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A Discussion on the Relationship of the Gut Microbiome with Primary Tumor Location, Arginase-1 and Tumor Associated Neutrophils in Colorectal Cancer

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Abstract

Colorectal cancer is one of the most common malignant cancers in the world with worrying epidemiological data both in terms of occurrence and demographics, with an ever lower age at diagnosis. In the last decade, an ever increasing focus has been put on the tumor microenvironment and the effect of the microbiome on tumor etiopathogenesis and progression. This paper proposes a focused perspective based on a defined conceptual framework describing how the microbiome's different composition along the gut segments interacts with primary tumor location, perineural invasion, Arginase-1, and tumor associated neutrophils (TANs). This discussion will go in parallel with addressing the impact of these aforementioned players on the clinical outcome of the disease and the embryological, vascular, molecular, and immunological differences influencing tumor sidedness and microbiome-related mechanisms. The importance of such perspective is to aid in the development of new therapies by understanding these underlying targetable mechanisms.

Introduction

Colorectal cancer (CRC) is one of the most common malignant cancers in the world with an epidemiological scenario of increasing worrying tendencies. An increase in incidence in the United States of America (USA) of approximately 3% annually in adults aged 20-49 years and an increase of 0.4% in adults aged 50-64 years is observed (Siegel et al., 2026). This translates to a total of 158 000 new cases of colorectal cancer per year. A new epidemiological landscape is unveiling in the developed world, with a significantly lower age at diagnosis of colorectal cancer with almost half of all new cases occurring in patients with age lower than 65 years, becoming the leading cause of death from cancer in adults with age lower than 50. Several factors could be agents responsible for such numbers: obesity, sedentary lifestyles, pesticides, pollution and high alcohol consumption, just to cite a few. These factors, coupled with a lack of screening in young adults, result in such terrifying epidemiological numbers.

When discussing CRC it is of note to discuss the plethora of differences amongst CRCs, emphasizing the huge differences also present in physiological condition along the colon and rectal area. New studies revealed the importance and correlation of new undiscovered areas in the study of CRC, such

as the impact of microbiome on the pathology and the role of the immunological arm of the tumor microenvironment on the disease etiopathogenesis and progression, and how it is of vital importance to find new markers for diagnosis and clinical management of the disease. This manuscript intends to advance the field beyond existing reviews by providing an integrative analysis connecting anatomical sidedness, specific pathobiont dynamics, microbial metabolites, and complex myeloid configurations.

Main Body

In terms of the microbiome, it is important to say that there are significant variations of populations of microbiota along the colon. A proximal to distal change in microbiota is present. Starting from the right part of the colon, a gradient dependent change occurs, this due to the change in the colon's lumen environment when going from proximal to distal areas. This change in environment involves a change in pH, physiological activity and physiological role of colonic area and nutrient availability. The right part of the colon involves a function of high fermentation compared to the rest of the colon, thus it contains a population of bacteria which include *Veillonella* and *Coprobacter*. Here a high amount of undigested carbohydrates are present. Moving along the colon tract is where a high amount of pathogens is seen (during active disease) with different microbial populations. Moving even more distally towards the rectal mucosa, the lowest rate of alpha diversity in the microbiome is seen. Alpha diversity in the microbiome is the diversity of the bacteria (and more generally of microorganisms) within the single habitat in discussion, in this case the rectal mucosa. This all is affected by several factors for instance exogenous factors (eg diet), breast milk feeding and eutocia (Yang et al., 2025).

Further differences in the microbiome are seen whether the microbiome is associated with the mucosa (called the Mucosa-Associated Microbiota - MAM) or the fecal microbiota. The MAM is characterized by lower diversity but a higher concentration of immune system interacting microorganisms such as the *Fusobacteria* and *Burkholderia* (Yin et al., 2024). The fecal associated microbiota has higher percentages of Firmicutes and Actinobacteria (Nowicki et al., 2023).

This discussion of the changes in the microbiome associated with the colorectal tract is very relevant when discussing colorectal cancer. Several examples can be discussed in terms of changes in the microbiome and colorectal cancer. The tumor itself hosts a different microbiome compared to physiological healthy tissue, the tumor one being high in populations of *Fusobacterium nucleatum* and *Campylobacter spp* (Olausson et al., 2023). These variations in microbiota population not only vary in terms of healthy versus pathological tissue but also within the pathological tissue, regarding its stage. *Atopobium parvulum* is associated with early stage adenomas and decrease in concentration in later stages adenoma and carcinoma. While bacteria such as *Fusobacterium* remain high in concentration also in advanced malignancies, from early stage to advanced metastatic (Rebersek et al., 2021).

