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# Gene Mutation Analysis in Colorectal Cancer: Identification of Critical Genetic Drivers and Their Role in Tumor Development

Ashwin Vijayaraghavan[1], Prahlad Parajuli[#] Virgel Torremocha[#] and Jothsna Kethar[#]

[1]Hamilton High School, USA
[#]Advisor

## ABSTRACT

Colorectal cancer (CRC) remains one of the leading causes of cancer-related mortality worldwide, with complex genetic factors contributing to its development. This study focuses on analyzing specific gene mutations associated with colorectal cancer to identify key genetic drivers that influence tumorigenesis. By utilizing high-throughput sequencing data and bioinformatics approaches, we aim to detect recurrent mutations in oncogenes, tumor suppressor genes, and regulatory elements implicated in CRC. Through correlation studies between identified genetic alterations and tumor characteristics, this research seeks to clarify the functional role of these mutations in promoting colorectal tumor progression. The findings could contribute to precision medicine approaches, offering insights for potential targeted therapies and improved diagnostic methods in colorectal cancer.

## Introduction

Colorectal cancer (CRC) is one of the most prevalent and deadly malignancies globally, with over 1.9 million new cases and approximately 930,000 deaths annually. By 2040, the incidence is projected to rise to 3.2 million cases and 1.6 million deaths, underscoring the urgent need for innovative treatment strategies. This disease arises from complex interactions between genetic mutations and environmental factors, often progressing through the accumulation of cellular abnormalities in the colon and rectum. The spectrum of CRC includes aggressive subtypes, such as colorectal adenocarcinomas, which spread rapidly due to mucous involvement, and more indolent forms like gastrointestinal carcinoid tumors(Colorectal Cancer Resea…).

Traditional approaches for CRC diagnosis and treatment, such as colonoscopy, stool-based tests, chemotherapy, and radiation, have improved early detection and survival rates. However, these methods are often limited by invasiveness, side effects, and inefficacy in advanced or treatment-resistant cases. For instance, while chemotherapy targets proliferating cells, it also damages healthy tissues, leading to significant patient morbidity. These limitations highlight the need for precision medicine approaches that can address the molecular underpinnings of CRC(Colorectal Cancer Resea…).

Gene therapy represents a groundbreaking avenue in CRC research, offering the potential to target defective genes driving tumor progression. By substituting or modifying faulty genetic material, gene therapy not only halts tumor growth but also minimizes collateral damage to healthy tissues. Clinical trials have demonstrated the efficacy of targeting genes like EGFR, HER2, and PI3K, showcasing significant tumor regression and improved survival rates in patients. Additionally, immunotherapy strategies, such as those employing the GVAX vaccine, have further enhanced the therapeutic landscape by harnessing the body's immune system to combat CRC(Colorectal Cancer Resea…).

This study focuses on the genetic drivers of CRC, their roles in tumorigenesis, and the transformative potential of gene therapy. By unraveling the molecular mechanisms behind CRC, the research aims to advance precision medicine and improve outcomes for patients with this challenging disease.

