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Expression of Wnt-Related Long Non-coding RNAs ITGB2-AS1, HNF1A- AS1, and Lnc-MAP6-1:3 in Patients with Colorectal Cancer

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Abstract

Background

Colorectal cancer (CRC) is among the most commonly diagnosed cancers globally and continues to be a major contributor to cancer-associated mortality. Aberrant activation of oncogenes and inactivation of tumor suppressor genes contribute to CRC development and are closely associated with dysregulation of long non-coding RNAs (lncRNAs). Recent evidence suggests that lncRNAs regulate tumor initiation and progression and may serve as valuable biomarkers for cancer diagnosis. This study investigated the expression profiles and diagnostic potential of three Wnt-related lncRNAs, ITGB2-AS1, HNF1A-AS1, and Lnc-MAP6-1:3, in colorectal cancer patients.

Materials and Methods

Thirty paired colorectal tumor tissues and matched adjacent non-tumorous tissues were collected from patients who underwent surgical resection. Total RNA was extracted using TRIzol™ reagent, and complementary DNA (cDNA) was synthesized using the ExcelRT™ Reverse Transcription Kit. Quantitative real-time PCR (qRT-PCR) was accomplished using RealQ Plus 2× Master Mix Green. Relative gene expression was analyzed using REST software, while statistical analyses were performed with SPSS. Receiver operating characteristic (ROC) curve analysis was used to evaluate the diagnostic performance of the investigated lncRNAs.

Results

qRT-PCR analysis demonstrated that ITGB2-AS1 and HNF1A-AS1 were significantly upregulated in colorectal tumor tissues compared with adjacent normal tissues ($P < 0.05$). Expression levels of ITGB2-AS1 exhibited a 13.43-fold increase in tumor compared to healthy non-tumor tissues. In contrast, Lnc-MAP6-1:3 did not demonstrate a statistically significant difference between tumor and normal tissues. ROC curve analysis indicated that ITGB2-AS1 had excellent diagnostic performance (AUC = 0.83, $P < 0.0001$), while HNF1A-AS1 and Lnc-MAP6-1:3 showed moderate diagnostic accuracy.

Conclusion

Our findings demonstrate that ITGB2-AS1 and HNF1A-AS1 are significantly overexpressed in tumor tissues and may serve as promising molecular biomarkers for CRC diagnosis. Further large-scale and independent studies are needed to validate their clinical utility and translational potential, and elucidate their biological functions in colorectal carcinogenesis.

Introduction

Colorectal cancer (CRC), comprising malignancies of the colon and rectum, is one of the most commonly diagnosed malignancies and a major contributor to cancer-related mortality worldwide. Despite advances in screening and therapeutic strategies, the global burden of CRC continues to rise, particularly in low- and middle-income countries and among younger adults. Therefore, identifying reliable molecular biomarkers for early diagnosis and targeted therapy remains a major clinical priority[1]. CRC development is a complex, multistage process characterized by the gradual accumulation of genetic and epigenetic abnormalities that promote malignant transformation[2]. Among the signaling pathways implicated in colorectal tumorigenesis, the Wnt/β-catenin pathway plays a central role. Under physiological conditions, Wnt signaling regulates cell proliferation, differentiation, migration, and tissue homeostasis. Binding of Wnt ligands to Frizzled receptors stabilizes β-catenin, allowing its nuclear translocation and subsequent activation of target genes. In CRC, mutations in key pathway components, particularly APC and CTNNB1 (β-catenin), result in constitutive activation of Wnt signaling, thereby promoting uncontrolled cellular proliferation and tumor progression[3, 4].

Long non-coding RNAs (lncRNAs) constitute a heterogeneous class of RNA transcripts longer than 200 nucleotides that lack protein-coding potential [5]. Based on their genomic localization and function, lncRNAs participate in diverse biological processes through regulation of chromatin remodeling, transcription, post-transcriptional gene expression, and RNA stability[5, 6]. They may function as molecular scaffolds, decoys, guides, or competing endogenous RNAs (ceRNAs) by interacting with proteins, messenger RNAs, and microRNAs [7].

