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ORIGINAL CONTRIBUTION

Association of late eating with colorectal adenomas: a cross-sectional study

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Abstract

Purpose Colorectal cancer (CRC) is linked to lifestyle exposures. However, changes in the CRC rates among younger populations remain poorly understood and suggest the existence of yet unidentified factor(s) that may contribute to colon carcinogenesis. Here, we investigated the potential role of time of eating in the risk of pre-cancerous colonic neoplasms (tubular adenoma: TA).

Methods We enrolled 663 participants undergoing screening colonoscopies. Data on food timing, dietary intake, sleep/wake patterns, and chronotype were collected through structured questionnaires. Late eating was defined as the consumption of food or snack within a 3-hour window of sleep onset for at least four days a week. Pathology reports confirmed the histology of colonic polyps, and adenomas were further classified into risk categories.

Results A total of 644 patients met criteria for our study. There were 270 (42.2%) participants classified as late eaters. Compared to non-late eaters, the odds of TA were higher in late eaters (OR = 1.46, 95% CI = 1.05–2.03, $p = 0.023$), an association which was strengthened after adjusting for multiple confounders (OR 1.98, 95% CI 1.19–3.28, $p = 0.008$). Late eating remained an independent risk factor for high-risk as well as multiple TAs.

Conclusion This study proposes late eating as a risk factor for colon tubular adenomas and underscores the potential role of less studied forms of circadian disruption imposed by time of eating in the development of colon neoplastic formation.

Keywords Food timing · Colon polyp · Lifestyle · Circadian

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Abbreviations

BMI	Body Mass Index
CI	Confidence Interval
CRC	Colorectal Cancer
OR	Odds Ratio
SD	Standard Deviation

Introduction

Colorectal cancer (CRC) is the third leading cause of cancer-associated deaths in the United States, with projections indicating over 50,000 deaths in 2023[1]. A multitude of lifestyle factors, such as poor diet quality/"Western" diet, excessive alcohol consumption, smoking, and lack of physical activity, are linked to an increased risk of colorectal cancer and precancerous lesions (polyps) [2]. However, emerging risk factors, especially those that are increasingly common in younger generations, remain underexplored.

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One of the hallmarks of our modern society is the disruption of the circadian rhythm. Circadian rhythm is defined as the ~24-hour sleep/wake cycle that is regulated by light/dark cues on the central clock in the brain [3, 4]. In addition to these centrally controlled oscillators, peripheral clocks are located within the organ systems to regulate physiologic function based on input from the central pacemaker [5]. Of interest, circadian oscillators of the intestine are mainly modulated by the timing of food consumption [5, 6]. While the shift in the light/dark cycle, the classical mode of circadian disruption, is shown to increase the risk of colorectal cancer, as endorsed by the International Agency for Research on Cancer (IARC), the role of other behaviors that could affect our circadian rhythm has not been studied [7]. One such behavior is consuming meals close to bedtime during our biological rest time, which is becoming excessively more common in our society [8]. Previous work by our group and others has suggested that feeding mice at rest could desynchronize the central and peripheral clocks, leading to circadian desynchrony [9]. However, whether such circadian desynchrony from such "abnormal" food timing could contribute to the development of colonic neoplasms has yet to be investigated.

To address these gaps in knowledge, we conducted a cross-sectional study to address the question of whether time of eating could affect the development of colonic adenomas.

Materials and methods

Study design and data collection

We recruited 663 participants who presented for screening colonoscopies between 2018 and 2022 at Rush University Medical Center. Of the 663 recruited patients, 644 met the criteria for our study. Exclusions encompassed individuals with a history of inflammatory bowel disease, Lynch syndrome, familial adenomatous polyposis, active malignancies, or incomplete data (Fig. 1). Participants completed questionnaires of demographics, a Food Timing Screener (FTS), and the Munich Chronotype Questionnaire (MCTQ). Demographics and clinical data were additionally extracted from electronic medical records. The Institutional Review Board at Rush University Medical Center approved the study under IRB number ORA: 14112503, and informed written consent was obtained from all participants.