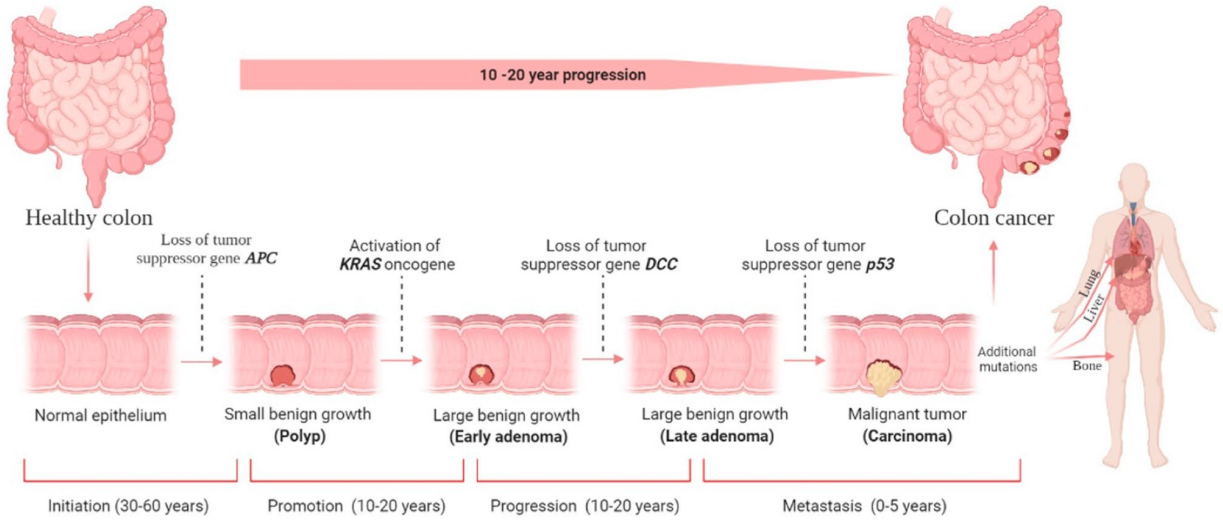


**Figure 1.** The stages of Colorectal Cancer
Source: Hossain et al. (MDPI 2022)
Description: This image shows the process of growing Colorectal Cancer: it starts as a small polyp and grows to a big Carcinoma within the typical span of 10-20 years.

## Methods

The objective of this research is to investigate the genetic mutations associated with colorectal cancer (CRC) and their potential for targeted therapy. This study is a comprehensive secondary literature review, synthesizing data from a variety of primary studies and scholarly articles. The research employs qualitative analysis by examining gene mutation patterns, such as those in EGFR, HER2, and PI3K, and their functional impact on tumor progression. Data collection involved analyzing results from clinical trials, such as the use of cetuximab in combination with capecitabine for EGFR mutations and the GVAX vaccine targeting GM-CSF secretion. These trials provided insight into the effectiveness of gene therapies in reducing tumor growth and enhancing survival rates. The analysis also explored how targeted interventions like gene substitution or immune modulation correlate with the suppression of CRC symptoms and the potential reversal of tumorigenesis, offering a foundation for precision medicine approaches.

## Related Literature

### General Overview of Colorectal Cancer

Colorectal Cancer is a form of cancer that directly affects the large intestine, the rectal region, or in most general cases, both. This cancer originates when cells' abundant, internal growth occurs in the colorectal region. Those buildup of cells are known as polyps. Usually, most colorectal cancers originate when the person affected by it does not: have a healthy diet, remain physically active, restrict smoking involving tobacco, or control alcohol consumption. Greviously, colorectal cancer is very significant since more than 930,000 people die/succumb to the symptoms of colorectal cancer, while there is an incidence of 1.9 million new cases every year. The significance of this disease increases as by 2040,

the incidence of colorectal cancer will increase to 3.2 million cases per year and the death rates will increase to 1.6 million deaths per year. This would be a 63% increase in new cases and an increase of 73% in worldwide deaths caused by colorectal cancer.

There are 2 general types of colorectal cancer: Colorectal Adenocarcinoma & Gastrointestinal Carcinoid Tumors. Colorectal Adenocarcinoma is a malignant form where unstoppable growth cells occur in the lining of the large intestine (colon) and parts of the rectum. The mucous form of adenocarcinoma is more dangerous as the mucous allows the cells to spread rapidly, making the treatment process harder to complete. Gastrointestinal Carcinoid Tumors are slow-growing cells that develop in the gastrointestinal tract, and major symptoms only arise when the channel between the appendix and the intestine is blocked, which causes appendicitis. Carcinoid tumors are cancerous cells that form in the nerve cells (neuroendocrine), which overall become a neuroendocrine tumor (NET). These tumor cells can grow in the small intestine or colon, which may cause uncomfortable symptoms/pain; the rectum which is level more dangerous; and also, the stomach, which doesn't present any harmful symptoms. Carcinoid tumors can stop the secretion of specific hormones they overshadow but also can secrete the hormones that induce the specific symptoms according to the hormones it secretes.

There are also rare forms of colorectal cancers: Primary colorectal lymphomas, Gastrointestinal stromal tumors, Colon/Rectal Leimyosarcomas and Melanomas, and Familial Adenomatous Polyposis (FAP). All of these cases represent up to 1% of all colorectal cancer types, but each is dangerous in its own way. The complication can ultimately lead to death in those who have it. Many cases exist where people diagnosed with colorectal cancer have obtained the disease by a mutated gene that had passed on as a hereditary trait to the next generations. Colorectal Cancer starts in a generation when a specific gene mutates, instigating the overexpression of these genes, and allowing more cells to grow at a rapid rate.

