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Exploring biomarkers, adipokines, and cytokines in patients with colon cancer: A comparative study with healthy controls

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Cancer is the main reason people die in rich countries and the second most common reason people die in poor countries. Colon cancer (CRC) is the third most common type of cancer in the world. The aim of the study is to analyze and compare the profiles of adipokines and cytokine in colon cancer patients and healthy controls, and determine their potential role in the pathophysiology of colon cancer. This case-control study included 150 colon cancer patients (90 men, 60 women) and 50 healthy controls (25 men, 25 women), aged 55-65 years. The research was conducted at Al-Haboubi Teaching Hospital from 1/1/2024 to 1/9/2024, with ethical approval. Blood samples were collected, clotting, and centrifuged to separate serum. The study found that colon cancer patients had higher BMI, smoking rates, and family history of cancer. They showed lower adiponectin levels and higher levels of leptin, resistin, visfatin, and chemerin. Levels of cytokines (TNF-α, IL-6, IL-10, IFN-γ, IL-1β) were significantly elevated. Lipid profiles revealed increased total cholesterol, LDL, and triglycerides, but lower HDL. Adipokines decreased with cancer stage, while TNF-α increased, with significant correlations among adipokines, cytokines, and lipids. The study links altered adipokines, cytokines, and lipid levels to colon cancer, with elevated TNF-α and reduced adiponectin indicating inflammation and metabolic dysregulation in disease progression.

Keywords: colonic neoplasms; biomarker; adipokine; cytokine; inflammation; tumor necrosis factor-alpha.

Introduction

Men and women in the US are both more likely to get colorectal cancer, which is the third most common type of cancer that leads to death. It is also the second most common type of cancer in the world. The significant utilisation of colonoscopy screening contributes to the decline in occurrence rates in Western nations. However, there is a growing trend of younger individuals contracting the condition (Siegel et al., 2023; Stoffel et al., 2020).

Approximately 5% of instances of colon cancer can be attributed to an inherited genetic mutation, primarily associated with Lynch syndrome (commonly referred to as hereditary nonpolyposis colon cancer or HNPCC) and familial adenomatous polyposis (FAP) (Raphela-Choma et al., 2024). The majority of instances of colon cancer occur randomly. Typically, the transition from a healthy layer of cells in the colon to a cancerous state occurs within a span of a few years. This occurs due to the gradual accumulation of genetic anomalies, leading to the formation of adenomas, which subsequently give rise to the growth of cancer cells. Some kinds of cancer, such as those associated with DNA mismatch repair (MMR) may involve distinct pathways (Gajendran et al., 2019).

Colon cancer screening is recommended and can be conducted using many methods. Various groups have distinct regulations for the initiation of screening and subsequent procedural measures. A colonoscopy is typically employed to obtain a tissue specimen for the purpose of confirming a diagnosis of colon cancer (Smith et al., 2019).

All individuals newly diagnosed with colon cancer should get a comprehensive colonoscopy and baseline carcinoembryonic antigen (CEA) test to detect prevalent genetic abnormalities. For individuals with invasive cancer, a baseline lung and abdominal-pelvic computed tomography (CT) scan is necessary (Kadhum Kharmeet et al., 2024).

Colorectal cancer ranks as the third most prevalent kind of cancer in the United States. The Surveillance, Epidemiology, and End Results (SEER) database recorded a total of 153,000 newly diagnosed cases of colon cancer in the year 2023. This represents 7.8% of the total number of newly diagnosed cancer cases. Approximately 70% of these malignancies occur in the colon, while the remaining 30% are located in the rectal region. Colon cancer affects 5.3% of men, making them more susceptible to it compared to women who typically develop it at a later age (68 vs. 72 years old) (Siegel et al., 2023).

Prior studies have demonstrated that the degree of risk associated with being overweight is contingent upon both the individual's gender and the particular location of the cancer within the body. Individuals with obesity, defined as having a body mass index (BMI) of 30 kg/m^2 or more, have an increased susceptibility to developing colon cancer. Colon cancer has a higher probability of occurring compared to rectal cancer, with men being more susceptible to it than women (Renehan et al., 2008; Ghodousi-Dehnavi et al., 2023).

Additionally, both men and women with abdominal adiposity, as measured by waist circumference or waist-to-hip ratio, have a strong association with colon cancer. Multiple systematic reviews and meta-analyses have synthesised the available evidence. Among women, the placement of fat is a more influential risk factor for colon cancer than body weight or BMI, as indicated by this data. There is a connection between changes in weight and an increased risk of colon cancer in individuals who have a high waist circumference (WC) at the age of 50 (Renehan et al., 2010).

