



Review

Regulatory Roles of Noncoding RNAs in the Progression of Gastrointestinal Cancers and Health Disparities

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Abstract: Annually, more than a million individuals are diagnosed with gastrointestinal (GI) cancers worldwide. With the advancements in radio- and chemotherapy and surgery, the survival rates for GI cancer patients have improved in recent years. However, the prognosis for advanced-stage GI cancers remains poor. Site-specific GI cancers share a few common risk factors; however, they are largely distinct in their etiologies and descriptive epidemiologic profiles. A large number of mutations or copy number changes associated with carcinogenesis are commonly found in noncoding DNA regions, which transcribe several noncoding RNAs (ncRNAs) that are implicated to regulate cancer initiation, metastasis, and drug resistance. In this review, we summarize the regulatory functions of ncRNAs in GI cancer development, progression, chemoresistance, and health disparities. We also highlight the potential roles of ncRNAs as therapeutic targets and biomarkers, mainly focusing on their ethnicity-/race-specific prognostic value, and discuss the prospects of genome-wide association studies (GWAS) to investigate the contribution of ncRNAs in GI tumorigenesis.

Keywords: gastrointestinal cancers; noncoding RNAs; chemoresistance; biomarkers; therapeutic targets; disparities



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1. Introduction

Gastrointestinal (GI) cancers account for more than 26% (4.8 million new cases) of the incidence rates and approximately 35% (3.4 million deaths) of all cancer-related deaths worldwide [1]. These numbers are predicted to increase by more than 50%, respectively, by 2040 [2]. Cancers of the stomach (approximately 1.0 million new cases), liver (840,000 cases), esophagus (570,000 cases), pancreas (460,000 cases), and colorectum (1.8 million cases) form the principal malignant conditions of the GI tract, which costs billions of dollars for treatment annually [3] and are imposing major challenges to public health [4]. Though the survival rates have improved with the advancements in radio- and chemotherapy and surgery, the prognosis for advanced-stage GI cancers remains poor. Therefore, new screening methods and therapeutic targets are required to improve GI cancer patient survival. With the advancement in cancer genomics, it is evident that most of the mutations or copy number changes associated with carcinogenesis are largely found in noncoding DNA regions [5,6]. Originally referred to as the “junk DNA”, they are now known to transcribe several noncoding RNAs (ncRNAs), which are implicated to regulate cancer initiation, metastasis, and drug resistance [7–9]. Some ncRNAs, such as lncRNA H19, miR-29a, miR-29b, and miR-29c, act as oncogenes or tumor suppressors [10]; however, the exact function and mechanism of action for most ncRNAs are still unknown. Based on the structure,

ncRNAs are classified further to include microRNAs (miRNAs), small interfering RNAs (siRNAs), antisense RNAs (asRNAs), long noncoding RNAs (lncRNAs), and circular RNAs (circRNAs) [11]. In this review, we summarize the regulatory functions of ncRNAs in GI cancer development and progression, chemoresistance, and health disparities. Additionally, we highlight their potential as therapeutic targets or biomarkers and discuss the prospects of genome-wide association studies (GWAS) to investigate the contribution of ncRNAs in GI tumorigenesis.

2. ncRNA-Mediated Regulation of Cell Signaling Pathways in GI Cancer Progression

During cancer development and progression, ncRNAs have been reported to have key regulatory functions. They are known to dysregulate several signaling pathways to promote cell proliferation, differentiation, and epithelial–mesenchymal transition (EMT) in various cancer types. LncRNAs and miRNAs are the most extensively studied ncRNAs in cancer research [12]. The lncRNA is a RNA molecule with transcript length of more than 200 nt [13], whereas miRNAs are smaller in length, usually around 18–25 nt [14]. In addition, miRNAs interact with lncRNAs and many downstream target genes to control their expression. LncRNAs act as “sponges” and sequester the miRNAs, eventually silencing them and their regulatory cascades [15]. In this section, we focus on the commonly sighted pathways, namely phosphoinositide 3-kinase/protein kinase B (PI3K/AKT), Wnt/ β -catenin, transforming growth factor- β (TGF- β), nuclear factor kappa B (NF- κ B), Notch, Hippo, and Ras/Raf/Mitogen-activated protein kinase/extracellular-signal-regulated kinase (Raf/MEK/ERK), that are dysregulated by several ncRNAs to facilitate the progression of various GI cancers (Figure 1).