Accumulating evidence indicates that dysregulated lncRNA expression contributes to the initiation, progression, metastasis, and therapeutic resistance of many malignancies, including colorectal cancer. Furthermore, the remarkable tissue specificity and stability of lncRNAs have made them attractive candidates as diagnostic and prognostic biomarkers [8–10]. Several lncRNAs have been identified as important regulators of the Wnt/β-catenin signaling pathway through modulation of β-catenin stability, regulation of transcription factors, and sequestration of microRNAs. Consequently, Wnt-associated lncRNAs have attracted considerable attention as potential biomarkers and therapeutic targets in CRC [11, 12].

ITGB2-AS1 is an antisense lncRNA located adjacent to the ITGB2 gene. Experimental studies in breast cancer, osteosarcoma, renal cell carcinoma, pancreatic cancer, and non-small cell lung cancer have demonstrated that ITGB2-AS1 promotes tumor cell proliferation, migration, invasion, and metastasis through competing endogenous RNA mechanisms and activation of signaling pathways including FAK and MMP9 [13]. However, direct experimental evidence regarding its role in colorectal cancer remains limited, with most studies restricted to bioinformatic analyses and pan-cancer expression profiling[14]. Therefore, further investigation is required to clarify its biological significance in CRC.

HNF1A-AS1, an antisense transcript of the HNF1A gene, is a well-characterized oncogenic lncRNA that is frequently overexpressed in colorectal cancer. Previous studies have shown that HNF1A-AS1 promotes cell proliferation, migration, invasion, epithelial-mesenchymal transition, glycolysis, and chemoresistance

through multiple molecular mechanisms, including the miR-34a/SIRT1/p53, miR-214/INHBA, miR-124/MYO6, and PI3K/AKT signaling pathways [15, 16]. Elevated expression of HNF1A-AS1 has also been associated with poor prognosis and lymph node metastasis in CRC[16, 17].

Lnc-MAP6-1:3 is a recently identified oncogenic lncRNA that has been primarily investigated in osteosarcoma. Functional studies have demonstrated that its silencing suppresses cell proliferation and colony formation while inducing apoptosis through regulation of the Bax/Bcl-2 and Wnt/β-catenin signaling pathways[18, 19]. However, its expression profile and clinical significance in colorectal cancer have not yet been investigated.

Given the emerging importance of Wnt-related lncRNAs in colorectal carcinogenesis, the present study aimed to evaluate the expression levels of ITGB2-AS1, HNF1A-AS1, and Lnc-MAP6-1:3 in colorectal cancer tissues and to assess their potential diagnostic value as molecular biomarkers for CRC.

Materials and Methods

Patient Selection and Tissue Collection

Thirty paired colorectal cancer tissues and matched adjacent normal colorectal tissues were collected from patients undergoing surgical resection at Firouzgar and Jam Hospitals, Tehran, Iran, between September 2024 and December 2025. The sample size was determined based on previous studies reporting an expected difference of approximately 0.30 in the expression levels of the target lncRNAs between tumor and normal tissues. Assuming a standard error of 0.20 and a significance level (α) of 0.05, the minimum required sample size was estimated to be 28 subjects per group. To compensate for potential sample loss, 30 paired tissue samples were ultimately included.

Patients were diagnosed with colorectal cancer based on clinical findings, colonoscopic examination, and histopathological confirmation. Inclusion criteria were: (i) the presence of gastrointestinal symptoms suggestive of colorectal malignancy (including rectal bleeding, abdominal pain, or persistent constipation); (ii) confirmation of colorectal tumor during colonoscopy; and (iii) pathological confirmation of CRC diagnosis and tumor stage. Patients with benign colorectal polyps, previous chemotherapy within two months before tissue collection, metabolic disorders, or other malignancies were excluded from the study. The research was conducted following approval by the Ethics Committee of Iran University of Medical Sciences, with written informed consent obtained from all participants before participation. Immediately after surgical resection, tissue specimens were preserved in RNAlater™ solution, transported to the laboratory under cold-chain conditions, and stored until RNA extraction. The clinical and pathological characterization of CRC patients included in Table 1.

Table 1. The clinical and pathological features of CRC patients.