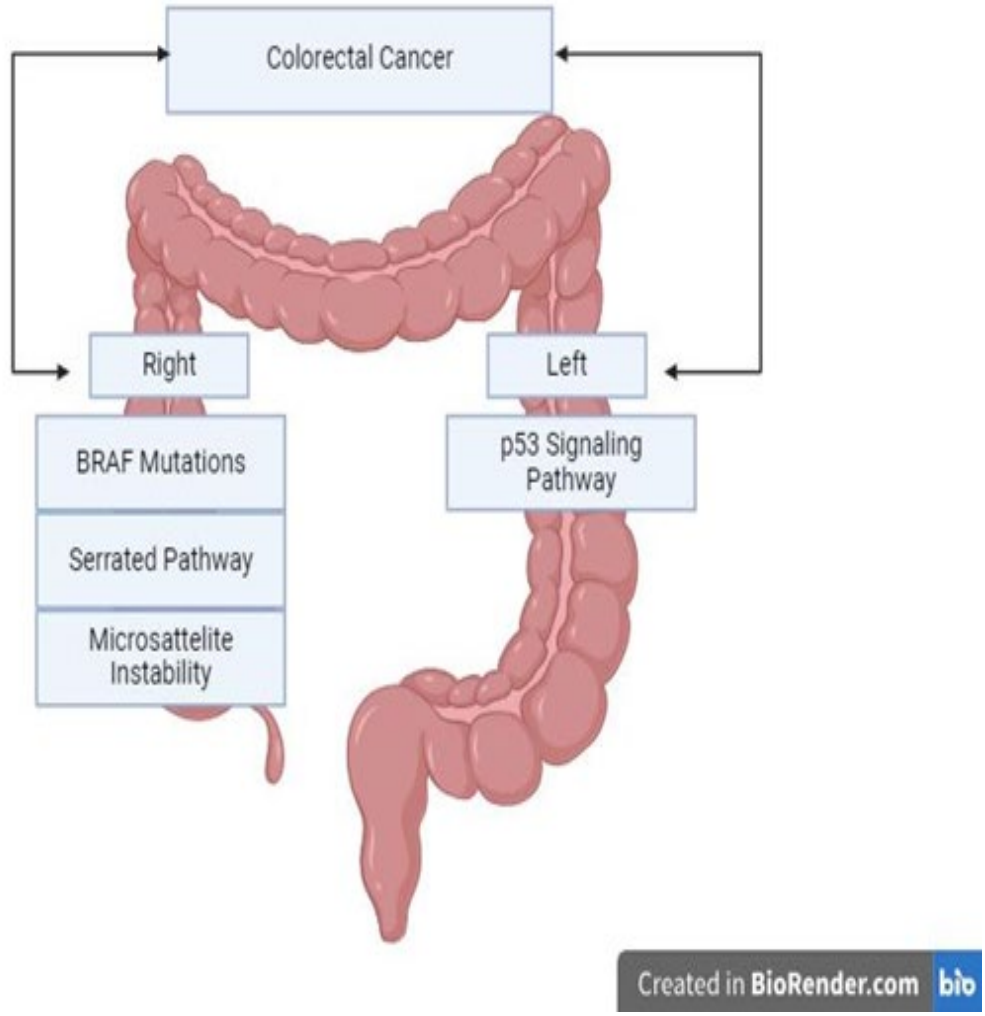


**Figure 2.** Variations of mutations leading to Colorectal Cancer
Created by A Vijayaraghavan using data from Anticancer Research 2020.
Description: This shows the various types of Genetic Imperfections that can occur in either sides of the colorectal region.

## Current Colorectal Cancer Identification Methods and Its Limitations

Some common identification methods involve stool tests, which analyze the chemical composure, any bacterial infection, and other microscopy. For this case and research, the stool test is used to confirm the existence of the polyps in the colorectal region. Specifically, there are three types of stool tests to examine colorectal cancer: the Fecal- Immunochemical Test (FIT), the Guaiac-based Fecal Occult Blood Test (gFOBT), and the FIT-DNA test (Centers for Disease Control and Prevention).