2.1. ncRNAs Regulate the PI3K/AKT Signaling Pathway

The PI3K/AKT signaling pathway plays a crucial role in the progression of GI cancers through various biological processes such as proliferation, metastasis, chemo- and radioresistance, or autophagy. In general, ncRNAs can regulate the PI3K/AKT signaling by directly targeting AKT in a few cases, while most of them target the negative regulators of this pathway. For example, in pancreatic ductal adenocarcinoma (PDAC), when upregulated in PDAC tissues and cells, miR-107 induces cell migration, invasion, and EMT by downregulating tumor suppressors *caveolin-1* and *PTEN* genes and by upregulating p-AKT [16]. However, certain miRNAs such as miR-34a and miR-125a-5p act as tumor suppressor miRNAs and induce apoptosis and reduce hepatocellular carcinoma (HCC) proliferation and metastasis [17]. Similarly, in Epstein–Barr-virus-associated gastric cancer (EBVaGC), miR-BART4-3p targets *AXL*-mediated PI3K/AKT activation to inhibit EMT in gastric cancer cells [18]. This pathway is also known to be associated with the M2 polarization of macrophages. Tumor-activated macrophages (TAMs) that are typically maintained in the more polarized M2 state have anti-inflammatory and tumor-promoting functions and are associated with pro-metastatic cancer phenotype [19]. Several miRNAs, such as miR-25-3p, miR-130b-3p, and miR-425-5p, are transferred by exosomes to colorectal cancer (CRC) cells via the CXCL12/CXCR4 axis to activate the PI3K/AKT signaling pathway to induce the M2 polarization of macrophages, which increases EMT and vascular endothelial growth factor (VEGF) secretion in CRC cells [20]. Additionally, lncRNA H19 regulates the PI3K/AKT pathway by functioning as a competing endogenous RNA and predicts poor prognosis in CRC patients [21]. This pathway is also known to be modulated by circular ncRNAs, such as circ_NRIP1, which increases colony formation, cell viability, migration, and invasion in esophageal cancer [22]. With the advancement in microarray and next-generation sequencing technologies, tools are available to display PI3K/AKT-reprogrammed ncRNA profiles. It would be interesting to integrate the ncRNA profiling and ncRNA target prediction tools to establish the link between PI3K, AKT, and ncRNAs to comprehensively understand the PI3K/AKT gene regulatory mechanisms, thereby exploring GI cancer pathogenesis and diagnosis in a personalized manner, as well as developing new therapeutic strategies.