Table 1

Sequences	Primers	Gene/lncRNA
5′-CGGAACCGCTCATTGCC-3′	Forward	β-Actin
5′-ACCCACACTGTGCCCATCTA-3′	Reverse	
5′-ACAGGGAGCCAGGACACC-3′	Forward	ITGB2-AS1
5′-GGAACCAGCACCAGGAACC-3′	Reverse	
5′-GGGAAGCTGAGGTTTTTCC-3′	Forward	HNF-1A-AS1
5′-CACACCAGTCTCAACTCCC-3′	Reverse	
5′-AGAAAAAGCACCTCTTCAAGTGAG-3′	Forward	LNC-MAP6-1:3
5′-TCTCCTACCTAGTATGTCCCT-3′	Reverse	

Study Design

This study investigated the expression profiles of the Wnt-related long non-coding RNAs (lncRNAs) ITGB2-AS1, HNF1A-AS1, and Lnc-MAP6-1:3 in CRC tissues. Total RNA was extracted using TRIzol™ Reagent, complementary DNA (cDNA) was synthesized using the ExcelRT™ Reverse Transcription Kit, and gene expression was estimated by real-time PCR (qRT-PCR) using RealQ Plus 2× Master Mix Green, as described previously [20].

RNA Extraction and DNase I Treatment

Total RNA was extracted from colorectal tissue samples using TRIzol™ Reagent (Thermo Fisher Scientific, USA) according to the manufacturer's instructions. Briefly, approximately 20–50 mg of tissue was homogenized in 1 mL of TRIzol™ reagent and incubated at room temperature for 5 min. Chloroform (200 µL) was added, and samples were vigorously mixed for 20 s followed by centrifugation at 12,000 × g for 15 min at 4°C. The aqueous phase was transferred to a new microcentrifuge tube, mixed with 500 µL of isopropanol, and centrifuged at 12,000 × g for 10 min at 4°C to precipitate total RNA. The RNA pellet was washed with 1 mL of 75% ethanol and centrifuged at 7,500 × g for 5 min at 4°C. After air-drying for approximately 5 min, the RNA pellet was dissolved in 50 µL of RNase-free water and incubated

at 55°C for 10 min. To eliminate residual genomic DNA contamination, RNA samples were treated with DNase I according to the manufacturer's protocol. RNA concentration and purity were determined using a NanoDrop spectrophotometer (Maestrogen Inc., Taiwan). Purified RNA samples were stored at − 80°C until further analysis.

cDNA Synthesis

Complementary DNA (cDNA) was synthesized using the ExcelRT™ Reverse Transcription Kit (SMOBIO, Korea). Each 20 µL reverse transcription reaction contained 1 µg of total RNA, 0.5 µL reverse transcriptase, 4 µL of 5× reaction buffer, 1 µL dNTP mixture, 2 µL of random hexamer/oligo(dT) primer mixture, and nuclease-free water to the final volume. Reverse transcription was achieved under the following conditions: 25°C for 10 min, 42°C for 60 min, and enzyme inactivation at 85°C for 5 min. The synthesized cDNA samples were stored at − 20°C until quantitative PCR analysis.

Quantitative Real-Time PCR (qRT-PCR)

Gene-specific primers for ITGB2-AS1 and Lnc-MAP6-1:3 were designed using Beacon Designer software and validated for specificity using NCBI Primer-BLAST. The primer sequences are presented in Table 2. Primers for HNF1A-AS1 and the endogenous reference gene (β-Actin) were found from formerly published studies or designed using the same strategy. Quantitative real-time PCR was performed using RealQ Plus 2× Master Mix Green Without ROX™ (Ampliqon, Denmark) on a Corbett RG-6000 Real-Time PCR System (Qiagen, Australia). Each 15 µL reaction contained 7.5 µL of 2× master mix, 1 µL of forward primer (5 pmol/µL), 1 µL of reverse primer (5 pmol/µL), 1 µL of cDNA template, and nuclease-free water to the final volume. PCR amplification was carried out under the following cycling conditions: an initial denaturation at 95°C for 10 min, followed by 40 cycles of 95°C for 15 s, 60°C for 20 s, and 72°C for 15 s. All reactions were performed in triplicate, and no-template controls (NTCs) were included in each run to monitor contamination. Amplification efficiency for each primer pair was evaluated using LinRegPCR software (version 12.x; Academic Medical Center, Amsterdam, The Netherlands). The relative expression levels of the target lncRNAs were normalized against β-actin, which served as the endogenous control.

