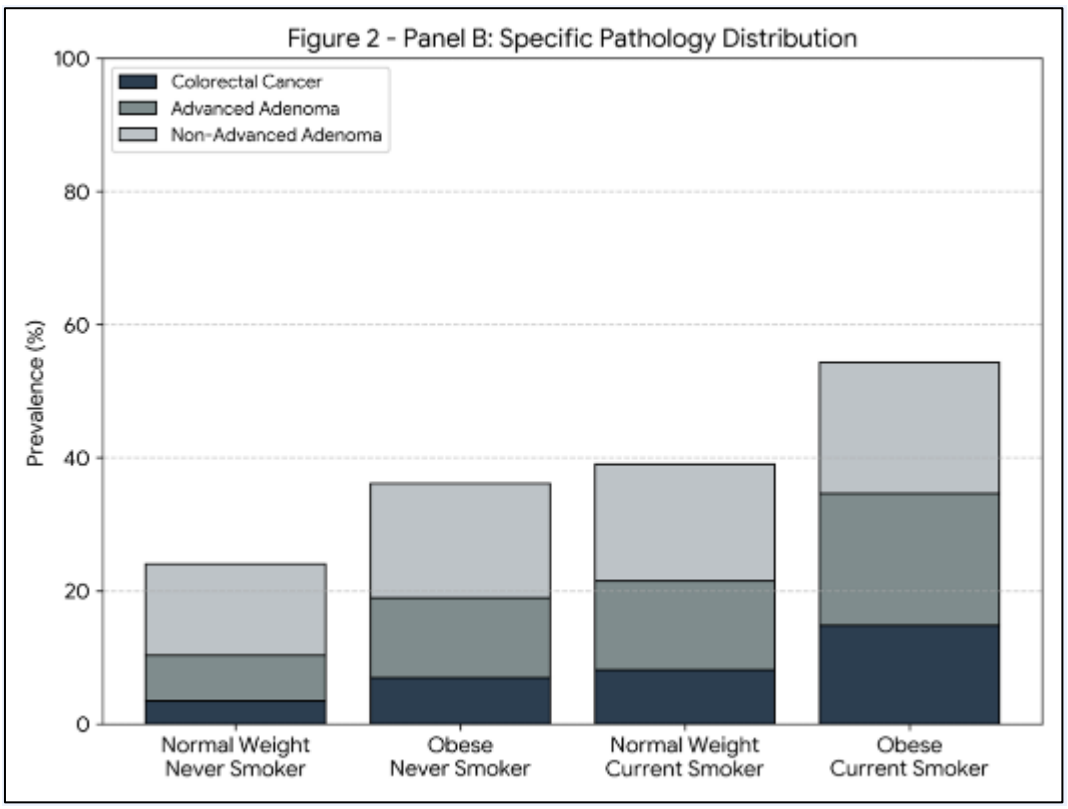
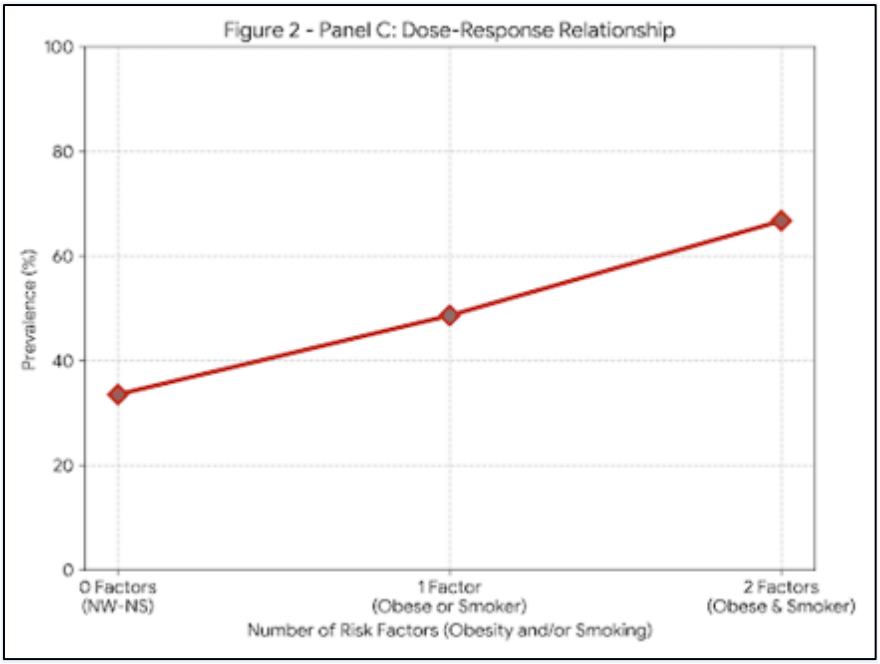