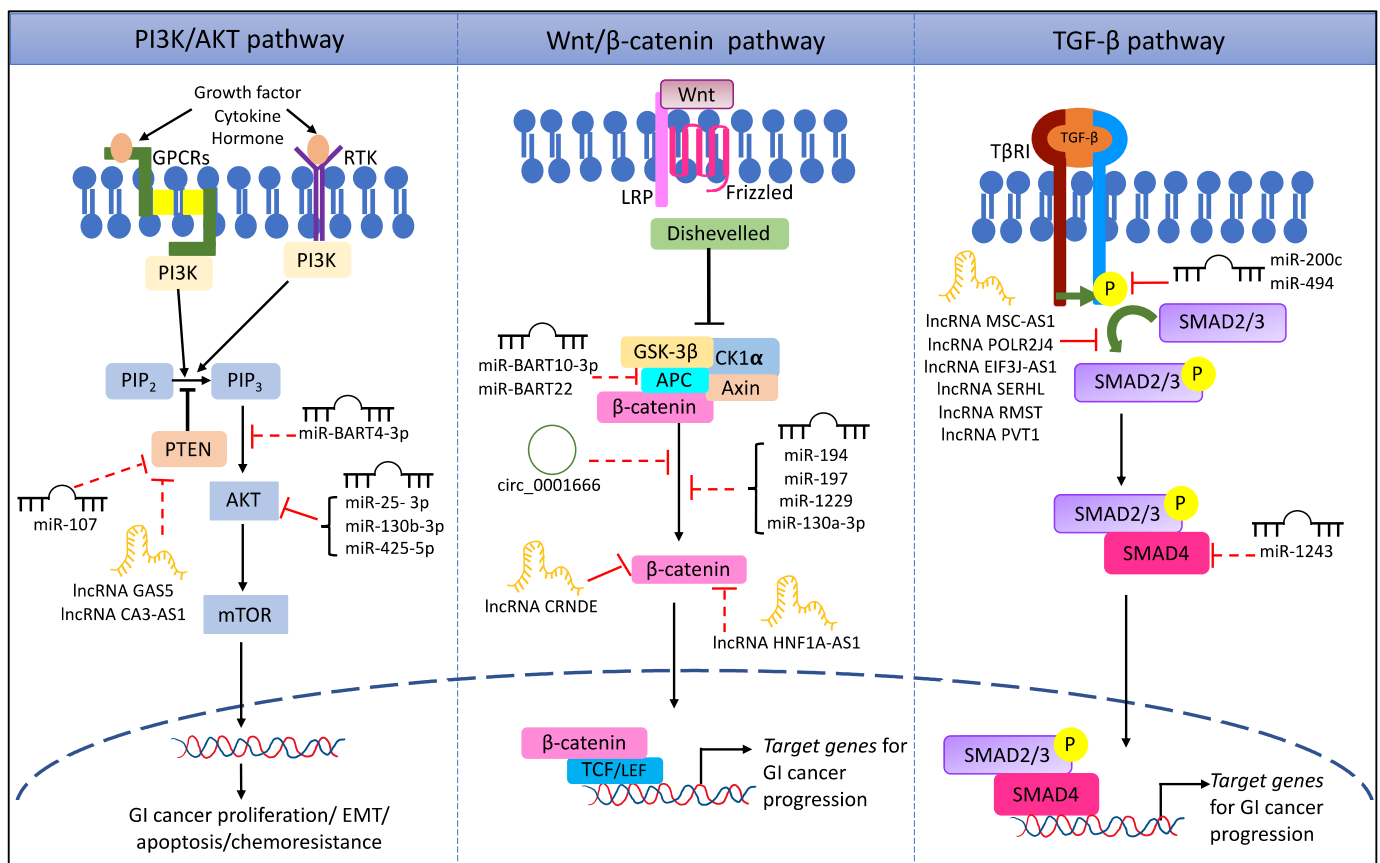


Figure 1. NcRNA-mediated regulation of signal transduction pathways in GI cancer progression. Representative illustration of different ncRNA-mediated dysregulation of A. PI3K/AKT, B. Wnt/ β -catenin, and C. TGF- β pathways. It should be noted that the ncRNA-mediated dysregulation of signal transduction pathways is not limited to the above pathways or ncRNAs; the red dotted line represents inhibition, and the red continuous frontal line represents activation by the respective ncRNA(s).

2.2. ncRNAs Regulate the Wnt/ β -Catenin Signaling Pathway

The Wnt/ β -catenin signaling pathway is another pathway that is highly modulated by ncRNAs to regulate cellular proliferation, differentiation, migration, genetic stability, and apoptosis in GI cancers. The aberrant activation of the Wnt pathway dysregulates the multifunctional protein β -catenin in GI cancers [23,24]. In addition, miRNA-20b and lncRNA NNT-AS1 are known to be upregulated in gastric cancer cells to promote cell proliferation, migration, and invasion via the Wnt signaling pathway [25,26]. In EBVaGC, miRNAs miR-BART10-3p and miR-BART22 play important roles in metastasis by activating the Wnt signaling pathway and targeting the adenomatous polyposis coli (*APC*) and Dickkopf-related protein 1 (*DKK1*) genes [27]. The Wnt/ β -Catenin signaling pathway is also dysregulated in hepatocellular carcinoma cells and tissues. The miRNAs miR-19a-3p/miR-376c-3p suppress the target gene *SOX6* to activate the Wnt/ β -catenin pathway [28]. These observations indicate that the differential regulation of the Wnt/ β -catenin pathway by specific ncRNAs plays an important role in GI cancer biology and could act as oncogenes. However, miR-194, miR-197, circ_0001666, miR-1229, and miR-130a-3p have been shown to inhibit the Wnt signaling pathway, preventing the progression of various GI cancers [29–32]. This trend of the up- and downregulation of the Wnt/ β -catenin pathway by specific ncRNAs is observed in GI cancers; however, further studies are required to understand the underlying mechanisms to promote an efficient therapeutic strategy by targeting ncRNAs associated with the Wnt pathway in GI cancers.

2.3. ncRNAs and the Other Signaling Pathways

Other pathways, including TGF- β , Hippo, MAPK, NF- κ B, Hedgehog, mTOR, and Raf/MEK/ERK pathways, are also found to be dysregulated by ncRNA during GI cancer progression; however, the number of studies is limited. The TGF- β signaling pathway is an evolutionarily conserved pathway that controls cell growth, differentiation, and development in various biological systems. In cancer cells, TGF- β performs a dual role via SMAD to either promote tumor suppression or inactivate the immune system to promote tumorigenesis that leads to changes in cell differentiation, causing epithelial–mesenchymal transition (EMT) [33]. A few miRNAs ($n = 17$) are known to be upregulated in hepatocellular carcinoma, of which miR-494 targets the SIRT3 and TGF- β /SMAD signaling pathways to promote cell proliferation and migration of hepatoma cells [34]. Similarly, miR-200c also targets the TGF- β 1/zinc-finger E-box-binding homeobox (ZEB1) pathway to induce EMT and promote cellular dissemination from the primary tumor and subsequent metastasis in CRC [35].

The dysregulation of the Hippo pathway, which controls cell growth, proliferation, and apoptosis, is associated with cancer development. In CRC, tumor suppressor miRNAs miR-30a-5p [36] and miR-375-3p [37] have been associated with the downregulation of the Hippo signaling pathway, limiting CRC proliferation, invasion, and migration. In the case of miR-375, it downregulates the yes1-associated transcriptional regulator (*YAP1*) gene, resulting in a reduced expression of connective tissue growth factor (*CTGF*), *cyclin D1*, and baculoviral inhibitor of apoptosis-repeat-containing 5 (*BIRC5*) target genes that are downstream of the Hippo–YAP1 pathway [37]. On the other hand, a circRNA hsa_circ_0128846 is seen to be upregulated in CRC tissues [38], which sponges miR-1184 to upregulate the Ajuba LIM protein (*AJUBA*) gene, which upregulates the Hippo–YAP1 pathway to promote CRC proliferation.

