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The Relationship between the Dietary Inflammatory Index and Colorectal Cancer

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ABSTRACT

Objective: Colorectal cancer is the third most common type of cancer worldwide and one of the leading causes of cancer-related deaths. Chronic inflammation is known to be associated with colorectal cancer. This study aimed to evaluate the relationship between the Dietary Inflammatory Index (DII) and colorectal cancer.

Materials and Methods: 149 patients over the age of 18 who underwent surgery with the diagnosis of colorectal cancer and 120 control patients in the same age group who were hospitalized for a non-cancer reason were included in the study. DII scores were calculated from the patients' 3-day 24-h food consumption records. The level of relationship between DII and colorectal cancer was analyzed with regression analysis.

Results: A total of 269 patients were included in the study, 149 in the study group and 120 in the control group. The mean age of the study group was 64.45±11.36, and that of the control group was 65.90±10.36, and the difference was not significant (p=0.280). A significant association was found between high DII score and colorectal cancer (odds ratio [OR]: 2.62, p<0.001). When adjusted for age and gender, high DII score was also found to be a risk factor for colorectal cancer (OR: 2.72, p<0.001).

Conclusion: There is a significant association between an inflammatory diet and high DII scores, which are a measure of it, and the development of colorectal cancer. High DII scores are a significant risk factor for colorectal cancer regardless of age and gender. This risk does not vary according to the location of the cancer and is similar for colon and rectum cancers.

Keywords: Colorectal cancer, Cytokines, Dietary habits, Nutrition

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INTRODUCTION

Colorectal cancer is the third most common type of cancer globally and a leading cause of cancer-related mortality.[1] Incidence and mortality rates of colorectal cancer are higher in developed countries, whereas they are lower in less developed regions such as Asia, Africa, and most of Latin America.[2]

Inflammation typically occurs as a natural response of the body to tissue injury or damage.[3] An acute inflammatory response plays a crucial role in the healing and regeneration process, usually leading to recovery within a few days.[4] Chronic inflammation, however, represents a persistent condition where tissue destruction and repair occur simultaneously due to the sustained presence of pro-inflammatory cytokines,

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often resulting from increased blood flow to the injured area mediated by mast cell-derived histamine.[5] Elevated levels of these cytokines are also believed to be associated with colorectal cancer.[6] Chronic inflammation is widely recognized to be associated with epithelial cancers, particularly colorectal cancer, which remains the most studied.[7,8]

Several studies have indicated a direct link between specific dietary components and inflammation.[9,10] There is growing evidence that these dietary elements influence both inflammation and colorectal cancer risk.[9,11]

The 2012 Continuous Update Project by the American Institute for Cancer Research/World Cancer Research Fund reported that consumption of pro-inflammatory foods such as red and processed meats is associated with an increased risk of colorectal cancer. Conversely, consumption of anti-inflammatory dietary fiber has an inverse relationship with colorectal cancer risk.[12] Additional dietary components known for their anti-inflammatory properties, such as tea and coffee, have also demonstrated various health benefits, including lower cancer incidence and mortality.[13,14] Comprehensive studies on dietary patterns have shown that unhealthy diets are linked to higher risks of colorectal cancer and adenomas, whereas healthy diets are associated with reduced risks.[15]

Research on the role of diet in inflammation and colorectal cancer suggests that dietary patterns represent a complex array of exposures involving cumulative and frequent interactions that affect inflammatory responses and outcomes. Although several dietary indices exist to evaluate diet quality, most lack the capacity to assess the inflammatory potential of a diet. In 2009, researchers at the University of South Carolina developed the first Dietary Inflammatory Index (DII), based on literature published until 2007, as a tool to summarize the inflammatory impact of dietary intake.[16] This index was later revised and updated in 2014.[17] The DII categorizes individuals' diets on a continuum from maximally pro-inflammatory to maximally anti-inflammatory. Higher DII scores indicate more pro-inflammatory diets, while lower scores reflect more anti-inflammatory dietary patterns.[17]

The aim of this study was to evaluate the relationship between DII scores and colorectal cancer.