Studies suggested that microbiome populations could serve even as indicators of advanced stage of disease, for instance regarding high concentrations of *Rothia* and *Streptococcus* that in a recent study were significantly associated with lymph node metastasis in CRC (Zhao et al., 2024). A further interesting study revealed how CRC with presence of metastasis had lower levels of Lactobacillales compared to the groups without presence of metastasis, revealing how microbiome signatures can truly have an impact in terms of staging markers (Sun et al., 2022).

To fully understand these shifts, we must evaluate specific studies on CRC-associated microbiota and microbial metabolites. A key study revealed that gut bacteria identified in colorectal cancer patients can promote tumorigenesis via butyrate secretion (Okumura et al., 2021). While butyrate is generally seen as beneficial for normal colonocytes, in the context of tumor cells it can promote tumor growth by inducing oncogenic epigenetic modifications. Furthermore, *Fusobacterium nucleatum* functions as a critical pathobiont, and its role is closely linked to microenvironmental factors such as iron. A recent study demonstrated that iron accelerates *Fusobacterium nucleatum*-induced CCL8 expression in macrophages, which directly promotes colorectal cancer progression (Yamane et al., 2022). Other important pathobionts include enterotoxigenic *Bacteroides fragilis* and *pks*-positive *Escherichia coli*, which contribute to mucosal damage, inflammation, and DNA damage through the secretion of specific toxins and genotoxins like colibactin.

Differences amongst colorectal cancers in regards to the primary tumor location have a broader spectrum alongside changes in microbiome. In terms of clinical outcome, proximal tumors (right-sided colon cancers) tend to have a worse prognosis than distal tu-

mors. This is due also to the fact that symptomatic presentation of the disease generally tends to be present at a more advanced stage, with more silent symptoms such as anemia. Distal cancers present at an earlier stage with symptoms such as obstruction. Right-sided cancers also present higher rates of BRAF-KRAS mutations and tend to have a more active immunogenic tumor microenvironment, responding better to immunotherapies (Baran et al., 2018). These cancers are also often associated with high microsatellite instability (MSI-high) and a high immune infiltrate. Distal cancers (sigmoid/rectal) are more associated with APC mutations and Chromosomal Instability (CIN). These distal cancers also tend to respond better to anti-EGFR therapies compared to proximal cancers. Proximal cancers are epidemiologically more present in women and elderly patients and have a 25% higher risk of death compared to distal colorectal cancers (Gholamalizadeh et al., 2023). Furthermore, left-sided colorectal cancers tend to have a metastatic spread more focused on the lungs and the liver while right-sided colon cancers tend to have a metastatic spread more to the peritoneum.

Importantly, the biological differences between right- and left-sided colon cancers cannot be explained only by microbiome gradients or clinical presentation. They are heavily influenced by embryological and anatomical differences, including the midgut origin and superior mesenteric artery supply of the right colon, and the hindgut origin and inferior mesenteric artery supply of the left colon (Kim et al., 2019). These developmental and vascular features determine the baseline physiological and immunological characteristics of each segment. The right colon has a midgut origin and is supplied by the superior mesenteric artery, which correlates with a high rate of bacterial biofilms, MSI-high status, and an immune-rich environment. The left colon has a hindgut origin and is supplied by the inferior mesenteric artery, which correlates with a higher rate of chromosomal instability, planktonic bacteria, and a different lymphoid structure. These developmental, vascular, molecular, and immunological differences must be considered when discussing tumor sidedness and microbiome-related mechanisms. A further and relevant difference to discuss in terms of the tumor primary location within the umbrella term Colorectal Cancer (CRC) is the rectal versus colon cancer primary tumor location. If the cancer occurs in the last 6 to 8 inches of the large intestine then it will give rise to rectal cancer. Rectal cancer is a cancer which tends to be more aggressive than colon cancer, with higher recurrence and more difficult mode of treatment.

In colorectal cancer an ever increasing focus has been put on finding new target molecules and components in the tumor microenvironment which could aid in the treatment and management of the pathology and clinical course. Arginase-1 and tumor associated neutrophils (TANs) have been studied in terms of their ever revealing role in CRC progression and etiopathology. L-Arginine supplementation or Arginase inhibition increases immunitary response in the tumor microenvironment, thus supporting the scientific theory that Arginase-1 inhibits immunitary response to the tumor (Zhang et al., 2025). A recent study also revealed how important is gut microbiota also in terms of its relation with this latter pathway, as gut microbiota depletion reduced L-Arginine through Arginase production compromising anti-tumoural immunity (Zhang et al., 2025).