In pancreatic cancer, miR-143 acts as a tumor suppressor by targeting the transforming growth factor (TGF)- β -activated kinase 1 (TAK1) to inactivate the MAPK and NF- κ B pathways, which subsequently prevents cell proliferation and migration and induces apoptosis and G1/S arrest [39]. A similar phenomenon is observed in liver cancer cells, where miR-129-5p inhibits the MAPK pathway by targeting the calcium calmodulin-dependent protein kinase IV (*CAMK4*) to reduce tumor progression [40]. The mammalian target of the rapamycin (mTOR) signaling pathway is downregulated by the tumor suppressor miR-195, which limits the proliferation of ECC cells by targeting the major nonhistone chromosomal protein that controls cell cycle, transformation, proliferation, and apoptosis, the high-mobility group protein A2 (*HMG A2*) gene [41]. On the contrary, miR-132 and lncRNA AL139002.1 upregulate the Hedgehog and MEK/ERK signaling pathways in pancreatic and gastric cancer, respectively [42,43]. Specifically, miR-132 targets the sonic hedgehog (*Shh*) gene to induce the proliferation of pancreatic cells by reducing the expressions of Caspase-3 and Caspase-9, thus suppressing cell apoptosis [42]. The hepatitis A virus cellular receptor 1 (*HAVCR1*) gene is targeted by lncRNA AL139002.1 in gastric cancer cells to activate cell proliferation via the MEK/ERK signaling pathway [43].

These studies clearly indicate the roles of ncRNAs in regulating several biological pathways that contribute to the fate of tumor development. With the rapid progress in ncRNA and RNA biopharmaceutical research, ncRNA-targeted therapies could be considered a promising alternative to surgical methods, especially for advanced GI cancers, for which treatment options are currently limited.

3. Role of ncRNAs in Chemoresistance in GI Cancers

Chemotherapy, a treatment approach predominantly practiced for the annihilation of cancerous cells by obstructing cellular growth and division, includes a cocktail of drugs such as adriamycin, platinum-based drugs, 5-fluorouracil (5FU), vincristine, and paclitaxel [44]. However, the development of chemoresistance remains a challenge for patients receiving chemotherapy, preventing better recovery rates for GI cancer patients. Cancer patients exhibit intrinsic or acquired chemoresistance by a multistep process leading to

interference with the cellular function. Network analyses have revealed that mechanisms underlying the roles of ncRNA-mediated chemoresistance are highly complex. The abnormal expression of ncRNA promotes the manifestation of chemoresistance by inactivating apoptosis signaling pathways, hindering cell cycle checkpoints, increasing cell proliferation, autophagy, DNA damage repair, cancer stem cells (CSCs), and EMT [44]. The ncRNAs, mostly the miRNAs and lncRNAs, have a pivotal role in inducing chemoresistance in GI cancers. These two families of ncRNAs commonly tend to target the cell cycle and several different signaling pathways (MAPK/ERK, PI3K/AKT, Wnt/B-catenin, Hippo, NF- κ B, and Notch) to confer drug(s) resistance in GI cancers. Table 1 lists the different ncRNA(s) that contribute to drug resistance in different GI cancers, along with their known molecular targets mediating the drug/multidrug resistance. Interestingly, lncRNAs such as lncRNA CRNDE, GAS5, and HOTAIR are seen to contribute to 5FU, adriamycin, oxaliplatin, cisplatin, gemcitabine, and/or doxorubicin resistance in multiple GI cancers by targeting different molecules/pathways (Table 1). For example, lncRNA CRNDE contributes to 5FU, oxaliplatin, and adriamycin resistance in CRC, gastric cancer, and HCC by targeting β -catenin and *TCF4*, *PICALM*, and *CELF2* and *LATS2*, respectively [24,45,46]. Conversely, multiple ncRNAs lncRNA CRNDE, lncRNA PCAT6, lncRNA SNHG6, miR-125b, miR-26a-5p, and miR-532-3p contribute to 5FU resistance in CRC by targeting β -catenin and *TCF4*, *HMG2*, *ULK1*, *APC*, *ULK1*, *ETS1*, and *TGM2* genes, respectively [24,47–49]. From these studies, it is clear that ncRNAs do have an apparent impact on modulating CRC chemoresistance in GI cancers. However, the number of studies conducted is limited, and we still lack a clear understanding of the mechanisms that regulate ncRNA-based chemoresistance in different GI cancers. It is crucial to further identify different ncRNAs and their upstream or downstream mediators to overcome the limitations of ineffective chemotherapy, relapse, and mortality in GI cancer patients.

