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GUT MICROBIOME AND COLORECTAL CANCER: MECHANISMS, BIOMARKERS, AND THERAPEUTIC IMPLICATIONS (A NARRATIVE REVIEW)

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Abstract

Background: *Colorectal cancer (CRC) remains a major cause of cancer morbidity and mortality worldwide. Emerging evidence implicates the gut microbiome as a modulator of CRC initiation, progression, and therapeutic response.*

Objective: *We performed a narrative review synthesizing recent mechanistic, diagnostic, and therapeutic literature on the gut microbiome–CRC axis, and highlight actionable research and clinical translation opportunities.*

Methods: *We conducted a focused narrative synthesis of primary research, reviews, and translational studies published between 2018–2025 across PubMed, Web of Science, and major journals.*

Results: *Evidence supports both tumor-promoting and tumor-protective roles of the microbiome. F. nucleatum consistently associates with CRC tissue and poorer outcomes through mechanisms including activation of Wnt/β-catenin signaling, immune suppression, and modulation of treatment response.*

Conclusions: *The gut microbiome is a compelling target for CRC prevention, diagnosis, and therapeutic optimization. Standardization of sampling, mechanistic human studies, and well-powered clinical trials are needed before routine clinical translation.*

INTRODUCTION

Colorectal cancer (CRC) remains among the leading causes of cancer-related death globally. The gut microbiome has emerged as a critical factor influencing carcinogenesis through immune modulation and metabolic reprogramming.

Methods

This narrative review prioritized recent high-quality studies (2018–2025) focusing on CRC–microbiome interactions.

Microbial signatures associated with CRC

CRC patients show enrichment of *Fusobacterium* and Bacteroides species and depletion of SCFA-producing commensals like Faecalibacterium prausnitzii.

Mechanisms linking microbiota to colorectal carcinogenesis

Mechanisms include genotoxicity (colibactin), inflammation (IL-6, TNF-α), adhesion/invasion (FadA), and metabolic reprogramming (secondary bile acids, SCFAs).

Fusobacterium nucleatum in CRC

F. *nucleatum* adheres via FadA, activates Wnt signaling, suppresses immunity, and correlates with chemoresistance.

Diagnostics and prognostics

Stool microbiome markers (e.g., F. *nucleatum* PCR) augment fecal immunochemical testing and predict survival outcomes.

Therapeutic strategies

Approaches include diet modification, probiotics, antibiotics, phage therapy, FMT, and microbiome-informed adjuvants.

Challenges and limitations

Heterogeneity in study design, sampling, and interindividual variability limits reproducibility and causality.

Conclusions

The gut microbiome offers promising pathways for CRC prevention and therapy, warranting further translational and clinical trials.

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