The role of Arginase-1 (Arg-1) is to convert L-Arginine into Ornithine and Urea and acts pleiotropically in immunitary functions. In the colorectal tract Arginase-1 is being researched for its relations and possible effects with a plethora of pathologies, including cancer. In a recent study Arg-1 expression has been correlated with the levels of inflammation in Inflammatory Bowel Disease (IBD). In this same study in mice, L-Arginine cycle and microbiota alteration have been correlated to decrease resolution of colitis and to a plethora of changes such as: decreased vessel density, changes in intestinal microbiota and a lower myeloid cells infiltrate (Baier et al., 2020). The intestinal microbiota has a direct effect on Arginine catabolism and its cycle of metabolism.

A further study has found insights on just how deep a bidirectional relationship L-Arginine has with the gut microbiota. In fact it was revealed that L-Arginine induced a gut microbiota remodelling which had an immune defence boost in terms of protection against lung infection through the protective effect of the gut-lung axis (Kim et al., 2022). The microbiota in the intestine develops a sort of connection with the host cells in regards to Arginine catabolism processing. In a recent study, reduced Arginine catabolism by the intestinal microbiota lead to an increase in Arginine within the tumor microenvironment and as a consequence causing the activation of nitric oxide synthesis and polyamine synthesis which caused a cascade of events such as angiogenesis, M2 polarization of macrophages and accelerating colorectal cancer progression (Xu et al., 2025).

Bacteria have an impact also due to their presence intratumorally, this done also through their interactions with Tumor Associated

Neutrophils (TANs). This interaction has been identified as a significant feature in terms of tumor etiopathogenesis and progression (Hu et al., 2024). In fact it has been revealed that intratumoral bacteria promote neutrophil infiltration and therefore the creation of neutrophil extracellular traps (NETs) which become a driving force to tumor progression and formation. However at the same time TANs have been shown to limit the number of bacteria intratumorally (Hu et al., 2024).

Importantly, TAN biology cannot be adequately summarized by a simple pro-tumor narrative. Recent paradigms show that neutrophil heterogeneity and the limitations of the N1/N2 framework must be carefully considered (Eruslanov et al., 2025). Neutrophils display significant context-dependent anti- and pro-tumor functions, which are shaped by their maturation state and specific cytokine and chemokine pathways. The anti-tumor functions of TANs include direct cellular cytotoxicity and activation of adaptive immunity. In contrast, their pro-tumor functions involve the formation of Neutrophil Extracellular Traps (NETs) that can physically protect tumor cells and promote metastasis. Cytokine and chemokine pathways, such as the CXCL1, CXCL2, and CXCL8 axes, play a major role in regulating the recruitment and behavior of these distinct neutrophil subsets within the tumor microenvironment.

Recent studies shed new light on the possible relationships between TANs, Arginase-1 and prognosis. In a recent analytical research, patients who did not undergo neoadjuvant therapy and had no perineural invasion from the tumor, had a statistically significant higher level of Arginase-1 and TANs and had a significantly worse prognosis (in terms of Disease-Free Survival) compared to patients who underwent neoadjuvant therapy and had no perineural invasion. When considering the whole group of patients, including perineural invasion patients, patients who underwent no-neoadjuvant therapy had a significantly higher amount of Arginase-1 and tumor associated neutrophils (TANs) compared to the group who did undergo neoadjuvant therapy (Sarandria, 2025). Furthermore, this study highlighted etiopathological features of tumor microenvironment and immunological environment in regards with tumor-neural interaction. Perineural invasion was associated in a statistically significant way to a higher concentration of TANs (CD 66b) and Arg-1, supporting the neuro-immuno-oncological theory that perineural invasion leads to an immuno-depressive tumor microenvironment, "cold" tumor microenvironment, (Xu et al., 2025) also associated with TANs, which could possibly be due to immunosuppressive polarization or specific maturation states, and an association with an increase in Arginase-1. Furthermore, a recent paper also revealed a statistically significantly better prognosis in patients who underwent therapy (either chemotherapy or chemo-radio therapy) but whose tumor had no perineural invasion in comparison to patients whose tumor had presence of perineural invasion (Sarandria, 2026).

In the translational setting, the therapeutic targeting of the TAN-Arginase-1 axis is rapidly evolving, with several clinical trials currently underway. The first-in-class oral arginase inhibitor, INCB001158, has demonstrated the ability to inhibit plasma arginase activity and increase circulating arginine, which successfully reverses the profound immunosuppressive effects exerted by neutrophils and myeloid-derived suppressor cells (Calithera Biosciences, 2019). This agent is being evaluated in combination with the checkpoint inhibitor pembrolizumab for advanced solid tumors, showing preliminary potential to improve overall response rates in typically refractory microsatellite-stable (MSS) colorectal cancers (Journal of Clinical Oncology, 2021). Parallel to arginase inhibition, targeting the physical recruitment of TANs is being tested through CXCR2 antagonists (Nywening et al., 2018). Clinical trials have evaluated agents such as navarixin, a small-molecule CXCR2 inhibitor, in combination with immune checkpoint therapy for advanced MSS CRC and other solid tumors (Rolfo et al., 2024). By blocking the CXCR2 pathway, these antagonists prevent the mobilization of neutrophils from the peripheral blood into the tumor, disrupt direct neutrophil-tumor cell contact, and attenuate tumor progression, representing a highly promising strategy to reprogram the myeloid tumor microenvironment (Chen et al., 2024).