Food timing screener

We employed the Food Timing Screener (FTS), which is a dietary assessment questionnaire that was previously developed and validated by our group [10], to collect information on frequency and timing of food consumption and sleep timing during a typical week. Participants provided their responses to the questionnaire at the time of recruitment in the endoscopy unit, with the questions administered by a trained research coordinator. Participants reported the time of eating meals and snacks, as well as the times of waking and falling asleep. Sleep-wake times were included in order to analyze the relationships between food timing and sleeping behaviors. To account for differences between food types, major dietary components were collected (e.g., wheat, vegetables, fruit, fried food, pickled food, and red meat) and were categorized as regular (consumption of the above foods as more than three times per week), and less frequent/occasional. A total of 639 patients completed the FTS and were included in the analysis. Late eating was defined as consuming food, whether a meal or a snack, within a 3-hour window before sleep onset for at least four days a week, as reported by the study participant [11, 12].

The munich chronotype questionnaire (MCTQ)

Patient chronotype was assessed using the Munich Chronotype Questionnaire (MCTQ), a validated and commonly used questionnaire to assess circadian rhythm [13]. This questionnaire includes questions on typical sleep behavior on workdays and free days. From this self-reported data, patient chronotypes were divided into early, intermediate, and late groups based on their calculated mid-sleep on free days (MSF). Early chronotype was defined as MSF before 3:00 am, intermediate if MSF was between 3:00 am and 5:00 am, and late chronotype if MSF fell beyond 5:00 am. Additionally, the MCTQ allows the calculation of the midpoint of sleep on free days based on the reported sleep and wake times.

Adenoma detection and risk-stratification

Pathology reports were reviewed to confirm the histology of the colonic polyps (i.e., precancerous lesions including tubular adenomas (TA) and sessile serrated adenomas (SSA) vs. benign hyperplastic polyps). Polyps were further classified into low- and high-risk based on their size, histology, and numbers (Supplementary Tables 1 and 2) [14]. Participants with normal colonoscopies were characterized as those who had hyperplastic polyps smaller than 10 mm, no adenomas (TA or SSA), and no CRC. Participants were defined as having low-risk TA if they had 1–2 TAs with low-grade dysplasia, each less than 10 mm in size. High-risk TA included all advanced adenomas, including adenomas greater than 10 mm, tubulovillous histology, high-grade dysplasia, or greater than 3 adenomas. In the context of serrated adenomas (SA), participants with low to moderate risk