Table 2
The clinicopathological features of CRC patients.

Parameters		Frequency	Percentage
Age	< 60	14	46.7
	≥ 60	16	53.3
Sex	M	19	63.3
	F	11	36.7
Tumor location	Colon	7	23.3
	Rectum	23	76.7
Polyp	+	9	30
		21	70
Colitis	+	14	46.7
	-	16	53.3
Metastasis	+	9	30
	-	21	70
Pathology	Well	23	76.7
	Moderate	5	16.7
	Poor	2	6.7
Family history	+	4	13.3
	-	26	86.7
Tumor size	< 2 cm	7	23.3
	2-3.99 cm	18	60
	≥ 4 cm	5	16.7
TNM	T2	18	60
	T3	9	30
	T4	3	10

Statistical Analysis

The relative expression levels of the target lncRNAs were determined using the Relative Expression Software Tool (REST, version 2.0.13; QIAGEN GmbH, Hilden, Germany). Statistical analyses were conducted using SPSS software (version 17.0; SPSS Inc., Chicago, IL, USA). Data distribution was

evaluated prior to statistical testing. Comparisons between the two groups were conducted using the independent-samples *t*-test for normally distributed data and the Mann–Whitney U test for data that did not follow a normal distribution. Comparisons among multiple clinicopathological groups (including tumor differentiation, tumor size, and disease stage) were performed using one-way analysis of variance (ANOVA) or the Kruskal–Wallis test, as appropriate. Receiver operating characteristic (ROC) curve analysis was conducted to evaluate the diagnostic performance of the investigated lncRNAs. Graphs were generated using GraphPad Prism (version 8.2.1; GraphPad Software, San Diego, CA, USA). A P value of < 0.05 was considered indicative of statistical significance.

Results

Expression of Wnt-Related lncRNAs in Colorectal Cancer Tissues

Quantitative real-time PCR analysis demonstrated that ITGB2-AS1 was significantly upregulated in colorectal cancer tissues compared with matched adjacent normal tissues. The expression level of ITGB2-AS1 was increased by approximately 13.43-fold in tumor tissues ($P < 0.05$; Fig. 1). Similarly, HNF1A-AS1 expression was significantly higher in colorectal cancer tissues than in adjacent normal tissues ($P = 0.0052$; Fig. 1). In contrast, although Lnc-MAP6-1:3 showed an increased expression trend in tumor tissues, the difference did not reach statistical significance ($P = 0.21$; Fig. 1). These findings advocate that ITGB2-AS1 and HNF1A-AS1 are significantly dysregulated in colorectal cancer and may contribute to CRC pathogenesis.

Diagnostic Performance of ITGB2-AS1, HNF1A-AS1, and Lnc-MAP6-1:3

Receiver operating characteristic (ROC) curve analysis was conducted to assess the diagnostic potential of the investigated lncRNAs for distinguishing colorectal cancer tissues from adjacent normal tissues. Among the investigated biomarkers, ITGB2-AS1 demonstrated the highest diagnostic accuracy, with an area under the ROC curve (AUC) of 0.83 (95% CI: 0.7627–0.9084), a sensitivity of 76.6%, a specificity of 80.0%, a cut-off value of 1.69, and a standard error of 0.037 ($P < 0.0001$; Fig. 2). HNF1A-AS1 exhibited moderate diagnostic performance, with an AUC of 0.67 (95% CI: 0.5755–0.7681), a sensitivity of 65%, a specificity of 61%, cut-off value of 1.68, and standard error of 0.049 ($P = 0.0012$; Fig. 2). Similarly, Lnc-MAP6-1:3 demonstrated moderate diagnostic value, with an AUC of 0.67 (95% CI: 0.5747–0.7734), sensitivity of 73%, specificity of 60%, cut-off value of 1.63, and standard error of 0.050 ($P = 0.01$; Fig. 2). Overall, these findings indicate that ITGB2-AS1 possesses the greatest diagnostic potential among the investigated lncRNAs for colorectal cancer.