Table 1. ncRNA-mediated chemoresistance targets in GI cancers.

ncRNA	GI Cancer Type	Expression	Drug(s)	Molecular Target(s)	Reference
lncRNA CRNDE	Colorectal cancer	Upregulated	5FU	β -catenin and <i>TCF4</i>	[24]
lncRNA PCAT6	Colorectal cancer	Upregulated	5FU	<i>HMG2</i>	[49]
lncRNA SNHG6	Colorectal cancer	Upregulated	5FU	<i>ULK1</i>	[48]
miR-125b	Colorectal cancer	Downregulated	5FU	<i>APC</i>	[47]
miR-26a-5p	Colorectal cancer	Downregulated	5FU	<i>ULK1</i>	[48]
miR-328	Colorectal cancer	Upregulated	5FU and HCPT	<i>ABCG2</i>	[47]
miR-532-3p	Colorectal cancer	Upregulated	5FU, cisplatin	<i>ETS1</i> and <i>TGM2</i>	[47]
lncRNA HOTAIR	Colorectal cancer	Upregulated	cisplatin	β -catenin, <i>GRG5</i>	[23]
lncRNA CACS15	Colorectal cancer	Upregulated	oxaliplatin	<i>ABCC1</i>	[50]
lncRNA LINC00525	Colorectal cancer	Upregulated	oxaliplatin	<i>ELK3</i>	[51]
miR-128-3p	Colorectal cancer	Upregulated	oxaliplatin	<i>Bmi</i> and <i>MRP5</i>	[47]
lncRNA GIHCG	Colorectal cancer	Upregulated	oxaliplatin and 5FU	unknown	[52]
lncRNA SCARNA2	Colorectal cancer	Upregulated	oxaliplatin and 5FU	<i>WGFR</i> and <i>BCL-2</i>	[53]
miR-451	Colorectal cancer	Upregulated	SN38	<i>ABC1</i>	[47]
miR-514b-3p	Colorectal cancer	Upregulated	cisplatin and irinotecan	<i>FZD4</i> , <i>NTN1</i>	[47]
miR-514b-5p	Colorectal cancer	Downregulated	cisplatin and irinotecan	<i>CDH1</i> , <i>CLDN1</i>	[47]

Table 1. Cont.

ncRNA	GI Cancer Type	Expression	Drug(s)	Molecular Target(s)	Reference
miR-138	Esophageal cancer	Downregulated	5FU and cisplatin	<i>Survivin</i>	[54]
lncRNA HOTAIR	Esophageal cancer	Upregulated	5FU	<i>MTHFR</i>	[55]
miR-221	Esophageal cancer	Downregulated	5FU	<i>DKK2</i>	[47]
miR-29c	Esophageal cancer	Downregulated	5FU	<i>FBXO3I</i>	[56]
miR-338-5p	Esophageal cancer	Downregulated	5FU	<i>ID-1</i>	[57]
lncRNA CCAT1	Esophageal cancer	Upregulated	cisplatin	<i>PLK1</i> and <i>BURB1</i>	[58]
lncRNA EMS	Esophageal cancer	Upregulated	cisplatin	<i>WTAP</i>	[59]
lncRNA LINC00337	Esophageal cancer	Upregulated	cisplatin	<i>TPX2</i>	[60]
lncRNA TUG1	Esophageal cancer	Upregulated	cisplatin	<i>NRF2</i>	[61]
miR-10b	Esophageal cancer	Downregulated	cisplatin	<i>PPARγ</i>	[62]
lncRNA ROR	Gastric cancer	Upregulated	adriamycin and vincristine	<i>MRP1</i>	[63]
lncRNA HCP5	Gastric cancer	Upregulated	oxaliplatin and 5FU	<i>AMPK</i> , <i>PGC1α</i> , <i>CEBPB</i>	[64]
lncRNA ARHGAP5-AS1	Gastric cancer	Upregulated	cisplatin, actinomycin, and 5FU	<i>METTL3</i>	[65]
lncRNA SNHG16	Hepatocarcinoma	Downregulated	5FU	unknown	[66]
lncRNA CRNDE	Hepatocarcinoma	Upregulated	adriamycin	<i>CEL2</i> and <i>LATS2</i>	[46]
lncRNA GAS5	Hepatocarcinoma	Upregulated	doxorubicin	<i>PTEN</i>	[67]
lncRNA HANR	Hepatocarcinoma	Upregulated	doxorubicin	<i>GSKIP</i>	[68]
lncRNA MALAT1	Hepatocarcinoma	Upregulated	sorafenib	<i>miR-140-5p</i>	[69]
lncRNA H19	Hepatocarcinoma	Downregulated	sorafenib or doxorubicin	<i>ELAVL1</i>	[70]
miR-138-5p	Pancreatic cancer	Upregulated	5FU	<i>Vimentin</i>	[47]
miR-27b	Pancreatic cancer	Upregulated	docetaxel	<i>ZEB1</i>	[47]
miR-34a	Pancreatic cancer	Upregulated	docetaxel	<i>ZEB1</i>	[47]
lncRNA SLC7A11-AS1	Pancreatic cancer	Upregulated	gemcitabine	<i>NRF2</i>	[71]
miR-1243	Pancreatic cancer	Upregulated	gemcitabine	<i>SMAD4</i>	[47]
miR-153	Pancreatic cancer	Upregulated	gemcitabine	<i>Snail</i>	[47]
miR-30a	Pancreatic cancer	Upregulated	gemcitabine	<i>Snail</i>	[47]
miR-34	Pancreatic cancer	Upregulated	gemcitabine	<i>Slug</i>	[47]
miR-509-5p	Pancreatic cancer	Upregulated	gemcitabine	<i>Vimentin</i>	[47]
lncRNA GAS5	Pancreatic cancer	Upregulated	gemcitabine and 5FU	<i>MST-1</i>	[72]

