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Original Article

Profiling Ultra-Processed Food Consumption Patterns and Their Association with Early-Onset Colorectal Cancer Subtypes

Azhar Rashid[1], Kiran Naved Khan[2], Amina Akbar[3], Nikhal Kumar[4], Abeera Gul Tarar[5], Anam Fatima[6], Bhoomeeka Nanda[7]

[1] Consultant Oncologist, Department of Clinical and Radiation Oncology, Neurospinal and Cancer Care Institute (NCCI), Karachi, Pakistan.
[2] United Medical and Dental College, Karachi, Pakistan.
[3] MPhil, Food Science and Technology, University of Chenab, Gujrat, Pakistan.
[4] MBBS, Liaquat University of Medical and Health Sciences (LUMHS), Jamshoro, Pakistan.
[5] Akhtar Saeed College of Allied Health Sciences, Lahore, Pakistan.
[6] PhD Chemistry, COMSATS University Islamabad (Lahore Campus), Lahore, Pakistan.
[7] Liaquat Institute of Medical & Health Sciences, Thatta, Pakistan.

✉ **Correspondence:** Kiran Naved Khan, Kirannavedkhan31@gmail.com

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ABSTRACT

Background: Associations between ultra-processed food (UPF) consumption and clinicopathological characteristics of early-onset colorectal cancer remain incompletely understood. Objective: To examine associations between UPF energy contribution and tumor stage, anatomical subsite, and histological differentiation among younger adults with colorectal cancer. Methods: This hospital-based cross-sectional study included 84 patients aged 18–49 years at diagnosis with histologically confirmed colorectal adenocarcinoma, recruited from tertiary oncology centers in Punjab, Pakistan, between November 2025 and February 2026. A semi-quantitative food frequency questionnaire and NOVA classification estimated UPF energy contribution. Clinical characteristics were extracted from medical records. Analyses included chi-square tests, crude odds ratios, and multinomial logistic regression adjusted for body mass index, smoking, and total energy intake. Results: Mean age was 38.6 ± 6.2 years. Advanced-stage disease occurred in 78.6% of patients consuming ≥35% of energy from UPFs versus 47.6% consuming <35% (crude OR: 4.03; 95% CI: 1.55–10.47; p = 0.003). Anatomical distribution differed between groups (p = 0.021). The reported adjusted association with poorly differentiated histology was greatest for intake ≥45% versus <25% (aOR: 4.12; 95% CI: 1.84–9.22). Conclusion: Higher UPF intake was associated with advanced-stage presentation and differences in tumor characteristics within this patient population. These associations do not establish causality or cancer risk. Keywords: Ultra-processed foods; Early-onset colorectal cancer; Cross-sectional studies; Tumor stage; Histological differentiation.

INTRODUCTION

Colorectal cancer is a major contributor to cancer incidence and mortality worldwide (1). Increasing diagnoses among adults younger than 50 years, commonly termed early-onset colorectal cancer (eoCRC), have raised

questions about exposures that may influence disease development and presentation in younger populations. Advanced stage at diagnosis and differences in anatomical distribution and pathological characteristics make this population an important focus of investigation, although these features should not be interpreted as evidence that all early-onset tumors are intrinsically more aggressive (2). Changes in diet, adiposity, physical activity, and other environmental exposures have been proposed as contributors to early-onset cancer trends, but their individual and combined roles remain incompletely understood (3).

Ultra-processed foods (UPFs) represent a potentially relevant dietary exposure. Within the NOVA classification system, these products are industrial formulations typically containing food-derived substances and additives that are uncommon in domestic food preparation. Examples include many packaged snacks, sugar-sweetened beverages, reconstituted meat products, and ready-to-eat meals; classification depends on their ingredients and processing characteristics rather than convenience alone (4). Their contribution to total dietary energy provides a measurable indicator of exposure, while differences in product composition mean that UPFs should not be treated as a nutritionally uniform category. This distinction is important when interpreting associations between overall UPF intake and colorectal disease.