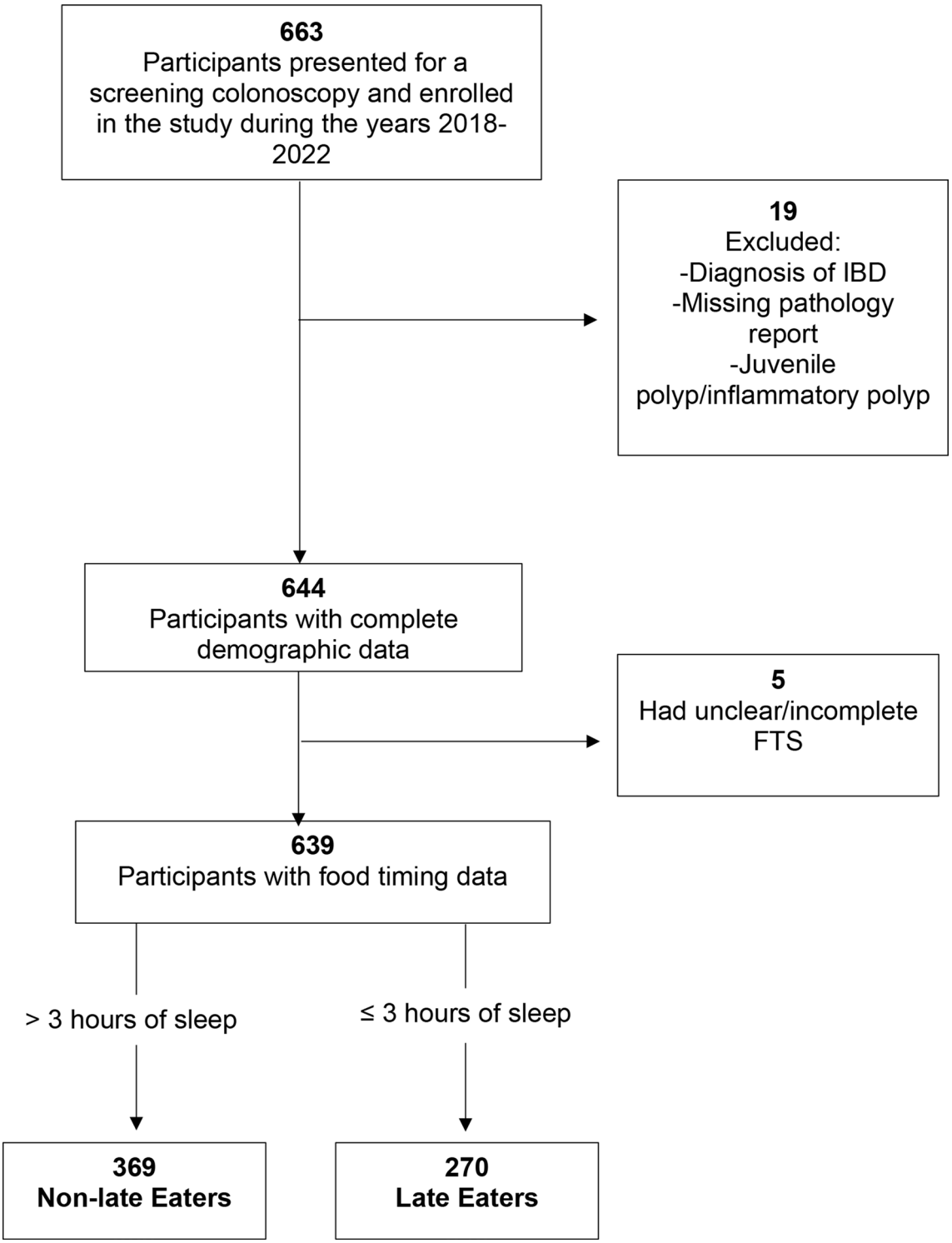

Fig. 1 Flow chart of study population

exhibited 1–4 sessile serrated polyps smaller than 10 mm in size, as well as hyperplastic polyps larger than 10 mm. High-risk SA included those with SSAs larger than 10 mm, individuals with more than 5 SSAs of any size, and those with serrated sessile polyps (SSP) displaying dysplasia or traditional serrated adenoma. Analyses were conducted comparing normal colonoscopies to a combination of low- and high-risk TA. Additionally, we separately examined the high-risk TA group.

Study outcomes and statistical analysis

The aim of this study was to examine whether late eating predisposed participants to developing colonic adenomas. Categorical variables were presented in terms of counts and percentages, while continuous variables were described with means and standard deviations. The appropriate statistical tests were employed for the analysis: the $\chi 2$ test for categorical variables and the t-test for continuous variables. We conducted a multivariable logistic regression analysis to explore the relationship between late eating and adenoma risk, adjusting for BMI, sex, race, age (<60 and ≥ 60), comorbidities (diabetes mellites type 2, coronary artery disease, hypertension, and hyperlipidemia), current smoking status, alcohol use, diet intake, eating frequency (≤ 3, >3 meal/snacks), fasting period in hours, family history of colorectal cancer, and previous history of adenoma. Age was made binary due to the significant association of age groups (<60, ≥ 60) with late eating. Statistical significance was defined as a P-value below 0.05. All data analysis was performed with IBM SPSS Statistics Version 26.

Ethics statements

Written, informed consent was obtained from all participants. We approached patients who met inclusion criteria and did not have any of the exclusion criteria and were undergoing a colon exam by colonoscopy as clinically indicated and determined by the referring primary care or gastroenterologist for cancer screening purposes. A trained research staff approached the patients on the day of the clinically planned colonoscopy to explain the study, go through the consent form, and answer any questions they may have. An informed consent was obtained before any research-related study activity was done. Participants were given the opportunity to discuss what their study entails with their family and friends and decide whether they would like to participate or not. All participants were being told that they would have no alteration to any care at their institutions if they decided not to participate, and those who were uncertain were not included in the study.