Adipokines are bioactive substances that are secreted by adipose tissues. There are many chemicals in this group, such as adiponectin, leptin, resistin, interleukin-6 (IL-6), and others. Many bodily functions are controlled by adipokines, such as hunger, growth, metabolism, fat storage, insulin sensitivity, blood pressure, immune reaction, inflammation, and blood clotting (Ruiz-Fernández et al., 2019). The accumulation and subsequent release of several adipokines in the adipose tissue of obese individuals has been associated with various health complications, such as rheumatoid arthritis, osteoarthritis, spi-

nal disc degeneration, and heart disease. Specifically, adipokines induce the production of proinflammatory cytokines, so establishing a link between obesity and the aforementioned diseases. Adipokines associated with adipose tissue have been substantially correlated with the onset of colorectal cancer (Molica et al., 2015).

Scientists have studied cytokines, mainly IL-6 and IL-8, as possible biomarkers that could help tell the difference between healthy and cancerous oral tumours. To find interleukins, many electrical and visual biosensors have been made (Kaur et al., 2022). Extensive research conducted on clinical units and animal models has identified numerous cytokines that exhibit substantial associations with various disorders. As a result, the Biology Collector (BIOCO) was developed and implemented in clinics to facilitate research on cytokine development platforms (Liu et al., 2021). Identifying and treating diseases using cytokines is crucial due to their ability to enhance immune system functionality. This is similar to the mechanism of sepsis based on pathophysiology, in which pro- and anti-inflammatory cytokines have distinct functions (Chaudhry et al., 2015). Identifying certain cytokines in the bloodstream enables physicians to detect cancer, select the most effective treatment, and monitor the disease's progression non-invasively and cost-effectively (Febbo et al., 2011).

The aim of this study is to analyze and compare the adipokines and cytokine in colon cancer patients and healthy controls, and determine their potential role in the pathophysiology of colon cancer.

Materials and methods

The case-control study included 150 individuals diagnosed with colon cancer, divided into 90 men and 60 women, plus 50 healthy controls divided into 25 men and 25 women. The ages ranged from 55 to 65 years. The Committee on Publication Ethics at the Thi-Qar Health Directorate, Al-Habbobi Teaching Hospital gave its approval to the study.

The study took place from January 1, 2024, to September 1, 2024, at the Cancer Center at Al-Haboubi Teaching Hospital. All of the people who took part in the study gave their ethical approval. Each person had 5 mL of blood drawn and put into a gel tube. The tube was then left at room temperature for 15 minutes until the blood clotted. A centrifuge spinning at 3500 rpm was used to separate the blood serum. The serum was then put into clean preservation tubes and kept at –20 °C until it was needed. We used colorimetric spectrophotometry to find the amounts of total cholesterol, LDL, HDL, and triglycerides, following the manufacturer's instructions from Biolabo, France. Levels of the following substances were measured: adiponectin (µg/mL), leptin (ng/mL), resistin (ng/mL), visfatin (ng/mL), and chemerin (ng/mL); TNF-α (pg/mL), IL-6 (pg/mL), IL-10 (pg/mL), IFN-γ (pg/mL), and IL-1β (pg/mL). The manufacturer, Bio-Techne (R&D Systems, USA).

Statistical analyses were conducted using SPSS (version 26). Data are expressed as mean ± standard deviation. Independent and dependent two-tailed t-tests were applied for normally distributed variables, while the Mann–Whitney U and Wilcoxon tests were used for non-parametric data. A P-value < 0.05 was considered statistically significant.

Results

The social and biological traits of the 150 people with colon cancer and the 50 people in the comparison group were looked at. In terms of age, there was no big difference between the groups (P = 0.88). But there were a lot more men in the colon cancer group (P < 0.01), they were heavier (28.4 ± 3.1 vs. 24.9 ± 2.5, P < 0.01), they smoked more (P < 0.01), and 26.7% of them had a family history of cancer, compared to 10% of the other group. These results can be seen in Table 1 and Figure 1.