MATERIALS AND METHODS

This study was conducted prospectively as a single-center study at the University of Health Sciences, Dışkapı Yıldırım Beyazıt Health Practice and Research Center, following the approval of the local ethics committee. Informed consent was obtained from all participating volunteers. This study was conducted in accordance with the Declaration of Helsinki.

The study included 149 patients (study group) diagnosed histopathologically with colorectal cancer and who underwent surgery at the General Surgery Department of the Dışkapı Yıldırım Beyazıt Training and Research Hospital between July 2020 and November 2021, as well as 120 patients (control group) who were followed and treated for non-cancer diagnoses during the same period. Patients included were over 18 years of age, volunteered to participate, and had complete data available. All patients in the study group had a histopathological diagnosis of colorectal cancer.

Exclusion criteria included: those unwilling to participate, individuals with inflammatory bowel disease, those with a first-degree family history of colorectal cancer, patients with genetic syndromes associated with colorectal cancer, those diagnosed with other cancers or metastases, individuals with alcohol or substance abuse, patients receiving hormone therapy or medical nutrition therapy for other conditions, individuals with incomplete data, and those with communication impairments rendering them unable to complete the questionnaire.

Data Collection Tools

Demographic data, such as age, gender, family history of cancer, smoking status, weight loss, physical activity levels, and history of rectal bleeding were collected. Biochemical parameters, including carcinoembryonic antigen (CEA), cancer antigen (CA) 19-9, and hemoglobin levels, as well as the frequency of consumption of alcohol, coffee, tea, garlic, turmeric, and saffron, were recorded. For colorectal cancer patients, tumor location and pathological diagnosis were also documented.

Each patient completed a 3-day 24-h dietary recall form (including 2 weekdays and 1 weekend day) for the week before surgery. For 10 additional food items not included in the BeBIS® 8.2 (Nutrition Information System) software but necessary for DII calculation, a food frequency questionnaire was completed according to the Turkish Dietary Guidelines (TÜBER). Physical activity levels were classified according to the World Health Organization guidelines.

DII Calculation

The DII was calculated using data from 24-h dietary recall forms analyzed with BeBIS® 8.2, based on 36 nutritional components (including alcohol, Vitamins B12, B6, cholesterol, energy, total fat, fiber, folic acid, carbohydrates, iron, magnesium, polyunsaturated and monounsaturated fatty acids, beta-carotene, caffeine, niacin, omega-3 and 6, protein, riboflavin, saturated fats, selenium, thiamine, trans fats, Vitamins A, C, D, and E, zinc, and total flavonoids), as well as the consumption frequencies of 10 additional items (ginger, saffron, turmeric, pepper, thyme, rosemary, coffee, tea, onion, and garlic).

Statistical Analysis

Data were analyzed using the Statistical Package for the Social Sciences (SPSS) version 22.0 (SPSS, Chicago, IL, USA). Continuous variables are expressed as mean±standard deviation and median (minimum–maximum), and categorical variables as

number and percentage. The Shapiro–Wilk test was used to evaluate the normality of data distribution. For comparisons, the independent t-test was applied to normally distributed variables, and the Mann–Whitney U test for non-normally distributed variables. Categorical variables were compared using the Chi-square test. The relationship between DII scores and colorectal cancer was assessed using regression analysis. Adjustments were made for age and sex. A $p<0.05$ was considered statistically significant.

RESULTS

A total of 269 patients were included in the study: 149 in the study group and 120 in the control group. The mean age was 64.45±11.36 years in the study group and 65.90±10.36 years in the control group, with no statistically significant difference between the two (p=0.280). Of the patients in the study group, 103 (69.1%) were male and 46 (30.9%) were female. In the control group, 90 (75.0%) were male and 30 (25.0%) were female. Gender distribution was similar between groups (p=0.288).