Discussion

Long non-coding RNAs (lncRNAs) have emerged as important regulators of tumor initiation, progression, metastasis, and therapeutic response. Increasing evidence indicates that dysregulated lncRNA expression contributes to colorectal carcinogenesis by modulating multiple signaling pathways, particularly the Wnt/β-catenin pathway. Abnormal activation of Wnt signaling contributes to aberrant cell proliferation, EMT induction, invasive behavior, and metastatic progression, making Wnt-associated lncRNAs attractive candidates for diagnostic and therapeutic applications[21, 22]. Consequently, numerous studies have investigated the clinical significance of Wnt-related lncRNAs as biomarkers for cancer diagnosis and prognosis [23, 24]. In this regard, investigating Wnt-associated lncRNAs may provide additional insight into the molecular mechanisms underlying CRC and may identify candidate biomarkers for diagnosis, prognosis, or therapeutic intervention.

In the present study, we evaluated the expression profiles of three Wnt-associated lncRNAs, ITGB2-AS1, HNF1A-AS1, and Lnc-MAP6-1:3, in colorectal cancer tissues. The identification of altered expression of these transcripts would support the concept that lncRNA-mediated regulation of Wnt signaling may contribute to CRC pathogenesis. Importantly, however, the three lncRNAs should not necessarily be considered equivalent molecular markers. Previous studies indicate that HNF1A-AS1 has a relatively well-established role in CRC, whereas the evidence for ITGB2-AS1 and Lnc-MAP6-1:3 has predominantly been generated in other tumor types. Consequently, their expression in CRC may represent a potentially important finding that requires functional validation.

Our findings demonstrated significant overexpression of ITGB2-AS1 and HNF1A-AS1 in colorectal cancer tissues compared with matched adjacent normal tissues. Furthermore, ROC curve analysis indicated that these lncRNAs, particularly ITGB2-AS1, possess promising diagnostic potential for distinguishing colorectal cancer from non-tumorous tissues. In contrast, Lnc-MAP6-1:3 did not exhibit significant differential expression in our investigation, suggesting that its diagnostic value in colorectal cancer may be limited or requires further validation in larger populations. Previous studies have consistently revealed the oncogenic role of ITGB2-AS1 in several human malignancies. In breast cancer, ITGB2-AS1 promotes tumor cell migration and invasion through upregulation of ITGB2, leading to activation of focal adhesion kinase (FAK) signaling and increased expression of matrix metalloproteinases[24]. Similarly, in clear-cell renal cell carcinoma, ITGB2-AS1 has been shown to enhance cell proliferation, migration, and invasion while suppressing apoptosis through regulation of the miR-338-3p/EGFR signaling axis [25]. Moreover, in non-small cell lung cancer, ITGB2-AS1 contributes to cisplatin resistance by inhibiting ferroptosis via activation of the [26]. Collectively, these findings support the role of ITGB2-AS1 as an oncogenic regulator involved in tumor progression across multiple cancer types. The potential relevance of ITGB2-AS1 to CRC is nevertheless biologically plausible. Integrin-related signaling has important functions in cell adhesion, migration, extracellular-matrix interactions, and metastatic progression, all of which are closely interconnected with Wnt/β-catenin signaling and epithelial–mesenchymal transition. Indeed, the integrin β2 (ITGB2) axis itself has been implicated in CRC metastasis; for example, LINC01272 was reported to promote CRC invasion and metastasis through a miR-876/ITGB2 regulatory axis. However, this evidence should not be interpreted as proof that ITGB2-AS1 regulates ITGB2 or Wnt signaling in CRC. If our observed alteration in ITGB2-AS1 expression is confirmed in larger CRC cohorts, functional

studies involving ITGB2-AS1 knockdown or overexpression, β-catenin localization, TCF/LEF transcriptional activity, and downstream Wnt targets would be necessary to determine whether this lncRNA has a causal role in CRC biology. Although direct functional evidence for ITGB2-AS1 in colorectal cancer remains limited, the marked overexpression observed in our study is consistent with its oncogenic role reported in other malignancies. These findings indicate that ITGB2-AS1 may contribute to colorectal tumorigenesis through similar molecular mechanisms involving cell proliferation, migration, invasion, and Wnt-related signaling pathways [27]. Additional mechanistic studies are needed to elucidate its biological function in colorectal cancer.