Adipokine levels were compared between the colon cancer group (n = 150) and the control group (n = 50). The results showed a significant decrease in adipokine levels in the colon cancer group (5.1 ± 1.2 µg/mL) compared to the control group (12.3 ± 2.1 µg/mL, P < 0.001). Similarly, leptin levels in the colon cancer group were significantly higher (22.4 ± 3.8 ng/mL) compared to the control group (11.2 ± 2.4 ng/mL, P < 0.001). Restin levels in the colon cancer group (14.7 ± 2.3 ng/mL) were significantly increased compared to the control group (9.5 ± 1.9 ng/mL, P < 0.001). Also, visfatin levels were higher in the colon cancer group (6.2 ± 1.5 ng/mL) than in the control group (3.4 ± 0.7 ng/mL, P < 0.001). Finally, the colon cancer group had higher levels of chemerin (15.3 ± 2.9 ng/mL) than in the control group (10.0 ± 1.5 ng/mL, P < 0.001). These findings are presented in Table 2 and Figure 2.

Table 1
Comparison of age, gender, BMI, smoking status, and family history of cancer

Characteristic	Colon cancer group (n = 150)	Control group (n = 50)	P-value
Age, years	60.5 ± 10.2	58.7 ± 9.5	0.88[NS]
Gender (male)	90	25	<0.01
Gender (female)	60	25	
BMI, kg/m²	28.4 ± 3.1	24.9 ± 2.5	<0.01
Smoking status (Yes)	65	15	<0.01
Smoking status (No)	85	35	
Family history of cancer	40 (26.7%)	5 (10%)	<0.01

Table 2
Differences in adiponectin, leptin, resistin, visfatin, and chemerin between groups

Adipokine	Colon cancer group (n = 150)	Control group (n = 50)	P-value
Adiponectin, µg/mL	5.1 ± 1.2	12.3 ± 2.1	<0.001
Leptin, ng/mL	22.4 ± 3.8	11.2 ± 2.4	<0.001
Resistin, ng/mL	14.7 ± 2.3	9.5 ± 1.9	<0.001
Visfatin, ng/mL	6.2 ± 1.5	3.4 ± 0.7	<0.001
Chemerin, ng/mL	15.3 ± 2.9	10.0 ± 1.5	<0.001

Cytokine levels were compared between the colon cancer group (n = 150) and the control group (n = 50). The results showed a significant increase in TNF-α level in the colon cancer group (65.4 ± 12.8 pg/mL) compared to the control group (22.3 ± 5.1 pg/mL, P < 0.001). IL-6 levels were also significantly higher in the colon cancer group (28.9 ± 5.6 pg/mL) compared to the control group (8.1 ± 2.3 pg/mL, P < 0.001). IL-10 levels were also significantly higher in the colon cancer group (15.2 ± 3.9 pg/mL) compared to the control group (5.6 ± 1.1 pg/mL, P < 0.001). Also, IFN-γ levels were higher in the colon cancer group (18.5 ± 4.5 pg/mL) compared to the control group (10.2 ± 1.7 pg/mL, P < 0.001). Finally, IL-1β levels in the colon cancer group (12.8 ± 2.2 pg/mL) were significantly increased compared to the control group (4.9 ± 1.4 pg/mL, P < 0.001). These findings are presented in Table 3 and Figure 3.

Table 3
Comparative analysis of TNF-α, IL-6, IL-10, IFN-γ, and IL-1β

Cytokine	Colon cancer group (n = 150)	Control group (n = 50)	P-value
TNF-α, pg/mL	65.4 ± 12.8	22.3 ± 5.1	<0.001
IL-6, pg/mL	28.9 ± 5.6	8.1 ± 2.3	<0.001
IL-10, pg/mL	15.2 ± 3.9	5.6 ± 1.1	<0.001
IFN-γ, pg/mL	18.5 ± 4.5	10.2 ± 1.7	<0.001
IL-1β, pg/mL	12.8 ± 2.2	4.9 ± 1.4	<0.001

Lipid levels were compared between the colon cancer group (n = 150) and the control group (n = 50). The results showed that total cholesterol levels were significantly higher in the colon cancer group (230.4 ± 35.2 mg/dL) than in the control group (185.2 ± 30.1 mg/dL, P < 0.001). LDL levels were also higher in the colon cancer group (150.2 ± 28.7 mg/dL) than in the control group (110.4 ± 25.6 mg/dL, P < 0.001). In contrast, HDL levels were significantly lower in the colon cancer group (40.3 ± 10.1 mg/dL) than in the control group (55.0 ± 12.5 mg/dL, P < 0.001). Also, triglyceride levels in the colon cancer group (180.1 ± 40.3 mg/dL) were significantly increased compared to the control group (120.2 ± 32.1 mg/dL, P < 0.001). These findings are presented in Table 4 and Figure 4. The relationship between adipokine and TNF-α levels and cancer stage in colon cancer patients was