Results

A total of 644 patients met the criteria for our study and were included in the final analysis (Supplementary Table 3). Participants had a mean (SD) age of 57.9 (10.4) and a BMI of 29.2 (6.4). At least one adenomatous or serrated polyp was found in 273 (42.4%) of all patients (Supplementary Table 1). Polyp detection rates were comparable among endoscopists.

Table 1 illustrates the characteristics of study participants, categorized into two groups: non-late eaters and late eaters. Among the 639 participants included in the study, 369 (57.7%) were classified as non-late eaters, while 270 (42.2%) were identified as late eaters. Notably, late eating was more prevalent in individuals under the age of 60, with 161 participants (59.6%), compared to 109 participants (40.4%) in the over-60 age category ($p=0.06$). There was no association between late eating and sex, race, BMI, chronic disease, smoking, alcohol use, or family history of CRC. The diet distribution between late and non-late eaters was comparable (Supplementary Table 4).

Late eating was associated with the presence of adenomas (Table 2). Compared to non-late eaters (124, 35.5%), late eaters had a higher TA rate (116, 44.6%) (OR 1.46, 95% CI 1.05–2.03, $p=0.02$). After adjusting our analysis for potential confounders, late eating remained significantly associated with TA (OR 1.98, 95% CI 1.19–3.28, $p=0.008$), indicating an independent association of late eating on the adenoma occurrence. There were no significant differences between the groups when analyzing metachronous adenomas.

Late eating had a significant association with high-risk TA (adjusted OR 2.07, 95% CI 1.05–4.06, $p=0.035$). Considering participants with multiple adenomas, we observed a 2.72-fold higher likelihood of having two or more adenomas (≥ 2) in late eaters (adjusted OR 2.72, 95% CI 1.36–5.41, $p=0.005$), an effect which increased in those with more adenomas (≥ 3 TAs, adjusted OR 4.10, 95% CI 1.66–10.15, $p=0.002$) (Table 2).

Discussion

Colorectal cancer (CRC) remains a significant public health concern in industrial countries [15]. Lifestyle factors, including "Western" dietary habits, alcohol consumption, and tobacco usage, have been widely recognized as contributing to an elevated risk of colon cancer and precancerous polyps [16, 17]. In lieu of a dramatic decline in exposure to some lifestyle factors, such as tobacco, in several industrial countries, the burgeoning incidence of CRC, especially among younger populations, implies the presence of yet unidentified lifestyle factor(s) that may contribute to colon carcinogenesis [18]. While significant research has explored the impact of diet type [19], there remains a gap in the potential role of diet timing in colon carcinogenesis.

To the best of our knowledge, there is no previous evidence on the role of the time of eating, specifically consuming larger meals closer to the physiologic rest time, in the risk of adenoma development. This clinical observation expands on our experimental studies, where eating during

Table 1 Characteristics of participants and adenoma risk according to food-timing status