Our findings regarding HNF1A-AS1 are also consistent with previous reports identifying this lncRNA as an important oncogenic regulator in colorectal cancer. Elevated HNF1A-AS1 expression has been associated with enhanced proliferation, migration, invasion, epithelial–mesenchymal transition, glycolysis, and lymph node metastasis in CRC[17]. Mechanistically, HNF1A-AS1 functions as a competing endogenous RNA (ceRNA), regulating multiple downstream signaling pathways, including the miR-124/MYO6, miR-34a/SIRT1/p53, miR-214/INHBA, and PI3K/AKT axes. Thus, altered HNF1A-AS1 expression in CRC may reflect not only a general disturbance of lncRNA expression but also a biologically relevant mechanism connecting non-coding RNA regulation with a central oncogenic pathway. In addition, increased serum levels of HNF1A-AS1 have been reported in patients with colorectal cancer, suggesting that this lncRNA may serve as a minimally invasive diagnostic biomarker [16]. The observed overexpression of HNF1A-AS1 in our study further supports its potential clinical utility in CRC diagnosis.

Unlike ITGB2-AS1 and HNF1A-AS1, the biological significance of Lnc-MAP6-1:3 remains poorly understood. Previous investigations have primarily focused on osteosarcoma, where Lnc-MAP6-1:3 has been reported to promote tumor progression by regulating the Bax/Bcl-2 apoptotic pathway and Wnt/β-catenin signaling. Knockdown of this lncRNA suppresses cell proliferation and colony formation while inducing apoptosis in osteosarcoma cells[18]. However, to the best of our knowledge, there are currently no comprehensive studies evaluating its role in colorectal cancer. The absence of significant dysregulation in our patient cohort may indicate tissue-specific functions or reflect the relatively small sample size. Therefore, further experimental and clinical investigations involving larger cohorts are required to determine whether Lnc-MAP6-1:3 contributes to colorectal carcinogenesis.

Overall, the present study demonstrates that ITGB2-AS1 and HNF1A-AS1 are significantly overexpressed in CRC tissues and exhibit promising diagnostic performance. These findings are consistent with previous reports describing their oncogenic functions in CRC and other malignancies. Nevertheless, several limitations should be considered when interpreting these findings. The relatively small sample size and single-center design may have limited the statistical power and generalizability of the results. Furthermore, this study focused exclusively on expression analysis and did not include functional experiments to investigate the molecular mechanisms underlying the observed dysregulation. Future studies should include larger multicenter cohorts, analyses of circulating lncRNAs, and mechanistic investigations using in vitro and in vivo models to validate the biological and clinical significance of

these lncRNAs in colorectal cancer. RNA-protein interaction assays and competing-endogenous-RNA analyses could additionally clarify whether these lncRNAs function through protein binding or miRNA sequestration. Finally, prospective studies should determine whether these transcripts have value in blood or other minimally invasive specimens and whether their expression adds predictive information beyond established CRC biomarkers.

Conclusion

In conclusion, the present findings contribute to the growing evidence that Wnt-associated lncRNAs may have clinical potentials for CRC patients. Among the investigated transcripts lncRNAs ITGB2-AS1 and HNF1A-AS1 are significantly upregulated in CRC tissues and HNF1A-AS1 has the most compelling CRC-specific evidence and appears to promote malignant behavior, at least in part, through activation of the Wnt/β-catenin pathway. ITGB2-AS1 and Lnc-MAP6-1:3 are also biologically plausible candidates because their oncogenic and Wnt-related activities have been demonstrated in other cancers, but their specific functions in CRC require further investigation. Collectively, these lncRNAs may represent promising molecular candidates for understanding Wnt dysregulation in CRC and, following appropriate validation, could contribute to the development of novel diagnostic, prognostic, or therapeutic strategies.

Declarations

Competing interests

The authors declare no competing interests.

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Author Contribution

AA, and ISF developed the theoretical formalism, PA and At.A performed the analytic calculations, and MR and SPT performed the numerical and clinical simulations. The authors contributed to the final version of the manuscript. AA supervised the project.