Prospective evidence provides a rationale for investigating UPF consumption in relation to colorectal cancer. In three US cohorts, Wang and colleagues reported an association between higher UPF consumption and colorectal cancer risk among men, with findings varying by sex and anatomical subsite (5). These observations support further investigation but do not establish that UPF intake causes cancer or determines tumor aggressiveness. Moreover, associations with incident colorectal cancer in broader adult populations do not directly answer whether dietary exposure differs across stage, histological grade, or anatomical subsite among patients with eoCRC. Proposed relationships involving diet, the intestinal microbiome, and inflammatory processes offer biological hypotheses, but they require direct investigation rather than inference from dietary associations alone (2,3).

The specific question addressed here is whether reported UPF consumption is associated with clinicopathological variation among younger adults who already have colorectal cancer. Cancer occurrence and tumor characteristics at diagnosis are distinct outcomes, and evidence concerning the former cannot automatically explain the latter. A hospital-based assessment in Punjab, Pakistan, can provide setting-specific information about UPF energy contribution and its relationship with stage, differentiation, and tumor location. Interpretation must nevertheless account for the possibility that symptoms, diagnosis, or treatment influence dietary reporting and that referral patterns and other patient characteristics affect the observed associations.

Accordingly, this cross-sectional study aimed to characterize reported UPF consumption, expressed as a percentage of total daily energy intake, among patients aged 18–49 years with histologically confirmed primary colorectal adenocarcinoma recruited from tertiary oncology centers in Punjab. It compared UPF energy contribution between early-stage (I/II) and advanced-stage (III/IV) disease and across proximal colon, distal colon, and rectal tumors, and evaluated the association between UPF intake categories and histological differentiation, accounting for body mass index, smoking status, and total energy intake in the histology analysis. The objective was to examine associations within the recruited patient population rather than estimate cancer incidence or establish causal effects.

MATERIAL AND METHODS

This observational cross-sectional study examined associations between ultra-processed food (UPF) consumption and clinicopathological characteristics among adults with early-onset colorectal cancer. The study was conducted at tertiary oncology centers in the industrial and urban core of Punjab, Pakistan, between November 2025 and February 2026. The setting was selected to investigate dietary exposure among younger patients receiving oncology care in an urban and industrial context. Comparisons were made within the colorectal cancer population according to disease stage, histological differentiation, and anatomical tumor subsite.

Patients were eligible if they were aged 18–49 years at primary diagnosis, had histologically confirmed primary colorectal adenocarcinoma, and had medical records documenting tumor subsite and stage. Exclusion criteria were a known hereditary colorectal cancer syndrome, including Lynch syndrome or familial adenomatous polyposis, pre-existing inflammatory bowel disease, or incomplete dietary records. Of 95 individuals screened during the study period, 84 completed the dietary and clinical assessments and constituted the final analytical sample.

Dietary intake was assessed using a semi-quantitative food frequency questionnaire adapted to include commercially available and traditional processed foods consumed in urban Pakistan. Reported food items were classified according to the NOVA framework, which distinguishes foods by the extent and purpose of processing (4). UPF exposure was expressed as the percentage of total daily energy intake contributed by foods classified within NOVA group 4. This percentage was calculated by dividing estimated daily energy intake from UPFs by estimated total daily energy intake and multiplying by 100.

UPF energy contribution was analyzed as a continuous variable for comparisons by disease stage and anatomical subsite. For descriptive comparisons, participants were categorized as having lower UPF intake, defined as less than 35% of total energy, or higher UPF intake, defined as 35% or more; each group included 42 participants. For regression analysis, intake was grouped into four categories: less than 25%, 25% to less than 35%, 35% to less than 45%, and 45% or more of total daily energy. The lowest intake category served as the exposure reference group.