Figure 2 - Panel B: Specific Pathology Distribution
Colorectal Cancer
Advanced Adenoma
Non-Advanced Adenoma
Prevalence (%)
100
80
60
40
20
0
Normal Weight Never Smoker
Obese Never Smoker
Normal Weight Current Smoker
Obese Current Smoker



Abbreviations: NWNS, Normal Weight Never Smoker; ONS, Obese Never Smoker; NWCS, Normal Weight Current Smoker; OCS, Obese Current Smoker. Legend: A) Prevalence of any clinically significant colorectal pathology across combined obesity and smoking status categories. B) Distribution of specific pathology types within each risk category. C) Dose-response relationship showing increasing prevalence with increasing number of risk factors (0 = normal weight never smoker, 1 = either obese or current smoker, 2 = obese current smoker). Error bars represent 95% confidence intervals.

Figure 2 Prevalence of Colorectal Pathology by Combined Obesity and Smoking Status

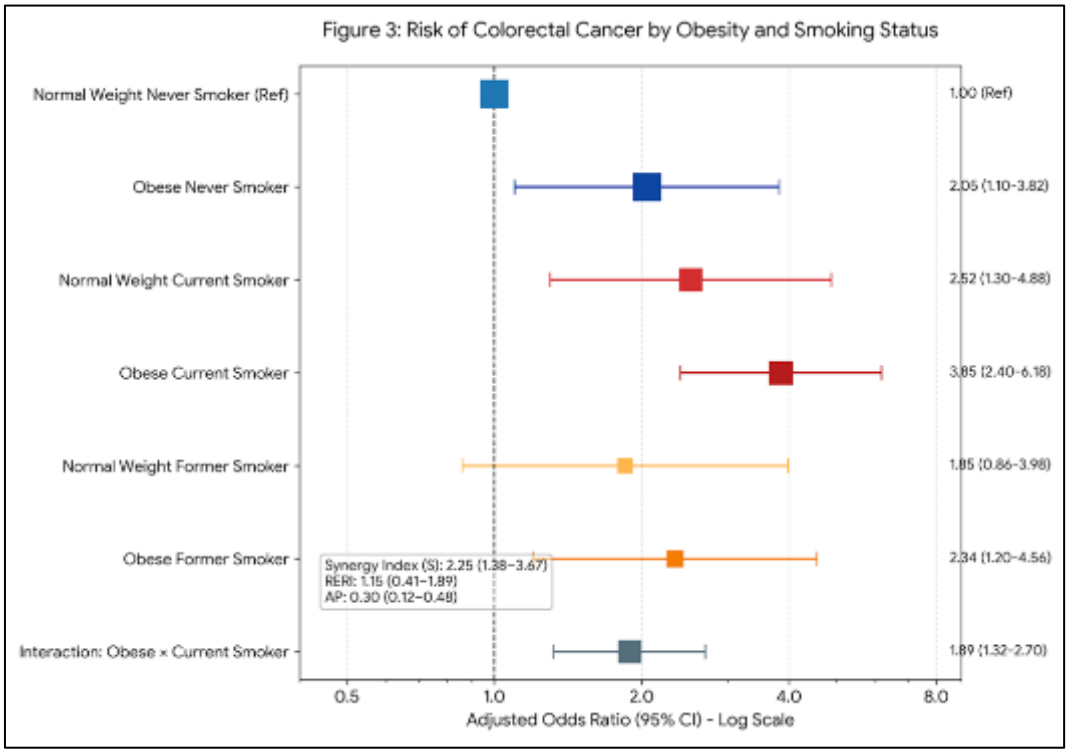


Figure 3 Adjusted Odds Ratios for Colorectal Cancer by Risk Factor Combinations

Legend: Forest plot displaying adjusted odds ratios for colorectal cancer across different combinations of obesity and smoking status, with normal weight never smokers as reference. Models adjusted for age, sex, and family history of colorectal cancer. Square size is proportional to sample size. Horizontal lines represent 95% confidence intervals. The interaction term (obese × current smoker) demonstrates significant synergy beyond additive effects.; *Abbreviations:* OR, Odds Ratio; CI, Confidence Interval; Ref, Reference; RERI, Relative Excess Risk due to Interaction; AP, Attributable Proportion