4. Genome-Wide Profiling of ncRNAs in GI Cancers

Genome-wide profiling of ncRNAs has garnered significant attention from researchers studying GI cancers due to their crucial role in transcriptional and post-transcriptional regulation. Novel miRNA-based signatures for the detection and prognosis of metastasis in GI cancers are established in many studies that show a promising future in understanding the development and treatment of patients. A genome-wide transcriptome profiling conducted by Shimura et al., in 2021, identified five miRNAs in the initial filtering phase,

but only three miRNAs (miR-30a-5p, -659-3p, and -3917) were significantly overexpressed in primary tumors from peritoneal metastasis in gastric cancer patients, making them potential miRNA signatures to identify peritoneal metastasis in gastric cancer patients [73]. In CRC progression, Kalmár et al. (2019) checked for lncRNA expression levels in colonic cancer biopsy samples and compared those with controls, which were further analyzed with Human Transcriptome Array (HTA). Sixteen lncRNAs were differentially expressed, including LINC02023, MEG8, AC092834.1, which were downregulated, and CCAT1 and CASC19 were upregulated [74]. Similar studies have also revealed the group of lncRNAs that promote liver metastasis in CRC patients [75]. A study involving the transcriptomic profiling of HCC tissues, performed via high-throughput RNA sequencing, found 214 differentially expressed lncRNAs, of which the expression of 4 lncRNAs (NONHSAT003823, NONHSAT056213, NONHSAT015386, and especially NONHSAT122051) is correlated with tumor cell proliferation, portal vein tumor thrombosis, and serum or tissue alpha-fetoprotein levels [76]. Further, genome-wide transcriptomic screening could also be performed from databases such as The Cancer Genome Atlas (TCGA). A large number of studies have identified ncRNAs from the TCGA database that are associated with a poor prognosis of patients with esophageal adenocarcinoma (EADC) and esophageal squamous cell carcinoma (ESCC) [77] or are associated with tumor size, N classification, clinical stage, or the risk of esophageal cancer recurrence [78,79]. Interestingly, the silencing of lncRNA-KIAA1244-2 identified in this study in esophageal cancer cells is known to inhibit cell proliferation and *TNFAIP3* expression; thus, it could be a potent therapeutic target for ESCC [79]. The TCGA database has also been analyzed to identify the expression profiles of lncRNAs in HCC tissues, among which two crucial lncRNAs termed “PVT1” and “SNHG7” were found to be involved in the recurrence of the tumor, and lncRNA unigene56159 was found to promote the migration and invasion of HCC cells through the miR-140-5p/SNAI2 axis, where it acted as a competing endogenous RNA (ceRNA) for miR-140-5p and down-regulated its expression [80]. Few genome-wide lncRNA-microarray studies have been performed with gastric cancer patients’ plasma and compared their results with healthy control plasma to identify lncRNA signatures, namely FAM49B-AS, GUSBP11, CTDHUT, TINCR, CCAT2, AOC4P, BANCR, and LINC00857, which are upregulated in the plasma from gastric cancer patients [81,82]. Implementing the screening of these lncRNAs from patient plasma for clinical diagnostic purposes would provide a promising noninvasive approach to detect gastric cancer in the future.

These studies conducted with different GI tumor samples have provided us with the dysregulated patterns of various lncRNAs; however, it is crucial to construct a ceRNA network to identify survival- and prognosis-related ncRNAs to understand GI cancer pathogenesis. In recent years, efforts have been made to develop ceRNA networks for correlating 77 lncRNAs [83], and 32 miRNAs with the overall survival and prognosis of HCC patients [84]. Despite the success of GWAS in the identification of hundreds of genetic ncRNAs associated with GI cancers, further comprehensive studies are required to provide more insights into the tumor progression and help develop screening tools for early GI cancer detection.

5. ncRNAs as Potential Therapeutic Targets or Biomarkers for GI Cancer Progression

In recent years, the study of the tumor microenvironment has played a crucial role in understanding the disease progression [85]. Current research shows a considerable amount of evidence of ncRNAs undergoing significant changes in GI cancer progression that can potentially be used as predictive biomarkers or prognostic measures. The ncRNAs open a wide array of scope for clinical advancement with diagnostic information retrieved with noninvasive, sensitive, and specific disease development detection. Advances in the prognostic prediction of gastrointestinal cancer using ncRNAs are rapidly progressing with recent advances, including using electrical biosensors as a promising alternative to developing fast and low-cost detection systems to quantify mRNA biomarkers [86]. Although new technologies and treatments are progressing toward increasing the survival

rates and treatments, the diagnosis of various types of cancers in patients is performed only when the disease has progressed to advanced stages. The aberrant expression of ncRNAs is associated with various cancers, and their ability to regulate the expression of various downstream target genes and their associated pathways has provided a rationale to pursue them for their untapped potential in early-stage biomarker and therapeutic drug development in GI cancers [87]. Preclinical studies have demonstrated the potential antitumor activity of synthetic miRNA- or lncRNA-based therapeutic molecules, with some showing promising results even in early-phase human clinical trials [88,89]. However, as the studies using ncRNA-based cancer therapeutics continue to evolve, there is a lot to unravel and understand about the precise molecular mechanisms and specific downstream therapeutic targets. Here, we summarized the identified lncRNAs and miRNAs involved in signaling networks and their potential as therapeutic targets or noninvasive alternatives for screening to traditional methods for GI cancers (Tables 2 and S1–S4).

