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Prognostic Model for Colon Cancer (COAD) Based on Migrasome-Related LncRNAs

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Abstract: Objective: Colon adenocarcinoma (COAD) is a leading cause of cancer-related mortality worldwide. In recent years, migrasomes—a newly identified class of extracellular vesicles—have attracted increasing attention for their potential role in intercellular communication. This study aimed to investigate the role of migrasome-associated long non-coding RNAs (CMiSlncRNAs) in COAD progression using bioinformatics approaches and to evaluate their potential as diagnostic biomarkers or therapeutic targets. **Method:** Migrasome-associated genes were identified through a comprehensive literature review and intersected with LncRNAs expressed in COAD. A total of 41 co-expressed CMiSLncRNAs were identified. Univariate Cox regression analysis revealed 13 CMiSLncRNAs with significant prognostic value. A prognostic model was constructed using LASSO regression combined with multivariate Cox proportional hazards analysis, and patients were stratified into high- and low-risk groups based on the median risk score. Kaplan–Meier analysis, principal component analysis (PCA), and functional enrichment analysis were performed to compare the two risk groups. Finally, the influence of CMiSLncRNAs on tumor immune infiltration, immune function, and drug sensitivity was investigated. **Results:** Thirteen CMiSLncRNAs with prognostic significance were identified. The prognostic model demonstrated strong discriminatory ability, with low-risk patients showing significantly better overall survival than high-risk patients across training, testing, and full cohorts. Multivariate Cox analysis confirmed that the risk score was an independent prognostic factor. Functional enrichment analysis indicated that CMiSLncRNAs are involved in pathways such as Hippo, mTOR, and Wnt signaling. Immune analysis revealed a more active immune microenvironment in the low-risk group, characterized by higher immune function scores and increased infiltration of activated NK cells and mast cells. Drug sensitivity analysis revealed distinct drug response profiles between the two risk groups. **Conclusion:** The CMiSlncRNA-based prognostic model offers novel insights for risk stratification and personalized treatment in COAD. These findings highlight the significant roles of CMiSLncRNAs in tumor progression, immune regulation, and drug sensitivity.

Keywords: Colon Cancer; Migrasome; LncRNA; Immunotherapy; Tumor Microenvironment
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1. Introduction

COAD is a multifactorial disease driven by complex interactions between genetic and environmental factors. The lack of

distinct early symptoms results in a substantial proportion of patients being diagnosed at advanced stages, thereby greatly diminishing treatment efficacy [1]. Despite continuous progress in therapeutic approaches, tumor metastasis and chemotherapy resistance remain pivotal determinants of patient prognosis.

Migrasomes, a newly characterized class of extracellular structures, were first described by Liang Ma and colleagues in 2014. These structures originate from retraction fibers left behind by migrating cells [2, 3]. With diameters ranging from approximately 50 to 100 nm, migrasomes are extracellular vesicles that contain specific proteins, RNAs, and organelles [3, 4], and play a critical role in material exchange and signal transduction between tumor cells [2]. In addition, their involvement in the regulation of vascular homeostasis, as well as in tumor invasion and metastasis, has been reported [5, 6]. Migrasomes have been identified in various tumor cell types, including human breast cancer MDA-MB-231 cells and human colon cancer HCT116 cells [5].

Long non-coding RNAs (lncRNAs) are a class of non-coding RNAs exceeding 200 nucleotides in length and are known to exert diverse regulatory functions within cells [7, 8]. Accumulating evidence indicates that lncRNAs can promote tumor proliferation and metastasis, positioning them as promising biomarkers and therapeutic targets in cancer [8-10]. For instance, H19 and HEIH are upregulated in gastric cancer and contribute to enhanced proliferation[11,12], while MNX1-AS1 facilitates colorectal tumor progression [8].

Both migrasomes and LncRNAs have been implicated in tumor progression; however, the functional role of migrasome-associated LncRNAs (CMiSlncRNAs) in tumors remains poorly understood. In this study, we employed bioinformatics approaches to systematically investigate the role of CMiSLncRNAs in the progression of colon adenocarcinoma (COAD) and to evaluate their potential as diagnostic biomarkers or therapeutic targets, aiming to provide novel strategies for the clinical diagnosis and treatment of colon cancer.