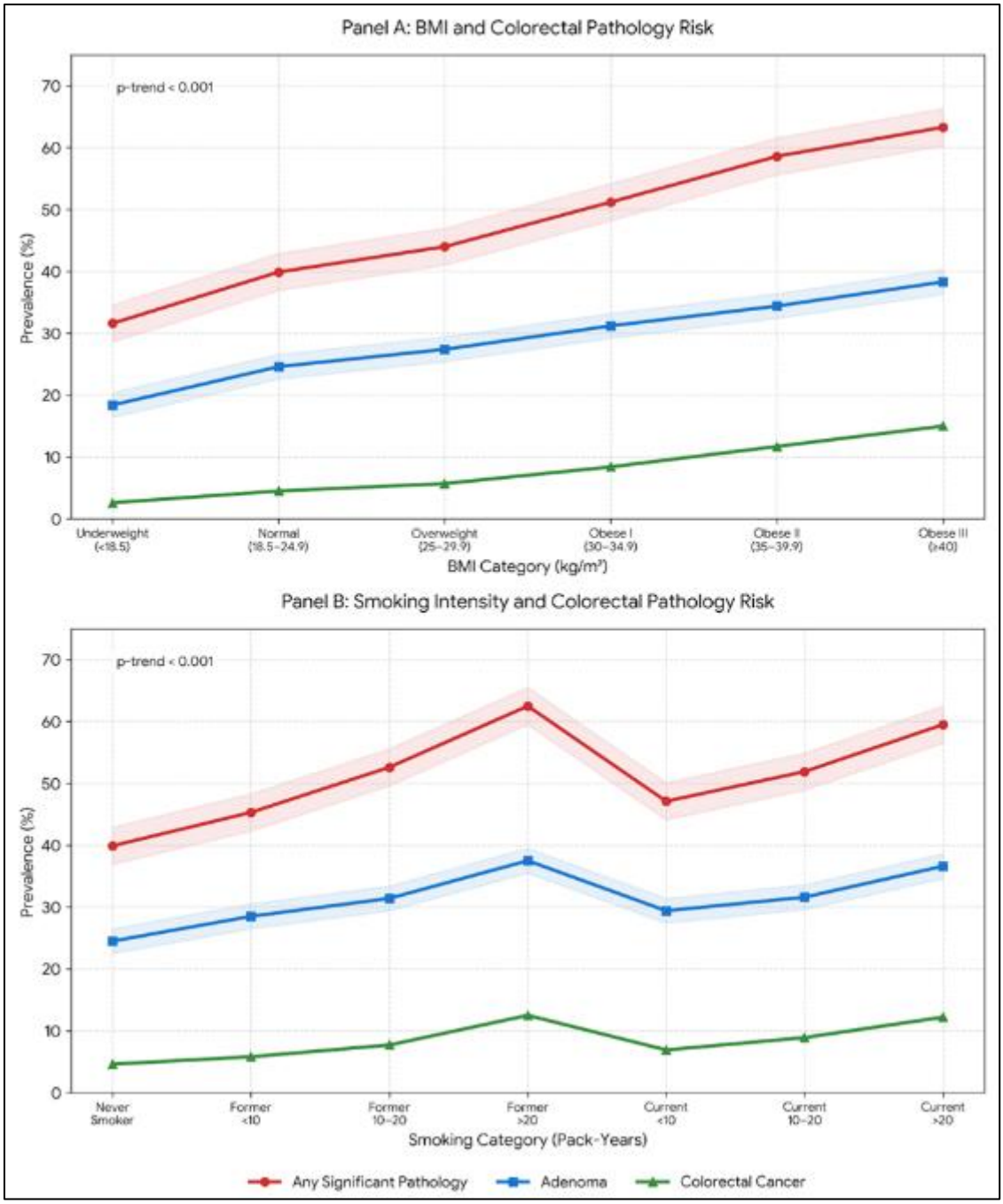


Figure 4 Dose-Response Relationships for Obesity and Smoking with Colorectal Pathology

Legend: A) Dose-response relationship between BMI categories and prevalence of colorectal pathology. B) Dose-response relationship between smoking intensity (pack-years) and prevalence of colorectal pathology. Both panels demonstrate significant positive trends (p for trend <0.001), supporting causal associations. Error bands represent 95% confidence intervals around trend lines.; *Abbreviations:* BMI, Body Mass Index.

3.2. Prevalence of Colorectal Pathology

Clinically significant pathology in 46.3% (n=559): adenomas 28.4% (n=343), hyperplastic polyps 12.1% (n=146), CRC 6.8% (n=82), serrated lesions 4.6% (n=55), IBD 3.9% (n=47), diverticular disease 18.2% (n=220). Multiple pathologies in 8.5%.

3.3. Associations Between Obesity and Colorectal Pathology (Table 2)

Obesity associated with any significant pathology (aOR=1.58, 95% CI: 1.23–2.03), adenomas (aOR=1.68, 95% CI: 1.24–2.28), advanced adenomas (aOR=1.85, 95% CI: 1.28–2.67), and CRC (aOR=2.15, 95% CI: 1.42–3.25). No significant associations with IBD or diverticular disease. Dose-response relationship observed (p for trend <0.001).