Table 2. List of ncRNAs that could be used as therapeutic targets or biomarkers for colorectal cancer.

ncRNA	Expression	Clinical Significance	Molecular Target(s)	Reference
lncRNA TP73-AS1	Upregulated	Biomarker	<i>TGF-α</i>	[90]
lncRNA SH3PXD2A-AS1	Upregulated	Biomarker and therapeutic target	<i>P57</i> and <i>KLF2</i>	[91]
lncRNA RP1-85F18.6	Upregulated	Diagnostic or prognostic biomarker	cell proliferation, cell cycle progression, and apoptosis	[92]
lncRNA DLEU7-AS1	Upregulated	Prognostic biomarker	Wnt/ β -Catenin signaling pathway	[93]
lncRNA BANCR	Upregulated	Prognostic biomarker	unknown	[94]
lncRNA HNF1A-AS1	Upregulated	Prognostic biomarker	Wnt/ β -Catenin signaling pathway	[95]
lncRNA LUADT1	Upregulated	Prognostic biomarker	unknown	[96]
lncRNA PANDAR	Upregulated	Prognostic biomarker	unknown	[97]
lncRNA RP11-708H21.4	Downregulated	Prognostic biomarker	AKT/MTOR pathway	[98]
lncRNA SBDSP1	Upregulated	Prognostic biomarker	unknown	[99]
lncRNA SNHG6	Upregulated	Prognostic biomarker	cell proliferation, cell cycle progression, and apoptosis	[100]
lncRNA ZEB1-AS1	Upregulated	Prognostic biomarker	<i>P15</i> , <i>ZEB1</i>	[101]
lncRNA-RP11-317J10.2	Downregulated	Prognostic biomarker	<i>Cyclin D1</i>	[102]
lncRNAs (RP1-170O19.17, RP11-785D18.3, RP11-798K3.2, XXbac-B476C20.9, RP11-481J13.1, and RP11-167H9.4)	Upregulated	Prognostic biomarkers	cGMP-PKG signaling pathway and cAMP signaling pathway	[103]
lncRNA AB073614	Upregulated	Prognostic marker and therapeutic target	PI3K/AKT signaling pathway	[104]
lncRNA LINC00858	Upregulated	Prognostic marker and therapeutic target	miR-22-3p	[105]

Table 2. Cont.

ncRNA	Expression	Clinical Significance	Molecular Target(s)	Reference
lncRNA SNHG6	Upregulated	Prognostic marker and therapeutic target	<i>Upf1</i> and <i>ZEB1</i>	[106]
lncRNA CA3-AS1	Upregulated	Therapeutic target	miRNA-93/ <i>PTEN</i> Axis	[107]
lncRNA Linc00659	Upregulated	Therapeutic target	PI3K/ <i>AKT</i> signaling pathway	[108]
lncRNA-HOTAIR	Upregulated	Therapeutic target	<i>P21</i>	[109]

6. The Potential Role of ncRNAs to GI Cancer Disparities

Race and ethnicity have long been associated with GI cancer health disparities. CRC incidence and mortality are highest in African Americans (AAs), followed closely by American Indians and Alaska Natives, and lowest in Asians/Pacific Islanders (APIs). During 2012–2016, CRC incidence rates in AAs were about 20% higher than those in Caucasian Americans (CAs) and 50% higher than those in APIs. The disparity for mortality is twice that for incidence; CRC death rates in AAs are almost 40% higher than those in CAs and double those in APIs [110]. Therefore, research is critically needed to understand these disparities and develop interventions to close the gap.