2. Methods

2.1 Data Processing and Analysis

COAD were obtained from The Cancer Genome Atlas (TCGA) database (https://portal.gdc.cancer.gov). The dataset included 524 clinical files, comprising 483 tumor samples and 41 non-tumor samples. Transcriptome data were processed using Perl (strawberry-perl-5.30.1) to generate separate mRNA and lncRNA expression matrices, as well as to process the corresponding clinical and survival data. Drug sensitivity was predicted using the Tumor Immune Dysfunction and Exclusion (TIDE) database, and data processing and visualization were performed with the “ggpubr” and “limma” R packages.

2.2 Screening and Analysis of Migrasome-Related LncRNAs

Migrasome-associated genes were integrated with mRNA data obtained from TCGA using the “limma” R package to construct a migrasome gene expression matrix. Subsequently, co-expression analysis was performed with predefined thresholds (corFilter > 0.4, $P < 0.001$), leading to the identification of 4,948 CMiSLncRNA. The results of the co-expression analysis were visualized as a Sankey diagram using the “ggalluvial” R package.

2.3 Construction and Validation of the Prognostic Model

The “limma” R package was used to integrate the CMiSlncRNA expression data of COAD patients with clinical data to generate an expression profile dataset. Subsequently, the dataset was randomly divided into training and testing sets at a 1:1 ratio using the “caret” R package. Univariate and multivariate Cox regression analyses were performed, and the least absolute shrinkage and selection operator (LASSO) algorithm combined with ten-fold cross-validation was applied to determine the penalty parameter corresponding to the point with the minimum cross-validation error. A prognostic model was then constructed based on the training set. The model building process was carried out using the “glmnet”, “survminer”, “timeROC”, and “survival” R packages, and the predictive accuracy of the model was evaluated using the testing set.

2.4 Prognostic Analysis and Nomogram Development Independently

To assess the independence of clinical features (age, sex, grade) and the model-derived risk score as prognostic factors, univariate and multivariate Cox regression analyses were performed. A nomogram integrating these clinical factors with the risk score was developed to predict 1-, 3-, and 5-year overall survival, and its performance was validated using calibration plots.

2.5 Examination of Principal Components and Functional Enrichment

The “scatterplot3d” R package was used for principal component analysis (PCA) to classify CMiSLncRNA expression and visualize the spatial distribution of low- and high-risk groups. KEGG and GO enrichment analyses were performed with the “org.Hs.eg.db” and “enrichplot” R packages, while gene set variation analysis (GSVA) was conducted using the “GSVA” R package.

2.6 Tumor Immunity Assessment

To investigate the association between immune cell infiltration and risk stratification, the CIBERSORT algorithm was employed to predict the correlation between risk scores and immune cell abundance. Single-sample gene set enrichment analysis (ssGSEA) was conducted using the “GSVA” R package, with resulting scores subsequently normalized. Box plots were then generated to illustrate the scores of immune-related functions across distinct risk groups.

2.7 Identification of Potential Therapeutic Drugs for Colorectal Cancer

To forecast potential therapeutic agents for colon cancer, drug sensitivity analysis was performed for both high-risk and low-risk groups. The “oncoPredict” R package was utilized to estimate drug response sensitivity in colorectal cancer patients stratified by risk group.

2.8 Statistical Analysis

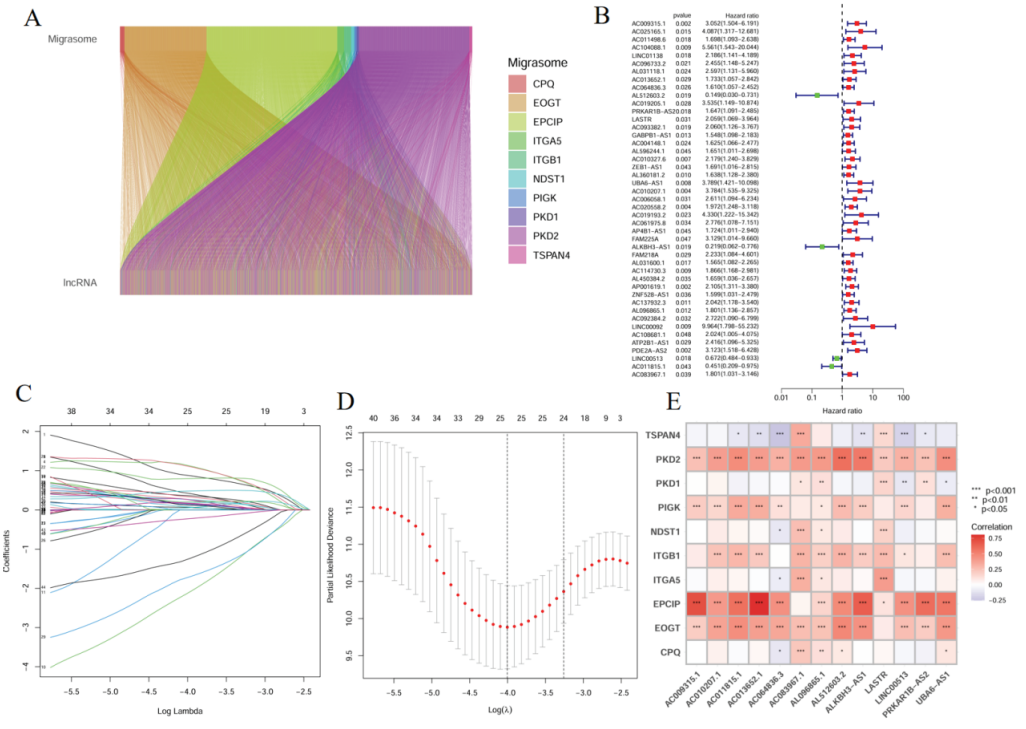
All statistical analyses were performed using R software (version 4.4.0) and Perl (strawberry-perl-5.30.1). Continuous variables were compared using the Wilcoxon rank-sum test, and categorical variables were analyzed using the chi-square test. $P < 0.05$ was considered statistically significant.