analyzed. The results showed a significant decrease in adipokine levels with the progression of the disease stages; the mean adipokine level in stage I was 10.5 ± 2.4 μg/mL, while it decreased to 8.7 ± 1.8 μg/mL in stage II, 4.2 ± 1.2 μg/mL in stage III, and 2.5 ± 0.8 μg/mL in stage IV, with a P value of less than 0.001. Similarly, TNF-α levels were significantly increased with the progression of the stage; While they were 25.2 ± 4.1 pg/mL in the first stage, they rose to 38.5 ± 6.3 pg/mL in the second stage, and 60.3 ± 9.4 pg/mL in the third stage, and reached 70.2 ± 11.5 pg/mL in the fourth stage, with a probability value of less than 0.001. These findings are presented in Table 5 and Figure 5.

Spearman correlation analysis revealed significant associations among adipokinin, leptin, TNF-α, IL-6, lipid profiles, and clinical parameters. Adipokinin showed a significant negative correlation with leptin (r = –0.65), TNF-α (r = –0.72), IL-6 (r = –0.63), total cholesterol (r = –0.78), and LDL (r = –0.75), while it was positively correlated with HDL (r = 0.60) and negatively with triglycerides (r = –0.55). Leptin positively correlated with TNF-α (r = 0.60), IL-6 (r = 0.70), total cholesterol (r = 0.55), and LDL (r = 0.68), but negatively with HDL (r = –0.55) and triglycerides (r = 0.50). TNF-α and IL-6 showed positive correlations with total cholesterol, LDL, and triglycerides, and negative correlations with HDL. Additionally, total cholesterol and LDL were strongly correlated (r = 0.85) and negatively correlated with HDL. These results are summarized in Table 6 and Figure 6.

Table 4
Differences in total cholesterol, LDL, HDL, and triglycerides

Lipid parameter	Colon cancer group (n = 150)	Control group (n = 50)	P-value
Total cholesterol, mg/dL	230.4 ± 35.2	185.2 ± 30.1	<0.001
LDL, mg/dL	150.2 ± 28.7	110.4 ± 25.6	<0.001
HDL, mg/dL	40.3 ± 10.1	55.0 ± 12.5	<0.001
Triglycerides, mg/dL	180.1 ± 40.3	120.2 ± 32.1	<0.001

Table 5
Relationship between adiponectin, TNF-α levels, and disease stage

Biomarker	Stage I (n = 30)	Stage II (n = 50)	Stage III (n = 40)	Stage IV (n = 30)	P-value
Adiponectin, μg/mL	10.5 ± 2.4	8.7 ± 1.8	4.2 ± 1.2	2.5 ± 0.8	<0.001
TNF-α, pg/mL	25.2 ± 4.1	38.5 ± 6.3	60.3 ± 9.4	70.2 ± 11.5	<0.001

Table 6
Relationships between adiponectin, leptin, TNF-α, IL-6, lipid profile, and clinical parameters

Parameter	Adiponectin	Leptin	TNF-α	IL-6	Total cholesterol	LDL	HDL	Triglycerides
Adiponectin	1	–0.65	–0.72	–0.63	–0.78	–0.75	0.60	–0.55
Leptin	–0.65	1	0.60	0.70	0.55	0.68	–0.55	0.50
TNF-α	–0.72	0.60	1	0.80	0.70	0.65	–0.60	0.45
IL-6	–0.63	0.70	0.80	1	0.75	0.50	–0.55	0.55
Total cholesterol	–0.78	0.55	0.70	0.75	1	0.85	–0.65	0.90
LDL	–0.75	0.68	0.65	0.50	0.85	1	–0.70	0.80
HDL	0.60	–0.55	–0.60	–0.55	–0.65	–0.70	1	–0.55
Triglycerides	–0.55	0.50	0.45	0.55	0.90*	0.80	–0.55	1

Discussion

Table 1 highlights significant differences in sociodemographic and clinical characteristics between the colon cancer and control groups. There was no significant age difference (p = 0.88), consistent with studies focusing on single-age groups. However, studies such as that by Lewandowska et al. (2022) emphasize age as a risk factor, with increased colon cancer incidence due to genetic mutations. The lack of significance here may be due to a narrower age range or early regional onset (Lewandowska et al., 2022). This study observed a significantly higher male predominance in the colon cancer group (P < 0.01), consistent with global trends, as men generally have a higher risk of developing colon cancer. This disparity may be attributed to hormonal factors, such as the protective effect of estrogen in women, or lifestyle differences, including dietary habits, which may contribute to the higher incidence in one gender (Yu et al., 2022).