3.4. Associations Between Smoking and Colorectal Pathology (Table 3)

Current smoking associated with any significant pathology (aOR=1.96, 95% CI: 1.48–2.60), adenomas (aOR=1.92, 95% CI: 1.40–2.64), advanced adenomas (aOR=2.08, 95% CI: 1.35–3.21), and CRC (aOR=2.45, 95% CI: 1.58–3.80). Former smokers had intermediate odds. Dose-response with pack-years (p for trend <0.001).

3.5. Combined Effects of Obesity and Smoking (Table 4, Figures 2-4)

Synergistic effects observed. Obese current smokers had highest odds:

- Any significant pathology: aOR=3.12 (95% CI: 2.18–4.46)
- Adenomas: aOR=3.05 (95% CI: 2.02–4.60)
- CRC: aOR=3.85 (95% CI: 2.40–6.18)

Interaction metrics for CRC: RERI=1.15 (95% CI: 0.41–1.89), AP=0.30 (95% CI: 0.12–0.48), S=2.25 (95% CI: 1.38–3.67), indicating greater-than-additive effects.

3.6. Subgroup Analyses

Associations consistent across subgroups. Obesity-CRC association stronger in men (aOR=2.68, 95% CI: 1.65–4.35) than women (aOR=1.62, 95% CI: 0.89–2.95). Smoking associations similar across age and sex.

4. Discussion

In this large clinical cohort of 1,207 Jordanian patients undergoing colonoscopy, obesity and smoking were each independently and synergistically associated with colorectal pathology, particularly adenomas and CRC. Current smokers had approximately twice the odds of clinically significant pathology compared with never smokers, and obese individuals had similarly elevated risks. Obese current smokers had a nearly fourfold increased odds of CRC, with significant synergy beyond additive effects.

The independent associations between obesity and colorectal neoplasia align with meta-analyses reporting an 18% increase in CRC risk per 5 kg/m^2 increase in BMI for men (Ma et al., 2013). Proposed mechanisms include insulin resistance, elevated IGF-1, chronic inflammation, and altered gut microbiota (Renchan et al., 2008; Bardou et al., 2013). Stronger associations in men may reflect higher visceral adiposity (Pischon et al., 2006).

The link between smoking and colorectal pathology is consistent with literature reporting 20–60% increased CRC risk in current smokers (Botteri et al., 2020; Liang et al., 2009). Tobacco smoke induces DNA adducts, oxidative damage, and impaired DNA repair. The dose-response relationship with pack-years reinforces causality.

The synergistic effect of obesity and smoking represents the most novel finding. Patients with both risk factors had substantially higher odds than predicted by adding individual risks. RERI values of 0.89 for any significant pathology and 1.15 for CRC indicate biological interaction. Possible mechanisms include systemic inflammation (elevated CRP, TNF-α, IL-6), oxidative stress, and insulin resistance (Bardou et al., 2013). Smoking may exacerbate metabolic disturbances associated with obesity, while obesity may enhance the bioavailability and activity of tobacco carcinogens.

4.1. Clinical and Public Health Implications

Individuals with obesity and/or smoking history may benefit from earlier or more frequent CRC screening. (2) Lifestyle modification programmes (smoking cessation, weight loss) should be integral to CRC prevention. (3) Region-specific evidence can inform national screening guidelines.

4.2. Limitations

Retrospective design may introduce selection bias. BMI measured at single time point. Smoking status self-reported. Residual confounding by unmeasured factors (physical activity, diet, alcohol) cannot be excluded. Single-centre tertiary care setting may limit generalizability.

4.3. Strengths

Large clinical cohort from Middle Eastern population, rigorous histopathological confirmation, comprehensive confounder adjustment, formal assessment of additive interaction.

5. Conclusion

In this Jordanian patient cohort, obesity and smoking were independently and synergistically associated with increased risk of colorectal pathology, including adenomas and CRC. Patients with both risk factors were at particularly high risk. These findings support incorporating obesity and smoking status into CRC risk assessment and screening decisions in Jordan. Priority should be given to smoking cessation and weight loss programmes as part of comprehensive CRC prevention strategies.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

The authors declare no conflict of interest.

Statement of ethical approval

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board (IRB) of the Royal Medical Services, Jordan, on **29 February 2026** under registration number **51_17/2025**. Final approval from the Educational and Technical Directorate was obtained on **7 April 2026**.

Statement of informed consent

Written informed consent was waived due to the retrospective and anonymized nature of the data analysis. All patient data were de-identified prior to analysis.

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