	Late Eating		
	No ($n=369$)	Yes ($n=270$)	*P* value
No. of participants, n (%)	369 (57.7)	270 (42.2)	
Age, mean (SD), y	58.4 (10.4)	57.1 (10.4)	0.12
Age, binary, n (%)			
<60	193 (52.3)	161 (59.6)	
≥60	176 (47.7)	109 (40.4)	
OR (95% CI)	1 [Ref]	1.35 (0.98–1.85)	0.039*
Sex, n (%)			
Male	155 (42.0)	125 (46.3)	
Female	214 (58.0)	145 (53.7)	
OR (95% CI)	1[Ref]	1.19 (0.87–1.63)	0.16
Race, n (%)			0.80
White	177 (48.6)	138 (51.3)	
Black	138 (37.9)	96 (35.7)	
Other[a]	49 (13.5)	35 (13.0)	
BMI, mean (SD), kg/m^2	29.3 (6.2)	29.1 (6.3)	0.69
Comorbidities,[b]n (%)			
Yes	206 (55.8)	140 (51.9)	
No	163 (44.2)	130 (48.1)	
OR (95% CI)	1 [Ref]	0.85 (0.62–1.17)	0.18
Current smoker, n (%)			
Yes	35 (9.5)	25 (9.3)	
No	334 (90.5)	244 (90.7)	
OR (95% CI)	1 [Ref]	0.98 (0.57–1.68)	0.52
Alcohol use, n (%)			
Daily	69 (18.7)	56 (20.7)	
None or weekly	300 (81.3)	214 (79.3)	
OR (95% CI)	1 [Ref]	1.14 (0.77–1.69)	0.29
Chronotype			<0.001**
Early	123 (38.0)	135 (57.4)	
Intermediate	165 (50.9)	86 (36.6)	
Late	36 (11.1)	14 (6.0)	
Family history of CRC, n (%)			0.12
First degree relative with CRC	32 (8.7)	32 (11.9)	
Second degree relative < 60 with CRC	20 (5.4)	12 (4.4)	
Second degree relative ≥ 60 with CRC	51 (13.8)	23 (8.5)	
No family history	266 (72.1)	203 (75.2)	
Previous history of adenoma			
No. of cases (%)	75 (21.1)	51 (19.4)	
OR (95%CI)	1 [Ref]	1.02 (0.67–1.55)	0.92

Table 2 Adenoma risk and late eating

	Late Eating		
	No ($n=369$)	Yes ($n=270$)	*P* value
All Tubular Adenoma			
No. of cases (%)	124 (35.5)	116 (44.6)	
Crude OR (95% CI)	1 [Ref]	1.46 (1.05–2.03)	0.023*
Adjusted OR (95%CI)[a]	1 [Ref]	1.98 (1.19–3.28)	0.008*
Low-risk Tubular Adenomas			
No. of cases (%)	73 (24.5)	65 (31.1)	
Adjusted OR (95%CI)[a]	1 [Ref]	1.78 (0.95–3.34)	0.074
High-risk Tubular Adenomas			
No. of cases (%)	51 (14.6)	51 (19.6)	
Adjusted OR (95%CI)[a]	1 [Ref]	2.07 (1.05–4.06)	0.035*
Three or more Tubular Adenomas			
No. of cases (%)	28 (10.2)	30 (16.1)	
Adjusted OR (95%CI)[a]	1 [Ref]	4.10 (1.66–10.15)	0.002**

Abbreviations: CI, confidence interval; OR, odds ratio

[a] - Adjusted for BMI, sex, race, age (<60 and ≥60), comorbidities, current smoking status, alcohol use, diet intake, family history of colorectal cancer, fasting on work and free days, eating frequency, and previous history of adenoma

the physiologic rest disrupted the circadian rhythm and promoted colon carcinogenesis in prone animals [9, 20]. The habit of late eating is particularly common in younger individuals, among whom CRC rates have been increasing [21, 22]. However, the potential association of late eating and adenoma in young adults could not be tested in our study since our participants were eligible for age-appropriate CRC screening and hence were not young. Nevertheless, we did observe a predilection among younger individuals (below 60) in our cohort for late eating habits compared to those older than 60. Indeed, adjustment for several established risk factors for CRC including age has made the association of late eating with the adenoma risk even stronger. Moreover, focusing on the closer surrogates of CRC, such as high-risk (based on pathologic features and size) and multiple TAs, revealed a significant effect of late eating, which further suggests an impact of late eating on colon carcinogenesis.

Overall, these results support a link between time of eating and the risk of adenoma development. Whether the diet type interacts with the diet time cannot be addressed here. However, we observed a comparable diet distribution between late and non-late eaters in our series, implying that the effect of eating time could be independent from the actual types of foods consumed. Potential interactions between time and type of diet could not be tested here due

to the sample size. Larger series can further help establish the role of time of eating on less common outcomes such as sessile serrated polyps or according to the polyp locations.