A significantly higher BMI was observed in the colon cancer group compared to the control group (P < 0.01), in line with many studies linking excess weight to an increased risk of colon cancer. Obesity contributes to carcinogenesis through chronic inflammation, insulin resistance, and adipokine dysregulation. However, studies, such as that by Li et al. (2022), suggest that the relationship between BMI and colon cancer risk may vary due to factors like age, lifestyle, comorbidities, and population differences (Li et al., 2022). The higher smoking rate in the colon cancer group (P < 0.01) aligns with studies linking smoking to an increased risk of colon cancer due to carcinogens affecting the gastrointestinal tract. However, not all studies show the same association, likely due to differences in smoking intensity, duration, or genetic and environmental factors. Additionally, the greater family history of cancer in the colon cancer group (P < 0.01) supports the well-established role of genetic predisposition in colon cancer risk, particularly hereditary conditions like familial adenomatous polyposis (FAP) and Lynch syndrome (Chen et al., 2021). However, recent studies such as that by Hossain et al., et al (2022), further demonstrate that lifestyle factors (e.g. diet, physical activity) may contribute to cancer in the absence of a family history, suggesting that the clinical interplay between genetic and environmental factors is highly complex (Hossain et al., 2022). As shown in Table 2, the levels of adipokines (adiponectin, leptin, resistin, visfatin, and chemerin) were significantly higher in the colon cancer group compared to the control group (P < 0.001). These results align with studies linking dysregulated adipokine profiles to oncogenesis, particularly in obesity-related cancers like colon cancer. Additionally, consistent with Mhaidat et al. (2022), adiponectin levels were significantly lower in the colon cancer group (5.1 ± 1.2 vs. 12.3 ± 2.1 μg/mL in the control group) (Mhaidat et al., 2022). On the other hand, higher leptin levels in the cancer patients (22.4 ± 3.8 ng/mL against 11.2 ± 2.4 in the control group) are supported by studies such as those by Wang et al., (2022), suggesting a role for leptin in enhancing cancer cell proliferation and metastasis through the activation of inflammatory pathways such as NF-κB (Jiang et al., 2022; Wang et al., 2021).

Increased circulating levels of resistin (14.7 ± 2.3 vs. 9.5 ± 1.9 ng/mL), visfatin (6.2 ± 1.5 vs. 3.4 ± 0.7 ng/mL), and chemerin (15.3 ± 2.9 vs. 10.0 ± 1.5 ng/mL) in our study are also in line with previously published data, which found associations between these adipokines and inflammatory pathways, insulin resistance, and tumorigenesis, as reported by Pham et al. (2022). Alternatively, studies such as that by Neira et al. (2024) found no significant difference in adipokine levels among certain cancer patient subgroups, potentially due to variations in population characteristics, sample size, or cancer stages. In conclusion, this study supports the hypothesis that adipokines play a critical role in regulating inflammatory and metabolic pathways linked to colon cancer. The findings emphasize the importance of adipokine levels in colon cancer while highlighting the variability across studies, which can be attributed to differences in methodology, populations, and disease stages (Özlü et al., 2023; Neira et al., 2024).

Table 3 shows significant differences between the colon cancer patients and the control group in all cytokines (higher in the colon

cancer patients); TNF-α (165 vs 65 pg/mL in controls), IL-6 (895 vs 275 pg/mL), IL-10 (81 vs 29 pg/mL), IFN-γ (593 vs 240 pg/mL), and IL-1β (675 vs 238 pg/mL), which indicates comparison values in the colon cancer group vs the control group ($P < 0.001$). These results also have been corroborated by a range of studies reporting that pro-inflammatory cytokine contributes significantly towards tumorigenesis, and tumor progression (Bhat et al., 2022). These cytokines are responsible for providing an inflammatory environment necessary for tumor growth. For instance, other inflammatory mediators, such as TNF-α and IL-6, are known to enhance cancer cell survival, proliferation, and metastasis by triggering some signaling pathways associated with chronic inflammation, including NF-κB and JAK/STAT. Moreover, increased levels of IL-10, an anti-inflammatory cytokine, can reflect a compensatory mechanism that attempts to restore the balance toward reducing inflammation (Bai et al., 2021; Muthusami et al., 2021).