A family history of cancer was reported in 46 patients (30.9%) in the study group and in 34 patients (28.3%) in the control group (p=0.651). The proportion of smokers was 47.7% in the study group and 46.7% in the control group, with no significant difference (p=0.872). Physical activity levels were also comparable between groups (p=0.259). Weight loss was observed in 63 patients (42.3%) in the study group and 19 patients (15.8%) in the control group, a statistically significant difference (p<0.001). Rectal bleeding was reported in 34 patients (22.8%) in the study group and 10 patients (8.3%) in the control group (p=0.001).

Fecal occult blood test positivity was observed in 13.4% of the study group and 0.8% of the control group. Among patients diagnosed with colorectal cancer, the median CEA level was 3 µg/L (range: 2–7 µg/L), and the median CA 19-9 level was 16 U/mL (range: 10–31 U/mL). Hemoglobin levels were similar in both groups (p=0.097). Table 1 summarizes the demographic characteristics.

Table 1. Demographic characteristics

Variable	Study group (n=149) (%)	Control group (n=120) (%)	p
Age (years)	64.45±11.36	65.90±10.36	0.280*
Gender			0.288**
Male	103 (69.1)	90 (75.0)	
Female	46 (30.9)	30 (25.0)	
Family history of cancer			0.651**
Yes	46 (30.9)	34 (28.3)	
No	103 (69.1)	86 (71.7)	
Smoking			0.872**
Yes	71 (47.7)	56 (46.7)	
No	78 (52.3)	64 (53.3)	
Physical activity			0.259**
Inactive (<150 min/week)	39 (26.2)	38 (31.7)	
Active (150–299 min/week)	72 (48.3)	46 (38.3)	
Very active (>300 min/week)	38 (25.5)	36 (30.0)	
Weight loss			<0.001**
Yes	63 (42.3)	19 (15.8)	
No	86 (57.7)	101 (84.2)	
Rectal bleeding			0.001**
Yes	34 (22.8)	10 (8.3)	
No	115 (77.2)	110 (92.7)	
FOBT positive	20 (13.4)	1 (0.8)	<0.001**
CEA (µg/L)	3 (2–7)	–	–
CA 19-9 (U/mL)	16 (10–31)	–	–
Hemoglobin (g/dL)	13.6 (11.0–14.8)	14.2 (8.7–19.0)	0.097***

*Independent samples t-test, **Chi-square test, ***Mann–Whitney U test. CEA: Carcinoembryonic antigen, CA: Cancer antigen, Hb: Hemoglobin, FOBT: Fecal occult blood test. Values are presented as mean±standard deviation, n (%), and median (min–max).

Tumor Location and Pathology

Among patients with colorectal cancer, tumors were located in the sigmoid colon in 42 patients (28.4%), right colon in 38 (25.7%), left colon in 27 (18.2%), rectum in 16 (5.9%), rectosigmoid junction in 14 (9.5%), and transverse colon in 11 (7.4%). The pathological diagnosis was adenocarcinoma in 132 patients (88.6%) and mucinous adenocarcinoma in 17 patients (11.4%). Table 2 summarizes the tumor location and pathology.

Regression Analysis

Table 3 summarizes regression analysis revealed a significant association between higher DII scores and colorectal cancer (odds ratio [OR]: 2.62, p<0.001). This association remained significant after adjusting for age and sex (adjusted OR: 2.72, p<0.001). When stratified by tumor location:

- High DII scores were significantly associated with colon cancer (OR: 2.37, p<0.001; adjusted OR: 2.51, p<0.001).
- When the colon was subdivided into proximal and distal regions:
  - Proximal colon cancer showed a strong association (OR: 3.07, p<0.001; adjusted OR: 3.15, p<0.001).
  - Distal colon cancer also showed a significant relationship (OR: 2.14, p=0.003; adjusted OR: 2.39, p<0.001).

High DII scores were similarly associated with rectal cancer (OR: 2.52, p=0.003; adjusted OR: 2.59, p=0.003).