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Data availability

The data sets generated during and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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Figures

Figure 1

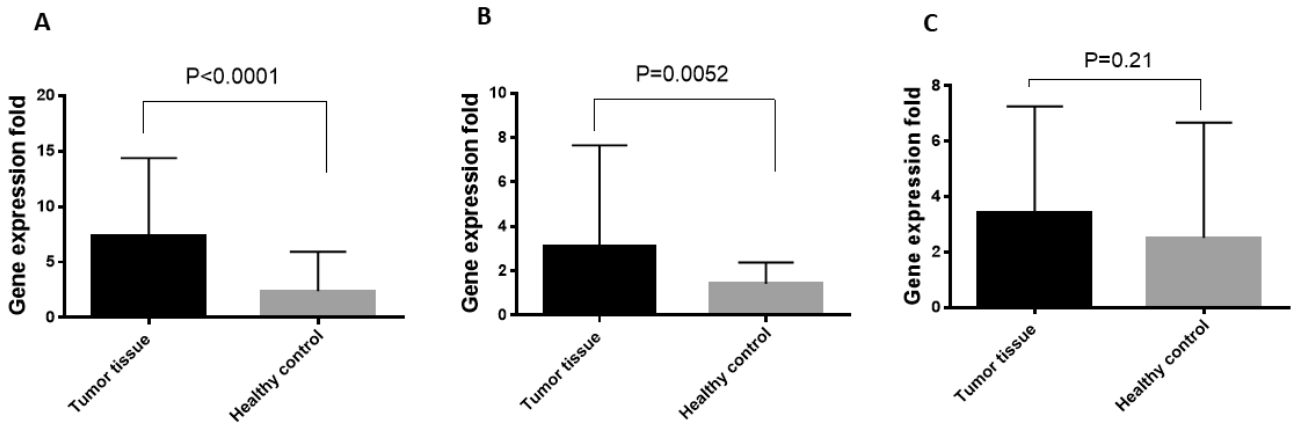


Figure 1

Relative expression levels of ITGB2-AS1, HNF1A-AS1, and Lnc-MAP6-1:3 in CRC. Relative expression levels of lncRNAs in colorectal cancer tissues compared with matched adjacent normal tissues, normalized to β-actin. ITGB2-AS1 (A) and HNF1A-AS1 (B) were significantly upregulated in colorectal cancer tissues (*P*<0.0001 and *P*=0.0052, respectively). Lnc-MAP6-1:3 (C) showed no statistically significant difference between tumor and normal tissues (*P*=0.21).

Figure 2

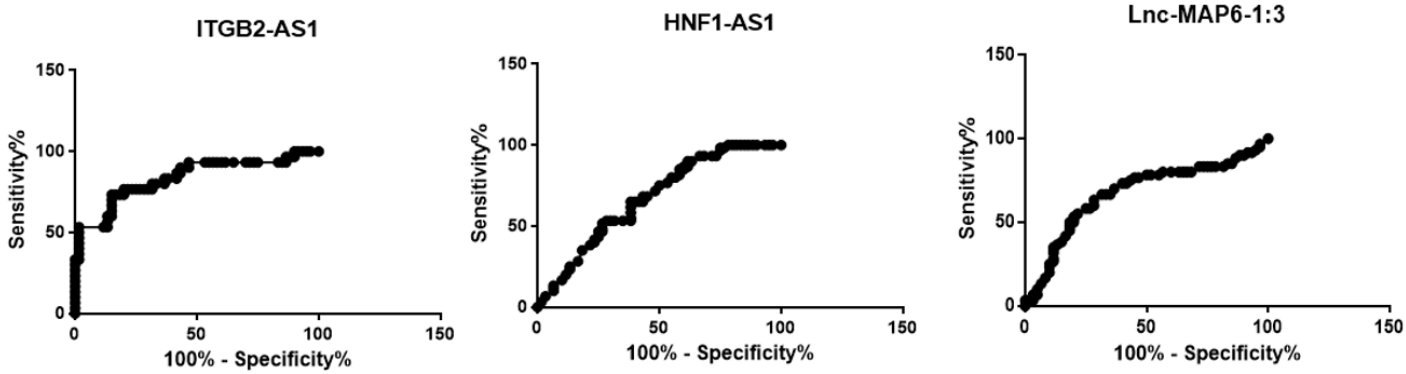


Figure 2

Diagnostic performance of lncRNAs for CRC patients. Receiver operating characteristic (ROC) curve analysis evaluating the diagnostic performance of ITGB2-AS1, HNF1A-AS1, and Lnc-MAP6-1:3 for distinguishing colorectal cancer tissues from adjacent normal tissues.