High levels of IFN-γ suggest an immune response can be induced by cancer, and are even elevated in early pre-cancer, but due to the context-dependent nature of IFN-γ in cancer (both tumor-suppressive and tumor-promoting), it must be interpreted carefully. These findings are consistent with studies by Roberts et al. (2024) and Ganesh et al. (2022), who found similar upregulation of cytokines in colon cancer. However, some reports, such as those by Koper-Lenkiewicz et al. (2021), have shown only modest increases in cytokine levels in cancer patients, possibly due to differences in population characteristics, disease stage, or sample size. This study also observed specific elevations in pro-inflammatory cytokines in the colon cancer group, with some of these cytokines potentially serving as biomarkers for the diagnosis and prognosis of colon cancer, provided the mechanisms involved are well studied in the lesions (Koper-Lenkiewicz et al., 2021).

As shown in Table 4, colon cancer patients exhibited significant lipid abnormalities compared to the control group, including higher total cholesterol, LDL, and triglycerides, and lower HDL ($P < 0.001$). Specifically, total cholesterol (230.4 vs. 185.2 mg/dL), LDL (150.2 vs. 110.4 mg/dL), and triglycerides (180.1 vs. 120.2 mg/dL) were higher, while HDL (40.3 vs. 55.0 mg/dL) was lower in the colon cancer group. These findings align with studies by Fang et al. (2021) and Ecker et al. (2021), highlighting altered lipid profiles in cancer patients. High cholesterol and LDL levels are linked to cancer progression, potentially due to inflammation and oxidative stress. Additionally, the low levels of HDL in the cancer group correlate with findings in the literature that HDL has anti-inflammatory and anti-tumor effects, and when it is diminished, this may facilitate tumorigenesis and metastasis (Loosen et al., 2022). These higher triglyceride levels noted in our study are consistent with the data from Yarla et al. (2022), which tied triglyceride levels to poor cancer outcomes. For example, the relationship between lipids and cancer is multifaceted and highly context driven (Yarla et al., 2022).

Table 5 demonstrates a significant correlation between serum adiponectin and TNF-α levels with the progressive stages of colon cancer. Adiponectin (ADPN) levels decrease markedly from 10.5 ± 2.4 µg/mL in Stage I to 2.5 ± 0.8 µg/mL in Stage IV ($P < 0.001$), while TNF-α levels increase from 25.2 ± 4.1 pg/mL in Stage I to 70.2 ± 11.5 pg/mL in Stage IV ($P < 0.001$). These findings align with previous studies, which suggest that low adiponectin levels are associated with advanced cancer stages and poorer prognosis, highlighting its anti-inflammatory and anti-tumor properties (Mihajlović et al., 2022; Marques et al., 2023). Inverse associations between adiponectin and TNF-α support the idea that reduced adiponectin in late cancer may reflect a dysregulated immune reaction and heightened systemic inflammation. TNF-α, a pro-inflammatory cytokine, is also known to be upregulated and thought to promote colon cancer tumorigenesis, metastasis and chemoresistance (Sinicrope et al., 2021).

The observed elevation of TNF-α levels in accordance with the development of the disease in this study is consistent with results obtained by Zheng et al. (2022) TNF-α may promote tumorigenesis by triggering multiple signaling pathways that promote the proliferation and survival of tumor cells (Zheng et al., 2022). The decline in adiponectin and the rise in TNF-α across cancer stages may serve as potential biomarkers for monitoring colon cancer progression. Variations in study results could be due to differences in sample size, patient characteristics, or laboratory methods used to quantify these markers. Despite its protective role in cancer, adiponectin's interactions with various adipokines and cytokines complicate its use as a sole prognostic marker (Capuozzo et al., 2023).

Conclusion

The study concludes that colon cancer is associated with significant alterations in adipokine and cytokine levels, alongside disrupted lipid profiles. Elevated levels of TNF-α and decreased adiponectin are strongly correlated with advanced disease stages, indicating their role in inflammation and metabolic dysregulation linked to cancer progression. Higher levels of leptin, resistin, visfatin, and chemerin, coupled with increased total cholesterol, LDL, and triglycerides, and decreased HDL, underscore the metabolic disturbances in colon cancer patients. These findings suggest that monitoring these biomarkers could enhance understanding of disease mechanisms and potentially improve diagnostic and prognostic approaches to colon cancer.

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