Table 2. Tumor location and pathology

Tumor location	**n (%)**
Sigmoid colon	42 (28.4)
Right colon	38 (25.7)
Left colon	27 (18.2)
Rectum	16 (5.9)
Rectosigmoid	14 (9.5)
Transverse colon	11 (7.4)
Tumor pathology	**n (%)**
Adenocarcinoma	132 (88.6)
Mucinous adenocarcinoma	17 (11.4)

DII Scores

The mean DII score in the study group was –3.86±1.04. In the control group, the mean DII score was –4.37±0.35. The distribution of DII scores in each group is shown in Figures 1 and 2, respectively. The average DII score was significantly higher in the study group than in the control group (p=0.008) (Fig. 3).

DISCUSSION

The results of our study demonstrate that higher DII scores are associated with an increased risk of colorectal cancer. Even after adjusting for age and sex, our findings show a positive correlation between DII scores and colorectal cancer, including both colon (proximal and distal) and rectal cancers. Considering that high DII scores indicate a pro-inflammatory dietary pattern, our results support the hypothesis that such diets are associated with increased colorectal cancer risk.

The association between dietary habits and colorectal cancer has been well documented in numerous studies.[18-20] A recent meta-analysis found that healthy dietary patterns are associated with reduced colorectal cancer risk, whereas Western-style diets and alcohol consumption increase risk.[21] The World Cancer Research Fund and the American Institute for Cancer Research conducted a systematic review of the evidence on food, beverages, and colorectal cancer, reporting that processed meats and alcoholic beverages elevate the risk. Conversely, dairy products, whole grains, and foods rich in dietary fiber were shown to reduce the risk of colorectal cancer, colorectal adenomas, and chronic colonic inflammation.[7]

Previous studies have examined the impact of specific foods such as red meat and nutrients, such as folate and zinc on colorectal cancer risk.[18,22,23] However, these foods and nutrients are typically consumed in combination with others, potentially confounding their individual effects. High intercorrelation

Table 3. Association between DII scores and colorectal cancers

Cancer type	**Control/Case**	**OR (95% CI)**	**Adjusted OR* (95% CI)**	**p**
Colorectal cancer	120/149	2.62 (1.69–4.05)	2.72 (1.74–4.27)	<0.001
Colon cancer	120/118	2.37 (1.56–3.60)	2.51 (1.63–3.86)	<0.001
Proximal colon cancer	120/49	3.07 (1.82–5.15)	3.15 (1.83–5.41)	<0.001
Distal colon cancer	120/69	2.14 (1.34–3.42)	2.39 (1.46–3.91)	0.003/<0.001
Rectal cancer	120/30	2.52 (1.37–4.66)	2.59 (1.38–4.89)	3

*Adjusted for age and sex. OR: Odds ratio; CI: Confidence interval; DII: Dietary inflammatory index.

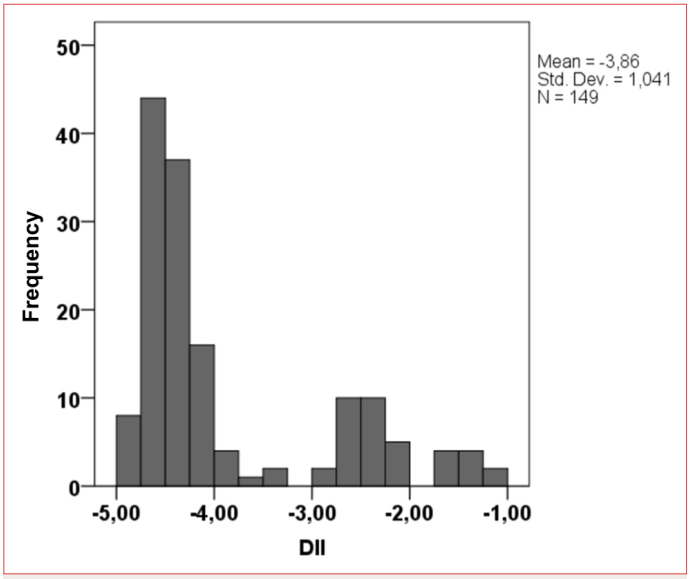

Figure 1. Distribution of dietary inflammatory index scores in the study group.