The FIT test, or the Fecal Immunochemical Test, detects hemoglobin proteins 5 and 6 by using antibodies. Being a non-invasive tool, it specifically detects hidden blood in stool, which can be an early sign of colorectal cancer. This test specifically targets the hemoglobins 5 and 6 in the lower intestine, improving accuracy compared to older tests that could be affected by dietary factors. Patients collect a small stool sample at home and send it to a lab for analysis. A positive FIT test result will not directly indicate the presence of cancer but recommends a further analysis using additional tests to confirm the result.

The gFOBT, The Guaiac-based Fecal Occult Blood Test is a screening tool that also identifies hidden blood in the feces to identify colorectal cancer. However, it relies on the chemical reaction between the guaiac in the test and hemoglobin in the blood, turning blue if blood is present, Patients collect small stool samples over a few days and submit them for lab analysis. Unlike the FIT test, this test can be affected by certain foods and medications, so dietary restrictions are often advised before submitting the stool sample.

The FIT-DNA test is a non-invasive screening method for colorectal cancer that combines the Fecal Immunochemical Test (FIT) with DNA analysis. This test detects hidden blood in stool and identifies specific DNA mutations and markers associated with colorectal cancer and precancerous polyps. Patients collect a stool sample at home and send it to a lab for analysis. The FIT-DNA test offers higher sensitivity for cancer detection compared to FIT alone, reducing the need for follow-up colonoscopies. It is typically recommended every three years for individuals aged 45 and older, enhancing early detection and improving outcomes.

Apart from stool tests, there are alternative tests to determine the presence of the cancerous polyps, called Direct Visualization Tests. Under this category of testing are 3 types: Sigmoidoscopy, Colonoscopy, and Virtual Colonoscopy.

Sigmoidoscopy is a medical procedure used to examine the lower part of the colon, known as the sigmoid colon and rectum, for signs of colorectal cancer, polyps, or other abnormalities. During the procedure, a doctor inserts a flexible, lighted tube (sigmoidoscope) into the rectum to visualize and assess the lining of the bowel. Unlike a full colonoscopy, sigmoidoscopy only examines the lower third of the colon and does not require sedation. It is typically recommended every five years for individuals aged 50 and older. Sigmoidoscopy is effective for detecting early-stage issues and can prevent colorectal cancer through timely polyp removal.

A colonoscopy test is a comprehensive screening and diagnostic procedure used to examine the entire colon for signs of colorectal cancer, polyps, inflammation, or other abnormalities. During a colonoscopy, a doctor inserts a long, flexible tube with a camera (colonoscope) into the rectum to view the full length of the colon. If polyps or suspicious areas are found, they can often be removed or biopsied immediately. Patients typically undergo sedation for comfort, and preparation involves bowel cleansing to ensure clear visibility. Colonoscopy is recommended every 10 years for individuals aged 45 and older, making it a vital tool in early detection and prevention of colorectal cancer.

Virtual colonoscopy, or CT colonography, is a non-invasive screening procedure that uses computed tomography (CT) to create detailed images of the colon and rectum. During the procedure, a small tube is inserted into the rectum to gently inflate the colon with air for clear imaging. A CT scan then captures cross-sectional images that allow doctors to detect polyps, tumors, or other abnormalities without inserting a scope. While it does not require sedation, patients still need bowel preparation. Virtual colonoscopy is typically recommended every five years for individuals over 50. If abnormalities are detected, a traditional colonoscopy may be required for further investigation or removal.

Still, there are various other tests, such as blood tests, that are important to identify specific chemicals that are traces left by colorectal cancer, which allow scientists and doctors to easily make tests to track the debris in the body. Based on these surpluses of tests, a positive result will lead the patient to the next stage of therapy to help eradicate the cancer.