While the existing cohorts did not implement any tool to specifically address the potential role of time of eating on the risk of colon cancer or polyps, recent evidence suggests, albeit indirectly, that such an association may exist [23, 24]. To answer this question directly, we would need the administration of systematic questionnaires to capture meal timing patterns in future cohorts.

Potential mechanisms that can link the time of eating to adenoma formation call for further research. Our experiments have shown that changes in mealtime (eating during rest phase) induced central-intestinal circadian desynchrony in mice and promoted gut microbial alterations ("dysbiosis") and intestinal carcinogenesis in polyposis mice that were exposed to another CRC risk factor such as alcohol [9, 20]. Dysbiosis from an "abnormal" time of eating has been characterized by an increase in proinflammatory bacteria and a reduction of protective bacteria that stabilize the gut barrier, such as short chain fatty acid (SCFAs) producing bacteria [20]. Indeed, alterations in the gut microbiota composition and reductions of SCFA producing bacteria preceded exaggerated colon carcinogenesis in polyposis mice [9, 20]. Targeting microbiota via increasing SCFAs producing bacteria partially reversed mucosal inflammation and barrier dysfunction and resulted in amelioration of polyposis. However, the extent to which these mechanisms are at play in humans needs to be investigated.

We are aware of the limitations of our studies. While we utilized the validated food questionnaire to estimate the eating behaviors of the participants, patterns of food consumption were subject to recall bias. The cross-sectional design limits our ability to establish causality between meal timing and the risk of adenoma development. While we found an association, we could not conclude that late eating causes the development of colon polyps based on our observations. Therefore, the observed associations should be interpreted with caution, and future case-control and prospective cohort studies are needed. However, the relatively large sample size for our primary outcome (tubular adenoma) are notable advantages of our cohort, which is evident by our ability to verify several previously established risk factors for colon adenomas (e.g., age, males, higher BMI, history of cigarette smoking, and alcohol drinking) (Supplementary Table 5). We are aware that dietary assessment tools, such as Food Frequency Questionnaires (FFQs) and the Automated Self-Administered 24-hour (ASA24) recall, are superior methods for accurate dietary profiling and should be considered for future studies. Although the FTS has been validated against ASA24® recalls [10], we did not administer ASA24® in the current study due to the significant time required, which would have interfered with clinical operations and subject recruitment. Consequently, we were not able to account for food calorie counts or total energy intake in our model. However, by employing a focused dietary profiling approach, we were able to screen for patients' dietary habits regarding certain foods without jeopardizing the clinical workflow, study uptake, and the primary study outcome. Of note, the overall dietary profile of our participants falls within the ranges reported in the US population. The proportion of participants who ate fruits and vegetables regularly is consistent with the rates reported from the analysis of the National Health and Nutrition Examination Survey (NHANES), which used standard FFQs. In NHANES, two-thirds or more of US adults are estimated to consume fruits or vegetables regularly [25].On the other hand, regular red meat consumption was reported by only a quarter of our study participants, which is also consistent with recent analysis of NHANES data indicating a disproportionate beef diet consumption in the US population. This analysis suggests that half of the consumed beef goes to a small portion (approximately 12%) of the population [26]. This emphasizes the relevance of our study population in illustrating these broader patterns of adenoma development.

In conclusion, our data reveals late eating as an independent risk factor for colonic adenomas. These findings could aid in developing a comprehensive risk assessment to mitigate the burden of colorectal cancer and call for further studies to elucidate the mechanisms linking eating patterns to the development of colorectal neoplasms.

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Author contributions DA: participant enrollment, administering structured questionnaires, data curation, data analysis, investigation, visualization; ERK: data curation, data analysis, investigation, visualization, and writing; MA: participant recruitment, administering structured questionnaires, data curation, visualization; JQT: participant recruitment, administering structured questionnaires, data curation; YC: investigation, and writing; FB: conceptualization, funding acquisition, investigation, project administration, supervision, validation, visualization, and writing. All authors revised and approved the final version of the manuscript.

Data availability The data supporting the findings of this study are available upon request from the corresponding author.

Declarations

Conflicts of interest The authors declare no potential conflicts of interest.

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