3. Results

3.1 LncRNA Identification of 13 Prognostically Significant Migrasome-Related LncRNAs

Expression data for 16,876 LncRNAs associated with COAD were retrieved from the TCGA database, and ten migrasome-related genes were collected from the literature. PCA was conducted to evaluate co-expression relationships between these LncRNAs and the migrasome-associated genes, resulting in the identification of 4,948 LncRNAs. A Sankey diagram was used to visualize the co-expression network between migrasome-related genes and their associated LncRNAs. (Figure 1A).

Figure 1: A The relationship between 10 migrasome-associated genes and LncRNAs. B Univariate Cox analysis shows that 41 LncRNAs are associated with OS prognosis. C, D Lasso analysis. E Co-expression analysis heatmap of 10 migration-associated genes and 13 LncRNAs;””:P<0.05,”**”:P<0.01,”***”:P<0.001.*

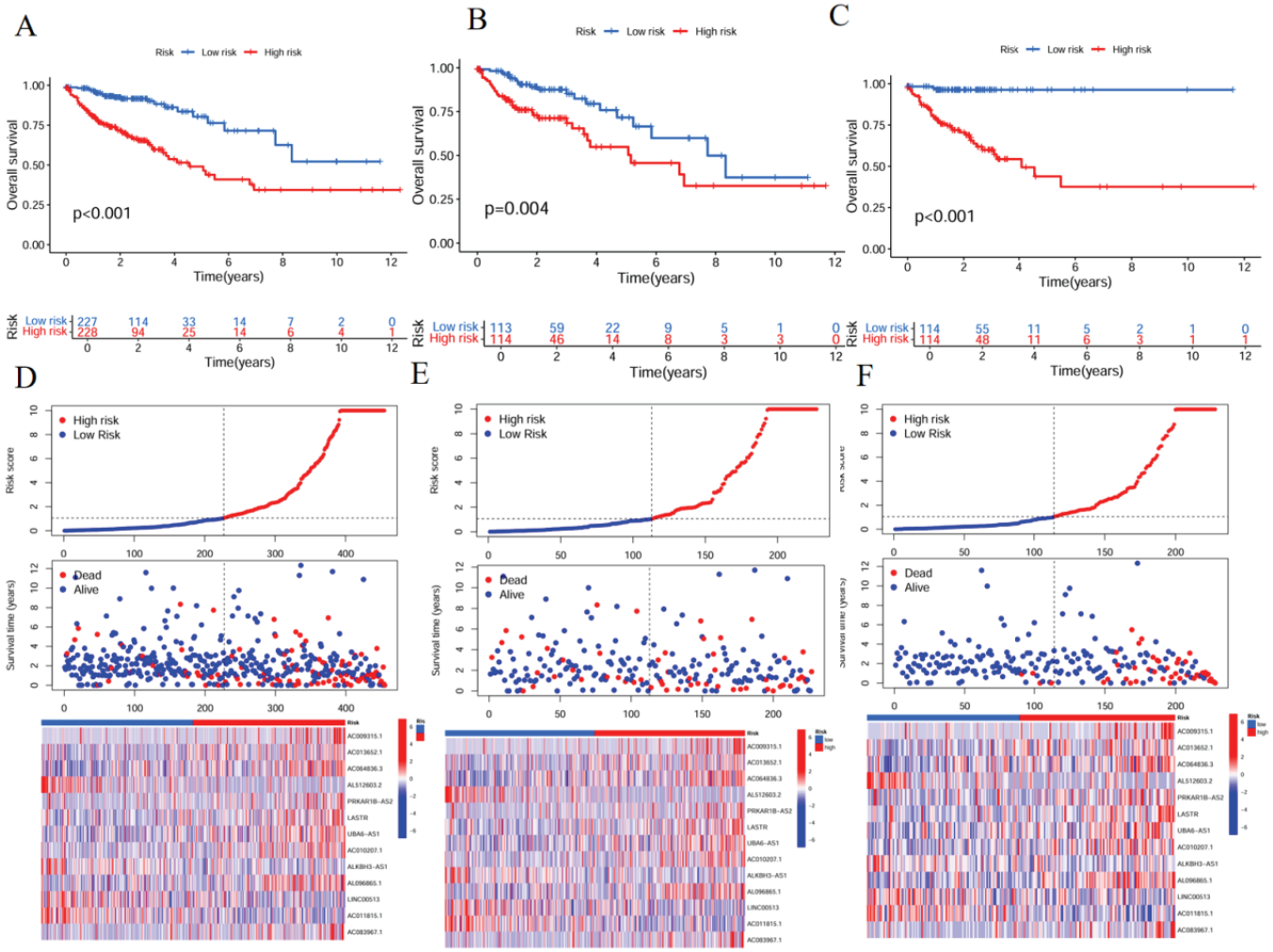


Univariate Cox regression analysis was conducted on the training set, identifying 41 LncRNAs as risk factors (HR > 1) and 4 LncRNAs as protective factors (HR < 1), as shown in the forest plot (Figure 1B). LASSO regression analysis was subsequently performed, resulting in the selection of 25 LncRNAs (Figure 1C, D). Multivariate Cox analysis further revealed 13 CMiSLncRNA with significant prognostic value in COAD. A heatmap was generated to display the co-expression patterns of these 13 LncRNAs and the 10 CMiSLncRNA (Figure 1E).

3.2 Evaluation of a Colorectal Cancer Prediction Model Based on CMiSLncRNA

The dataset was randomly divided into training and testing sets with balanced sample distribution. Patients were subsequently classified into low-risk and high-risk groups based on their risk scores. Kaplan–Meier survival curves demonstrated that, in the training, testing, and full cohorts, patients in the low-risk group had significantly better overall survival (OS) than those in the high-risk group (Figure 2A–C). Heatmaps depicting the expression patterns of the 13 CMiSLncRNAs in low- and high-risk groups revealed consistent expression profiles across the training set, testing set, and the entire cohort (Figure 2D–F).

Figure 2: A Evaluation of the risk model in all samples.B Risk model in the test group.C Risk model in the validation group. D Prediction of the risk model in all samples.E Prediction of the risk model in the test group.F Prediction of the risk model in the validation group.