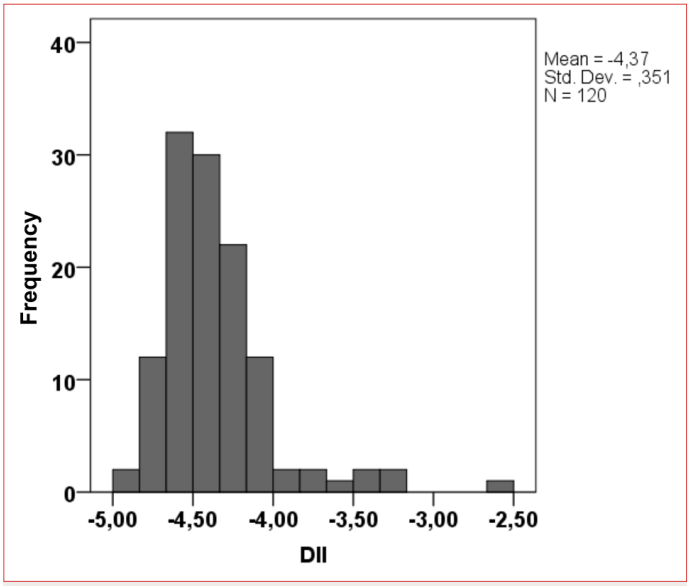

Figure 2. Distribution of dietary inflammatory index scores in the control group.

among food items and the resulting loss of statistical power make it difficult to isolate the risk associated with any single nutrient. Unlike such approaches, the DII was developed by focusing on the functional impacts of dietary components on inflammation. The DII score is based on a systematic review and scoring of the literature regarding inflammation and diet. It also standardizes the intake of pro- and anti-inflammatory dietary components across populations using reference values, allowing for cross-cultural comparisons.[24,25]

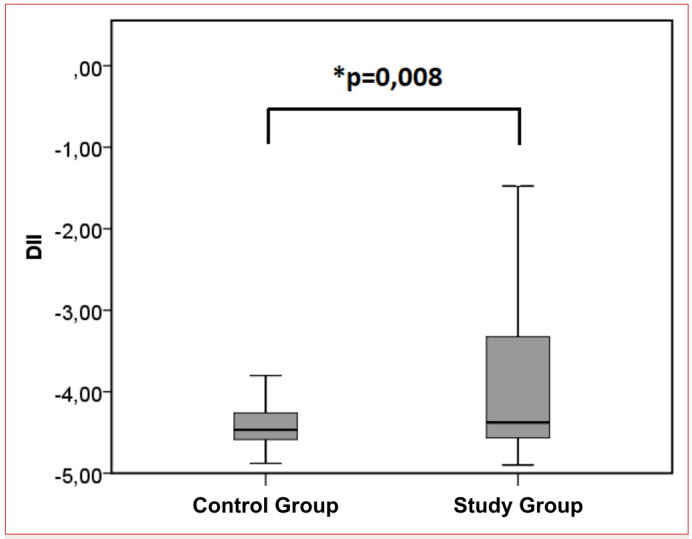

Figure 3. Comparison of dietary inflammatory index (DII) scores between study and control groups. (Mann–Whitney U test, p=0.008 – indicating significantly higher DII scores in the study group).

Therefore, in our study, we investigated the association between colorectal cancer development and DII scores calculated from patients' overall dietary patterns, rather than the intake of specific foods. Shivappa et al.[18] calculated DII scores based on a questionnaire involving 121 food items among 34,703 women aged 55–69 and reported that increased DII scores were associated with higher colorectal cancer incidence. Park et al.[26] found a similar association in a study involving 923 colorectal cancer cases and 1,846 controls using a 106-item semi-quantitative food frequency questionnaire. Harmon et al.[19] conducted a multiethnic cohort study of 190,963 individuals aged 45–75 from various ethnic backgrounds, followed for over 20 years, and found that increased DII scores were linked to greater colorectal cancer risk. Our study supports these findings and confirms the significant association between DII scores and colorectal cancer risk in our population.