Clinical information was extracted from institutional electronic health records and pathology reports. Tumor stage was classified using the TNM staging system and grouped as early-stage disease, comprising stages I and II, or advanced-stage disease, comprising stages III and IV. Histological differentiation was categorized as well, moderately, or poorly differentiated. Anatomical tumor subsite was categorized as proximal colon, distal colon, or rectum. Demographic and potential confounding variables included age, sex, body mass index, smoking status, and total daily energy intake. Body mass index was expressed in kilograms per square meter, and smoking status was categorized as never smoking or current/former smoking.

Analyses were performed using IBM SPSS Statistics version 28.0. Continuous variables were summarized using means and standard deviations, and categorical variables were summarized using frequencies and percentages. The Shapiro–Wilk test was used to assess the distribution of continuous data. An independent-samples t-test compared mean UPF energy contribution between patients with early-stage and advanced-stage disease. One-way analysis of variance compared mean UPF energy contribution across proximal colon, distal colon, and rectal tumors, followed by Tukey post-hoc comparisons to evaluate differences between anatomical groups.

Multinomial logistic regression was used to examine the association between UPF intake categories and histological differentiation, with poorly differentiated histology as the outcome contrast of interest. Models included body mass index, smoking status, and total daily energy intake as adjustment variables. Associations were summarized using adjusted odds ratios and 95% confidence intervals. Statistical significance was defined as a p-value below 0.05. The reported analyses were based on the 84 participants who completed the dietary and clinical assessments.

RESULTS

Of 95 individuals screened, 84 completed the dietary and clinical assessments, corresponding to a completion proportion of 88.4%. Participants had a mean age of 38.6 ± 6.2 years, and 44 (52.4%) were male. The groups consuming <35% and ≥35% of total energy from ultra-processed foods (UPFs) each included 42 participants. Mean BMI was 24.9 ± 3.1 kg/m^2 in the <35% group and 27.9 ± 4.0 kg/m^2 in the ≥35% group. Current or former smoking was reported by 10 (23.8%) and 16 (38.1%) participants, respectively (Table 1).

Advanced-stage disease was present in 53 participants (63.1%). Its frequency differed between UPF intake groups, occurring in 20 of 42 participants (47.6%) consuming <35% of energy from UPFs and 33 of 42 (78.6%) consuming ≥35% (p = 0.003; Table 2). The corresponding crude odds ratio for stage III/IV versus stage I/II disease was 4.03 (95% CI: 1.55–10.47; Table 3).

Table 1. Demographic characteristics of participants by ultra-processed food intake

Characteristic	Total (N = 84)	UPF <35% of energy (n = 42)	UPF ≥35% of energy (n = 42)
Age, years, mean ± SD	38.6 ± 6.2	39.1 ± 5.9	38.1 ± 6.5
Male sex, n (%)	44 (52.4)	23 (54.8)	21 (50.0)
Female sex, n (%)	40 (47.6)	19 (45.2)	21 (50.0)
BMI, kg/m², mean ± SD	26.4 ± 3.8	24.9 ± 3.1	27.9 ± 4.0
Never smoking, n (%)	58 (69.0)	32 (76.2)	26 (61.9)
Current/former smoking, n (%)	26 (31.0)	10 (23.8)	16 (38.1)

Abbreviations: BMI, body mass index; SD, standard deviation; UPF, ultra-processed food. Percentages use the respective column denominators.

Table 2. Tumor stage and anatomical subsite by ultra-processed food intake

Characteristic	Total (N = 84), n (%)	UPF <35% of energy (n = 42), n (%)	UPF ≥35% of energy (n = 42), n (%)	p-value
Stage I/II	31 (36.9)	22 (52.4)	9 (21.4)	0.003
Stage III/IV	53 (63.1)	20 (47.6)	33 (78.6)	—
Proximal colon	22 (26.2)	15 (35.7)	7 (16.7)	0.021
Distal colon	28 (33.3)	16 (38.1)	12 (28.6)	—
Rectum	34 (40.5)	11 (26.2)	23 (54.8)	—

Abbreviation: UPF, ultra-processed food. Percentages use the respective column denominators. P-values are from two-sided Pearson chi-square tests without continuity correction. The p-value beside stage I/II applies to the overall stage comparison; the p-value beside proximal colon applies to the overall anatomical-subsite comparison.