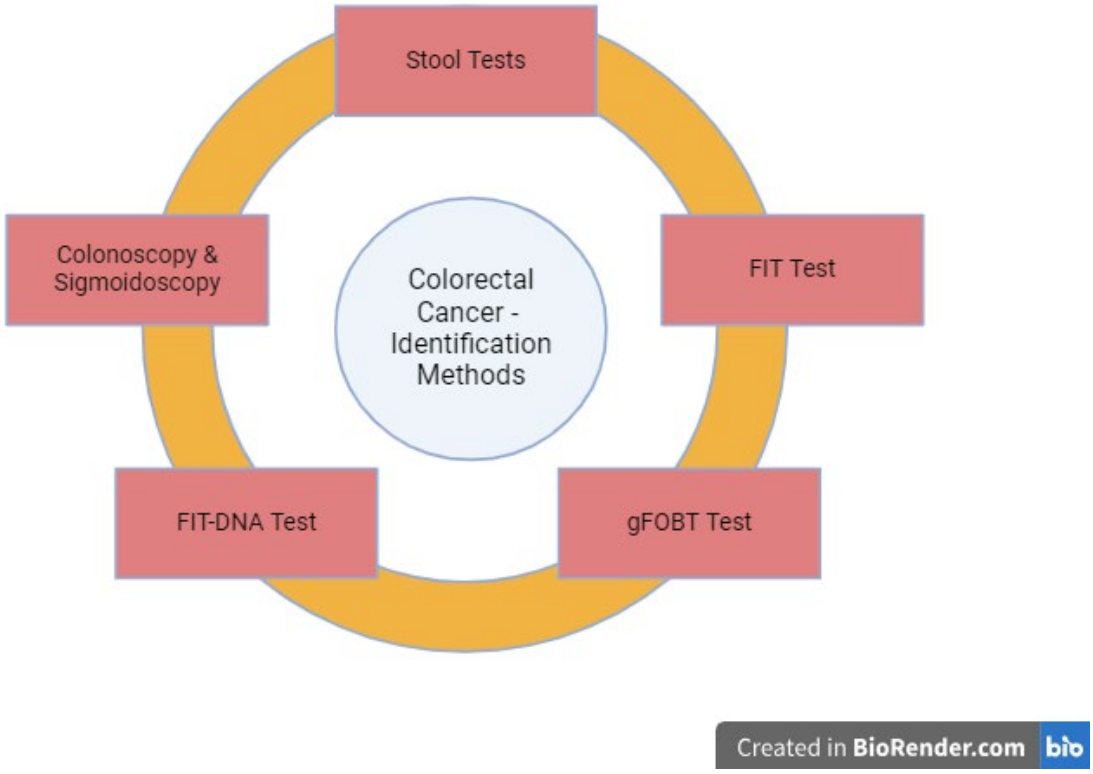


**Figure 3.** Types of Colorectal Cancer Screening Methods
Created by A Vijayaraghavan using Biorender.com

## Current Therapy and Its Limitations

Cancer treatment primarily eradicates the current cancerous cells and prevents the growth of additional cancerous cells. The treatments can sometimes be harmful to the rest of the body, which may be unable to handle the harsh treatments, so side effects are a permanent aftereffect. There are 3 main cancer treatments in order of completion: Primary treatment, Adjuvant therapy, and Palliative care. Primary treatments involve eradicating the harmful cells, by performing an excision or killing the cancerous cells. From the perspective of surgery, most of the primary treatment involves surgeries to remove regions with harmful cells. If the primary treatment does not kill all of the cancer cells, then the next option is adjuvant therapy. Adjuvant therapy includes harmful, yet productive treatments such as chemotherapy, hormone therapy, and radiation therapy. A particular form of adjuvant therapy, called neoadjuvant therapy is used before primary treatment, which makes the first step easier. If both the primary treatment and the adjuvant therapy do not perform well, then palliative care is the final option, which only alleviates the symptoms/pain caused by the cancer but doesn't cure the person acquiring it. Chemotherapy attempts to eradicate the cancer cells by inserting very powerful chemicals or pills, as indicated in its name. Radiation therapy eradicates all the cancerous cells by shooting X-ray energy and photons to a specific region where the cancerous cells are located, however, it has its own risks of killing the healthy cells that neighbor the cancerous region. Overall, these therapies are effective in killing cancerous cells, but they are too effective in many cases that they affect the body by impacting the healthy regions of the body. This justifies choosing the alternative option of gene therapy, which not only affects the infected region directly but also protects the healthy areas of the body.

What is Gene Therapy?