The greatest and non-modifiable risk factor for sporadic colorectal cancer is age. The incidence of both colorectal polyps and cancer increases significantly after age 50.[27] Many environmental factors have also been implicated in colorectal cancer development, with dietary patterns being among the most significant. Diet plays a critical role not only in cancer onset but also in the formation and recurrence of polyps.

There are multiple pathways by which pro-inflammatory diets may increase colorectal cancer risk. Such diets can elevate systemic inflammation, potentially inducing insulin resistance, which in turn raises levels of insulin, triacylglycerol, and non-esterified fatty acids. These metabolic disturbances

can promote excessive proliferation of colonic epithelial cells and increase their exposure to reactive oxygen species.[28] Diets rich in red and processed meats are high in N-nitroso compounds, which can damage DNA. In contrast, fruits and vegetables contain micronutrients with antioxidant and anti-tumor properties, as well as fibers that reduce intestinal transit time.

The DII score reflects the collective impact of various foods, nutrients, and flavonoids known to influence inflammation.[29,30] The most potent anti-inflammatory dietary components include polyphenols and antioxidants, which produce localized anti-inflammatory effects, especially through modulation of the gut microbiota. These compounds can also reduce levels of reactive oxygen species and prevent cancer initiation and progression. Phytochemicals found in the diet can inhibit colorectal cancer cell proliferation and interrupt the cell cycle.[31]

The DII is a robust indicator of a person's overall dietary inflammatory potential. High scores reflect a diet with strong pro-inflammatory capacity. It is well established that inflammation increases susceptibility to colorectal cancer.[32-34] Inflammatory cytokines, such as tumor necrosis factor-alpha have been shown to induce insulin resistance by inhibiting insulin receptors. Insulin resistance may contribute to cancer development through elevated insulin, glucose, or triglyceride levels. Furthermore, activation of the cyclooxygenase-2 pathway may promote local proliferation, angiogenesis, and mutagenesis – all of which can be stimulated by cytokines such as interleukin-6. These mechanisms clarify how an inflammatory diet, as defined by high DII scores, contributes to colorectal cancer development. Our findings support these mechanisms by demonstrating their clinical implications.

Dietary habits are shaped by culture and socioeconomic factors and often change over a person's lifetime. People rarely maintain the same diet throughout life, and it is still unclear which nutrients, in what amounts and frequency, and during which life stages exert the greatest influence on cancer development. In our study, DII scores were calculated based on recent dietary intake. However, because cancer develops over extended periods and the timing of its onset is often unclear, more accurate results would require long-term data. Therefore, prospective cohort studies with long-term follow-up are needed for more definitive conclusions.

CONCLUSION

There is a significant association between inflammatory diets, as measured by higher DII scores, and the development of colorectal cancer. Elevated DII scores represent a dietary pattern with high pro-inflammatory potential and were found to be a meaningful risk factor for colorectal cancer, independent of age and sex. This risk does not vary based on tumor location; the association between high DII scores and cancer is consistent for both colon and rectal cancers. These findings suggest that reducing the inflammatory potential of the diet may be an important preventive strategy against colorectal cancer. Further prospective studies involving long-term dietary monitoring and larger populations are recommended to establish more robust evidence and inform public health interventions.

DECLARATIONS

Ethics Committee Approval: The study was approved by University of Health Sciences, Dışkapı Yıldırım Beyazıt Health Practice and Research Center Ethics Committee (No: 91/01, Date: 06/07/2020).

Informed Consent: Informed consent was obtained from the patients.

Conflict of Interest: The authors declare that there is no conflict of interest.

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