Table 3. Unadjusted association between ultra-processed food intake and advanced-stage disease

UPF intake, % of total energy	Participants, n	Stage III/IV, n (%)	Crude OR	95% CI
<35	42	20 (47.6)	1.00	—
≥35	42	33 (78.6)	4.03	1.55–10.47

Abbreviations: CI, confidence interval; OR, odds ratio; UPF, ultra-processed food. The exposure reference category is UPF intake <35% of total energy; the outcome comparison is stage III/IV versus stage I/II. The OR is unadjusted, and its CI was calculated using the log-odds Wald method. Percentages use the respective row denominators.

Table 4. Reported adjusted associations between ultra-processed food intake categories and poorly differentiated tumor histology

UPF intake, % of total energy	Adjusted OR	95% CI	p-value
<25	1.00	—	—
25 to <35	1.45	0.62–3.38	0.392
35 to <45	2.68	1.14–6.30	0.024
≥45	4.12	1.84–9.22	0.001

Abbreviations: CI, confidence interval; OR, odds ratio; UPF, ultra-processed food. The exposure reference category is UPF intake <25% of total energy. Estimates were reported from multinomial logistic regression adjusted for body mass index, smoking status, and total daily energy intake.

Anatomical tumor distribution also differed between UPF intake groups (p = 0.021). Rectal tumors accounted for 11 cases (26.2%) in the <35% group and 23 (54.8%) in the ≥35% group, whereas proximal colon tumors accounted for 15 (35.7%) and 7 (16.7%), respectively. Distal colon tumors occurred in 16 (38.1%) and 12 (28.6%) participants, respectively (Table 2).

In the adjusted histology analysis, the odds ratios associated with UPF energy contributions of 25% to <35%, 35% to <45%, and ≥45%, relative to <25%, were 1.45 (95% CI: 0.62–3.38; p = 0.392), 2.68 (95% CI: 1.14–6.30; p = 0.024), and 4.12 (95% CI: 1.84–9.22; p = 0.001), respectively. The confidence interval for the 25% to <35% category included unity, while the highest intake category had the largest reported adjusted estimate for poorly differentiated histology (Table 4).

DISCUSSION

In this hospital-based cross-sectional study of adults with early-onset colorectal cancer, higher reported ultra-processed food (UPF) energy contribution was associated with a greater proportion of advanced-stage disease and differences in anatomical tumor distribution. Rectal tumors constituted a larger proportion of cases in the higher-intake group. The reported adjusted histology estimates also suggested an association between greater UPF consumption and poorly differentiated tumors. These findings address variation in presentation among patients already diagnosed with colorectal cancer; they do not establish whether UPF consumption increases cancer incidence or accelerates disease progression.

The findings are broadly compatible with the hypothesis that dietary exposures may contribute to colorectal cancer heterogeneity, although direct comparison with prospective incidence studies requires caution. Wang and colleagues reported an association between higher UPF consumption and colorectal cancer incidence among men in three US cohorts, with variation by sex and anatomical subsite (5). Their study evaluated subsequent cancer

occurrence, whereas the present analysis compared characteristics within an existing cancer population. Consequently, agreement in the general direction of association does not establish that the same processes explain cancer development and stage or grade at presentation. Differences in population characteristics, dietary composition, exposure assessment, and study design further limit direct comparison.

The anatomical findings likewise require distinction between tumor distribution and subsite-specific cancer risk. The larger proportion of rectal tumors in the higher-UPF group indicates a difference in case composition, but it cannot establish an increased population risk of rectal cancer. Reviews of early-onset colorectal cancer describe variation in anatomical presentation and emphasize that environmental exposures, inherited susceptibility, and diagnostic circumstances may contribute to observed clinical patterns (2). The present results therefore support examining tumor location separately in dietary research, while providing insufficient evidence to identify a rectum-specific causal pathway.