Single nucleotide polymorphisms (SNPs) located in the miRNA functional regions are involved in GI cancer susceptibility, often in a race-specific manner. Over a decade ago, it was revealed in two CRC prognostic research studies that the effect of SNP rs4919510 in miR-608 varied by race [111]. In CAs, the homozygous-variant genotype, GG, is associated with a significant increase in the risk of death, and in AAs, a protective association between the GG genotype and survival was observed [112]. Several studies have been carried out in Chinese populations investigating the association between genetic variants located in miRNAs and GI cancer susceptibility. SNPs rs2839698 in long noncoding RNA (lncRNA) gene H19 and rs2682818 in miR-618 were found to be associated with an elevated CRC risk, while miR-196a2 rs11614913 T > C polymorphism was shown to reduce the esophageal cancer risk in the Chinese study participants [113–115]. SNPs in miR-host genes (MIR17HG and MIR155HG) contributed to CRC and liver cancer susceptibility in Han Chinese populations [116–118]. Esophageal cancer patients were found to express an immune-related prognostic enhancer RNA, lncRNA AC007255.1, and gastric cancer patients expressed aberrant ncRNAs (miRNA-936, miRNA-1306-3p, miRNA-3185, miRNA-6083, miRNA-659-3p, miRNA-6792-3p, lnc-ABCC5-2:1, lnc-MB21D1-3:5, and lnc-PSCA-4:2) in two recent studies from China [119,120]. One of the early studies to evaluate the prognostic value of miRNAs in CRC based on patient race/ethnicity demonstrated that miR-20a, miR-21, miR-106a, miR-181b, and miR-203 expressed two-fold higher in AA CRC patients than in their CA counterparts [121]. In 2016, a miR-1291-FOXA2-AGR2 signaling pathway was reported to control the suppression of pancreatic tumorigenesis in CA patients [122]. Oncogenic miRNAs (miR-17, miR-21, miR-182, miR-210, and miR-222) overexpressed in vitro in three newly established AA CRC lines, compared with the CA CRC lines [123]. When stratified by race (Asian and European), out of the 12 studies investigating polymorphisms in ncRNAs and susceptibility to CRC, only miR-146a rs2910164 was associated with a decreased risk of CRC in Europeans [124].

Several investigations have provided evidence supporting the role of the epigenetic regulation of miRNAs in racial cancer health disparities [125]. Hypermethylation of miR-9, miR-124, miR-137, miR-548, miR-663, miR-1207, miR-1279, miR-2682, miR-6130, and miR-182 was observed in AA CRC patients, while miR-34 was found to be hypermethylated in CA patients [126–128].

Some CRC racial disparities can be explained by differences in access to care, cancer screening, paucity of clinical data, and other socioeconomic factors [129,130]. However, reasons for ethnicity-based disparities are complex and remain even after adjustment for these

factors. Consequently, a review of recent advances in the understanding of ethnicity-specific factors, including genetic, epigenetic, and environmental factors, related to tumorigenesis is important for evaluating our progress toward eliminating the disparities.

7. Discussion

GI cancers are common, both in the United States and worldwide. Early detection is still crucial for an effective treatment, which is however challenging due to the invasive screening methods or limited access to health care facilities. Recent advances in high-throughput sequencing technologies have revealed critical information about a variety of ncRNAs. Numerous reports have been documented to demonstrate the role of ncRNAs in tumor initiation and progression. The aberrant expression of ncRNAs has been observed to accompany DNA damage, immune escape, and cellular metabolic disorders in various cancer types, making it an interesting area of research to understand the pathogenesis of cancer. In this review, we highlighted the function of ncRNAs in modulating various cell signaling pathways to induce GI cancer progression by epigenetic gene regulation, EMT, and development of drug resistance. However, it must be noted that, with the huge number of uncharacterized ncRNAs, the ncRNAs reviewed here are probably only a small proportion of the functionally relevant ncRNAs in GI cancer progression. Further, ncRNAs can be detected in plasma, have remarkably high tissue specificity, and are related to site-specific clinicopathological parameters including overall survival, recurrence, and metastasis; thus, they can be used as potential diagnostic and therapeutic markers in respective GI cancers. With the rapid development of gene-editing tools, the feasibility of CRISPR-Cas9-based ncRNA targeting in tumor cells is currently being explored [131,132]. However, the possibility of their off-target effects due to the low specificity of ncRNAs needs further validation. Other approaches such as combination therapy, with ncRNA-mediated targeted therapy using nanomedicine or immunotherapy, may be promising to treat GI cancers in the future. As early detection is still required to curb the spread of GI cancers and their efficient treatment, ncRNA-based screening tools could provide new noninvasive methods for GI cancer screening using patient blood/plasma samples. Finally, we noted the GI cancer health disparities and the predisposition of ncRNA in the respective ethnic groups. Conducting further studies to identify genetic markers in minority groups is crucial to reducing the mortality rate in these populations. In the United States, Hispanics are known to have a significantly higher incidence of GI cancers and worse cancer-related outcomes when compared with non-Hispanic white (NHW) patients. However, to the best of our knowledge, no ncRNA profiling studies have been conducted in this ethnic group. It is critical to study Hispanic GI cancers to identify potential ethnicity-specific biomarkers or targets for developing novel therapeutic interventions.

Taken together, ncRNA research has increased our understanding of the complexity of GI cancer progression and metastasis, although an understanding of their mechanistic function is only beginning to emerge. The major challenge remains the absence of appropriate therapeutic targets and detection systems for GI cancers, which could possibly be overcome by ncRNA-centered GI cancer research and their translation into clinical applications in the near future.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/cells11152448/s1>, Table S1: List of ncRNAs that could be used as therapeutic targets or biomarkers for gastric cancer [133–169]; Table S2: List of ncRNAs that could be used as therapeutic targets or biomarkers for pancreatic cancer [170–200]; Table S3: List of ncRNAs that could be used as therapeutic targets or biomarkers for liver cancer [201–250]; Table S4: List of ncRNAs that could be used as therapeutic targets or biomarkers for esophageal cancer [251–289].

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