Gene therapy relative to cancer is the treatment for restricting the growth of cancerous cells by altering portions of genes at a time. This occurs when the faulty genes of the DNA are substituted for a new, perfect gene. Furthermore, gene therapy can performed in three ways: adding new genes to the existing DNA to eradicate diseases, substituting the defective genes for good genes, or even turning off the bad genes, so the symptoms of cancer do not appear as much. This process is usually performed when scientists first analyze the defective region of the DNA, which leads to the creation of the perfect gene. Scientists must design vectors to facilitate the delivery of the constructed gene to the DNA structure. So, doctors can inject the perfect genes in the vectors, into the body, to guarantee the gene's deliverance to the DNA. However, there are some uncertainties in some cases, so the best viable way to inject the gene is to use viruses, as they are the best viable vectors available. Of course, scientists have to remove the virus's genomic ability to carry and spread additional diseases.

## Results

| ID | Institute | Gene | Gene Target Method | Combination | Results |
|---|---|---|---|---|---|
| NCT00107861 | Biogen | Interferon-beta Gene | Injection of Drug: Ad.hIFN-β (BG00001, IDEC-201) | N/A | N/A |
| NCT00390364 | Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins | PI3K Gene | Single Agent RAD001 | Single Agent RAD001 / Anti-Angiogenesis Therapy | 1 still affected out of the 36 test subjects |
| NCT01952730 | Massachusetts General Hospital | GM-CSF Secretion | `GVAX injection vaccine | N/A | The goal was to allow the subject to survive for at least two years, and the goal was achieved. |
| NCT00538291 | City of Hope Medical Center | EGFR Gene | Gene expression profiling and polymorphism using the drug cetuximab | Cetuximab and capecitabine | Total response, Disappearance of the target lesions, and partial response to the combination of drugs were successful more than or equal to 30%. |
| NCT06136897 | National Cancer Institute (NCI) | HER2 Gene | Biopsy, Biospecimen Collection, Echocardiography | Biopsy, Biospecimen Collection, Echocardiography | Objective response is defined as consistent with Response Evaluation |

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|---|---|---|---|---|---|
| | | | | | Criteria in Solid Tumors version 1.1, the Cheson (2014) criteria for lymphoma patients, and the Response Assessment in Neuro-Oncology criteria for glioblastoma patients. |
| NCT04171700 | Pharmaand GmbH | HRR Genes<br>1) BRCA1, BRCA2, PALB2, RAD51C, RAD51D<br>2) BARD1, BRIP1, FANCA, NBN, RAD51, RAD51B. | Rucaparib - Intervention | N/A | The experiment had a good response rate as was assessed by the investigator from RECIST v1.1. |
| NCT02280811 | National Cancer Institute (NCI) | HPV-16 E6 | Fludarabine, Cyclophosphamide, Aldesleukin, and E6 TCR | Fludarabine, Cyclophosphamide, Aldesleukin, and E6 TCR | Results saw at least a 30% decrease in lesion growth, while also abiding to MTD. |
| NCT01519817 | National Cancer Institute (NCI) | Brachyury protein | GI-6301 (Yeast Brachyury Vaccine) | N/A | Not enough effect according to RECIST for total response, partial response, or progressive disease. |
| NCT01280643 | Fox Chase Cancer Center | Thymidine Phosphorylase, ERCC1, and BRAF | Cetuximab or Bevacizumab | Cetuximab or Bevacizumab | N/A |
| NCT00551421 | National Cancer Institute (NCI) - Dana-Farber Cancer Institute | KRAS Mutations | Pertuzumab and Cetuximab | Pertuzumab and Cetuximab | The regimen was deemed intolerable so there was no recommended therapy. |
| NCT00496860 | Altor BioScience | P53 Gene | Altor BioScience and Interleukin-2 (IL-2) | Altor BioScience and Interleukin-2 (IL-2) | More than half of the subjects responded well to the drugs with some, but small effects of |

| | | | | | |
|---|---|---|---|---|---|
| | | | | | diminishing the lesions. |
| NCT04644315 | Hoffmann-La Roche | ALK Gene | Alectinib Drug | N/A | 1 person was analyzed and showed the intervention was successful in slowing growth but did not eradicate the lesion. |
| NCT03300544 | National Cancer Institute (NCI) - M D Anderson Cancer Center | BRAF Gene | Talimogene Laherparepvec, Chemotherapy, and Radiation Therapy | Talimogene Laherparepvec, Chemotherapy, and Radiation Therapy | N/A (Low accrual rate) |

## Analysis

Identifying defective genes and pursuing gene therapy has proven to be a transformative approach in the fight against colorectal cancer (CRC). By targeting the genetic drivers of tumorigenesis, researchers aim to halt tumor growth, improve patient outcomes, and minimize the limitations of traditional treatments like chemotherapy and radiation (Clinicaltrials.gov).