Several biological explanations are plausible but were not investigated directly. UPFs comprise diverse industrial formulations, and NOVA classification identifies processing characteristics rather than a single nutrient or chemical exposure (4). An association with total UPF energy contribution could therefore reflect multiple aspects of diet, including the foods displaced by these products, their nutritional composition, and correlated eating behaviors. Changes in the intestinal microbiome, metabolic regulation, and inflammatory processes have been proposed as possible links between environmental exposures and early-onset carcinogenesis (2,3). However, this study measured neither individual additives nor microbial, inflammatory, or molecular markers. Its findings cannot distinguish among these explanations or attribute the observed associations to specific ingredients.

The association with advanced-stage disease also has potential nonbiological explanations. Stage at diagnosis reflects the extent of disease at detection and does not directly measure the rate of tumor progression. Dietary habits may be associated with socioeconomic circumstances, access to care, symptom recognition, or diagnostic delay, none of which was adequately characterized here. Moreover, the stage estimate was unadjusted. The higher mean BMI and greater proportion of current or former smokers in the higher-UPF group demonstrate that exposure groups differed in other characteristics. Although the histology analysis included BMI, smoking status, and total energy intake, these adjustments cannot establish independence from all relevant confounding influences.

The reported histology estimates warrant particularly cautious interpretation. Larger estimates in successive intake categories are compatible with an exposure gradient, but they do not establish a dose–response relationship without an appropriate trend analysis. The confidence intervals also indicate uncertainty about association magnitude. Furthermore, the separate analyses of histological differentiation and anatomical location do not demonstrate a specific association with poorly differentiated rectal cancer. Combining these outcomes in the interpretation would imply a joint analysis that was not performed.

The study's strengths include its focus on younger patients, histological confirmation of colorectal adenocarcinoma, and extraction of clinical characteristics from health records and pathology reports. Use of a locally adapted dietary questionnaire allowed assessment of foods relevant to the recruitment setting, while the NOVA framework provided a defined basis for classifying UPF exposure (4). Examining stage, grade, and anatomical location separately also preserved clinically meaningful distinctions that would be obscured by treating all colorectal cancers as a single outcome.

Important limitations constrain interpretation. The cross-sectional design does not establish temporal ordering between dietary exposure and disease characteristics. Symptoms, diagnosis, or treatment could have influenced intake or recall, and questionnaire-based estimates are susceptible to reporting and classification error. Recruitment from tertiary oncology centers may favor particular clinical presentations and limits generalizability beyond the participating patient population. Restricting analysis to cancer cases may also introduce selection bias if dietary exposure and other determinants both influence entry into the study. Exclusion of known hereditary syndromes does not rule out unrecognized inherited susceptibility. The sample size limits detailed subgroup analysis and raises concerns about sparse categories and model stability, particularly for adjusted histological comparisons. Residual confounding and the loss of information associated with categorizing continuous dietary exposure provide additional reasons for caution.

Clinically, these findings identify dietary exposure as a potential correlate of presentation rather than a validated prognostic or screening marker. They do not demonstrate that changing UPF intake alters tumor behavior, treatment response, or survival, and they provide no basis for adopting the study's intake thresholds as clinical decision rules. Broader research on early-onset cancer supports evaluating exposures across the life course and distinguishing etiological hypotheses from established preventive strategies (3). Prospective studies with dietary assessment before diagnosis, clearly defined clinical outcomes, and adequate control of confounding would help determine whether the associations observed here persist under stronger temporal and analytical conditions.

CONCLUSION

Among the recruited adults with early-onset colorectal cancer, higher reported UPF energy contribution was associated with advanced-stage presentation and differences in anatomical tumor distribution, including a greater proportion of rectal tumors. The reported histology analysis also suggested an association with poor differentiation. These findings identify possible relationships between dietary exposure and clinicopathological presentation within this patient population, without establishing causality, cancer risk, or a basis for clinical screening decisions.

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