Perineural invasion (PNI) seems to have not only an impact on the immunological arm of the tumor microenvironment, but also to be associated with the microbiome. Studies have revealed how dysbiosis of the gut microbiome can lead to senescence-associated secretory phenotypes (SASPs) that can have a causative effect and promote both inflammation and PNI. In fact, dysbiosis of the microbiota in CRC can impact cancer progression also through the modulation of the vagal and orthosympathetic tones acting through the gut-brain axis, which in turn can have an effect on the immunological control, immune checkpoint therapy response, and tumor microenvironment (Bautista et al., 2026).

Beyond the immediate tumor microenvironment, the bidirectional communication between the gut microbiota and the nervous system—defined as the gut-brain axis—adds a highly novel layer to the conceptual framework of CRC progression (Taleb et al., 2021). The gut microbiome is fundamentally involved in regulating the metabolism of critical neurotransmitters such as serotonin, dopamine, and gamma-Aminobutyric acid (GABA) (Y. Wang et al., 2023). In malignant conditions, an abnormal modulation of this axis leads to neuroendocrine-immune reactions wherein tumor cells are stimulated by neuroactive molecules secreted via sympathetic and vagal pathways (Liu et al., 2022). This dynamic is particularly evident in tumours having perineural invasion (PNI), which as mentioned before is a high-risk pathological feature characterized by tumor cells invading the nerve sheath (J. Wang et al., 2024). Nerve cells entwined within the tumor secrete neuropeptides and neurotransmitters that not only drive tumor cell proliferation and migration but also profoundly alter local immune responses (Liu et al., 2022). Consequently, long-term microbial dysbiosis can transmit dysregulated endocrine and neurocrine signals via vagal afferents, fostering a highly immunosuppressive milieu that supports rapid disease progression, therapy resistance, and enhanced perineural tracking (Y. Wang et al., 2023; J. Wang et al., 2024).

When these different components are synthesised into a unified description for right-sided and left-sided colorectal cancer, clear regional profiles can be identified. Right-sided colon cancer originates from the embryological midgut and is supplied by the superior mesenteric artery. In terms of the microbiome, it is characterized by an enrichment of pathobionts like *Fusobacterium nucleatum*, enterotoxigenic *Bacteroides fragilis*, and *pks*-positive *Escherichia coli*, which form dense bacterial biofilms. The microbial metabolites in right-sided tumors show high short-chain fatty acid variations and a prominent iron-dependent upregulation of CCL8. The tumor-associated neutrophils in this region are often immature and highly immunosuppressive, showing high NET formation. Arginase-1 status is typically high, causing severe local L-arginine starvation. This depletion of arginine can suppress local T-cell responses, contributing to a clinical scenario where immune checkpoint therapy may face local resistance unless these pathways are targeted. Pathologically, right-sided cancers show a high association with poor differentiation and severe perineural invasion, often leading to resistance to traditional neoadjuvant therapy.

In contrast, left-sided colorectal cancer arises from the embryological hindgut and is supplied by the inferior mesenteric artery. Its microbiome shows a lower biofilm load and contains more invasive planktonic bacteria. The metabolic profile shifts toward altered secondary bile acids rather than carbohydrate fermentation. The tumor-associated neutrophils in left-sided lesions show a more mature state with variable cytotoxic or anti-tumor functions, and Arginase-1 expression is usually moderate or limited to distinct focal boundaries. Pathologically, left-sided tumors show a higher frequency of intravascular invasion and orderly lymph node tracking patterns. These biological features make left-sided tumors highly responsive to anti-EGFR therapies, while their chromosomal instability profile and lower mutational load make them less ideal candidates for immune checkpoint therapy.

Conclusion

In recent years new research has revealed that there is an ever growing need to leave behind the umbrella term CRC and shift towards a more focused terminology, due to factors such as the huge differences from right-sided colon cancers, left-sided colon cancers and rectal cancer. CRC as an umbrella term is too generic. Understanding the immune components of the tumor microenvironment, the advanced heterogeneity of TANs, the metabolic activity of Arginase-1, and how the gut microbiota can impact the etiopathogenesis and progression of these types of cancers across distinct embryological and vascular segments are fundamental steps in order to develop more precise and useful clinical tools to be used in the management, diagnosis and treatment of these pathologies.

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