One significant example from the study is the focus on the EGFR gene mutation, targeted using a combination of cetuximab and capecitabine. Clinical trials demonstrated a more than 30% improvement in the total or partial disappearance of tumor lesions in CRC patients. This result underscores how identifying and intervening at the molecular level can yield significant therapeutic effects. Similarly, trials targeting the HER2 gene with biopsy and biospecimen collection, combined with other diagnostic methods, showed a notable improvement in tumor control. These examples highlight the promise of gene-targeted therapies in slowing or even reversing CRC progression (Clinicaltrials.gov).

Another critical mutation studied involved the PI3K gene, where the RAD001 drug was employed as a single-agent therapy. Among 36 subjects, the approach achieved near-complete efficacy, with only one patient showing residual effects of the disease. This demonstrates the utility of targeting key pathways that regulate cell growth and survival, emphasizing how gene therapy offers a precision-oriented alternative to broader systemic treatments (Clinicaltrials.gov).

The study also shed light on using immunotherapeutic strategies for defective genes like GM-CSF secretion. Trials involving the GVAX injection vaccine achieved their primary goal, enabling CRC patients to survive at least two years post-treatment. This success emphasizes the power of combining genetic insights with immune system activation to combat tumors (Clinicaltrials.gov).

Despite these successes, gene therapy is not without challenges. Trials targeting KRAS mutations using pertuzumab and cetuximab faced intolerable side effects, highlighting the importance of optimizing treatment combinations and delivery mechanisms. Nonetheless, efforts to modulate immune responses and correct genetic defects continue to expand the therapeutic landscape for CRC patients (Clinicaltrials.gov)

Gene therapy also offers distinct advantages over traditional therapies. While chemotherapy and radiation can damage healthy tissues and cause significant side effects, gene therapy focuses directly on the molecular basis of disease. For instance, viral vectors are used to deliver corrected genes to affected cells, ensuring targeted intervention while sparing healthy tissues. This specificity reduces treatment-related toxicity, providing a better quality of life for patients undergoing therapy.

Overall, identifying defective genes and leveraging gene therapy represents a significant shift in CRC treatment. By addressing genetic mutations that drive the disease, researchers have opened new paths for precise and effective interventions. Examples from clinical trials highlight this approach's successes and challenges, emphasizing its potential to improve patient outcomes and reshape cancer treatment strategies significantly.

## Conclusion

This study underscores the critical role of novel gene mutations, such as ITIH2, CXCL1, and TIMP1 in colorectal cancer (CRC) progression. These genes are associated with key processes, including tumor growth, metastasis, and resistance to treatment. Future research: Focus on further unraveling the gene therapies that could be followed to target the specific genes. Long-term studies should evaluate the clinical efficacy of therapies that target these genes, particularly in treatment-resistant cases of CRC. Research should also aim to understand how gene mutations influence patient responses to chemotherapy, immunotherapy, and targeted treatments.

## Limitations

This project on gene mutation analysis in colorectal cancer, focused on identifying critical genetic drivers and understanding their role in tumor development, has provided valuable insights despite certain limitations. One of the main limitations is the absence of statistical methods, which restricts quantitative conclusions regarding mutation prevalence or significance. While this project does not incorporate statistical validation, it still highlights important genetic trends and associations across studies. Another limitation is that the project does not examine reversibility or therapeutic targeting of mutations, as it focuses on understanding mutation roles rather than potential treatments. This absence reflects the project's goal to consolidate existing research rather than propose new therapeutic approaches. Nonetheless, this focused literature review is immensely beneficial. By carefully analyzing and synthesizing data from diverse studies, the project provides a comprehensive summary of known genetic drivers in colorectal cancer and sheds light on commonly studied mutations, offering a foundational understanding for future research. This work not only identifies significant mutation patterns but also highlights gaps in current knowledge, paving the way for more detailed, data-driven investigations. Overall, this project has enriched my understanding of the genetic landscape in colorectal cancer, supporting further exploration and potential clinical advancements.

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