3.3 The CMiSLncRNAs-Based Model Independently Predicts Outcomes for Colon Cancer Patients.

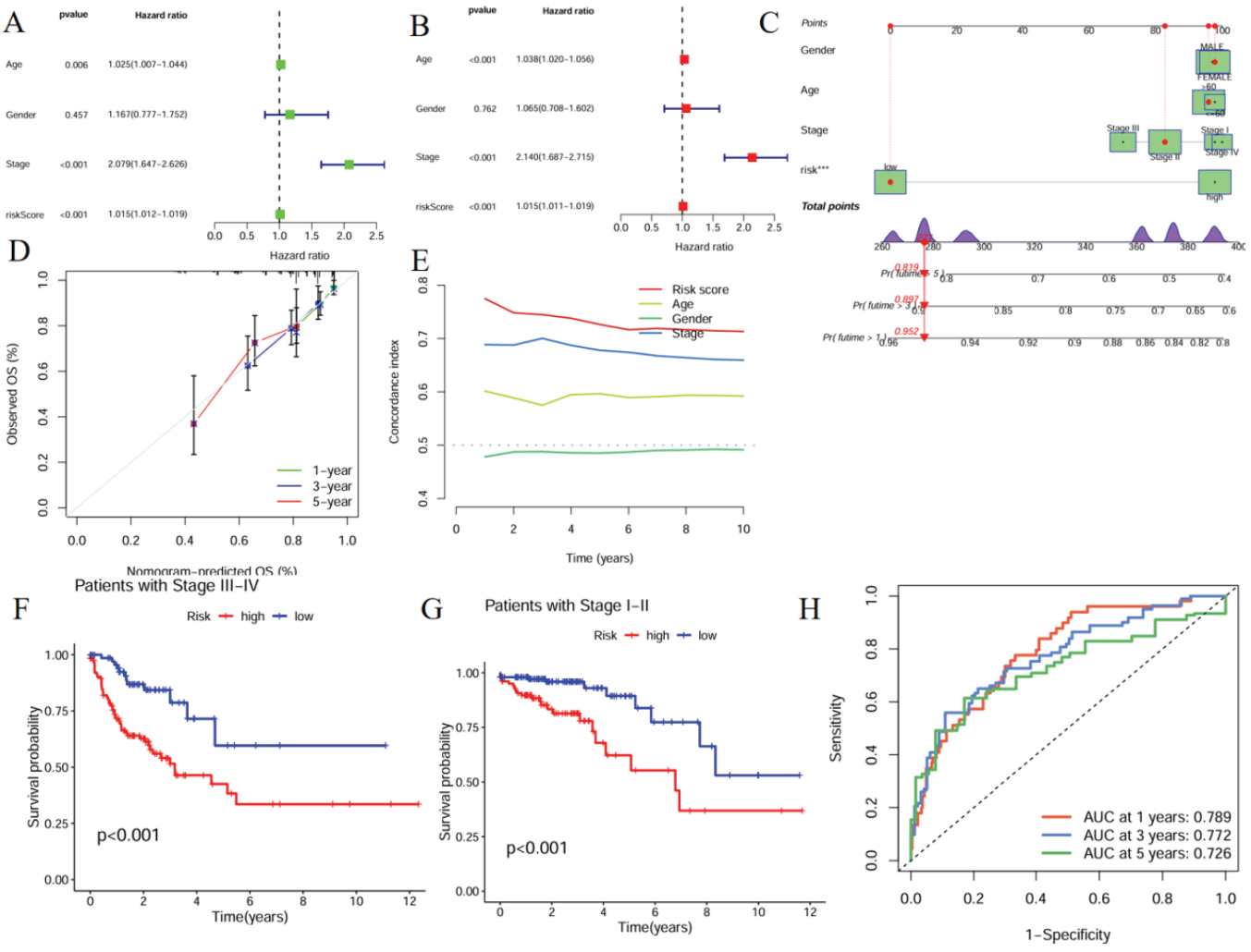
Univariate and multivariate Cox regression analyses were performed to evaluate whether the CMiSLncRNA-based prognostic model served as an independent prognostic factor for COAD (Figure 3A, B). Multivariate Cox regression analysis revealed that age (HR = 1.038, 95% CI: 1.020–1.056, P < 0.001), tumor stage (HR = 2.140, 95% CI: 1.687–2.715, P < 0.001), and risk score (HR = 1.015, 95% CI: 1.011–1.019, P < 0.001) were independent prognostic factors (Figure 3B).

A prognostic nomogram was subsequently constructed incorporating sex, age, stage, and the risk model to predict 1-, 3-, and 5-year survival rates of COAD patients (Figure 3C). Calibration curves demonstrated good predictive accuracy of

the nomogram for 1- and 3-year survival rates (Figure 3D). The concordance index (C-index) was calculated to assess the predictive performance of the model, with results indicating that the risk score derived from the model exhibited the highest predictive accuracy (Figure 3E).

To evaluate the prognostic value of the CMiSLncRNA-based model across different disease stages, separate survival analyses were conducted for COAD patients stratified by stage I–II and stage III–IV (Figure 3F, G). Survival analysis revealed that low-risk patients had significantly better clinical outcomes compared to high-risk patients in both stage I–II ($P < 0.001$) and stage III–IV ($P < 0.001$) subgroups (Figure 3F, G). Furthermore, based on the entire cohort, the area under the curve (AUC) values for 1-, 3-, and 5-year survival were 0.789, 0.772, and 0.726, respectively (Figure 3H), suggesting that the CMiSlncRNA signature exhibits moderate prognostic predictive capability.

Figure 3: A-B Uni-Cox and multi-Cox analyses of clinical pathological factors and risk scores with overall survival.C Nomogram for predicting overall survival.D Calibration curves for 1-year, 3-year, and 5-year overall survival.E CIR-score and other clinical indicators were evaluated using c-index curves.F-G Kaplan-Meier (KM) curves for clinical prognosis based on staging.H AUC curves for 1-year, 3-year, and 5-year survival.



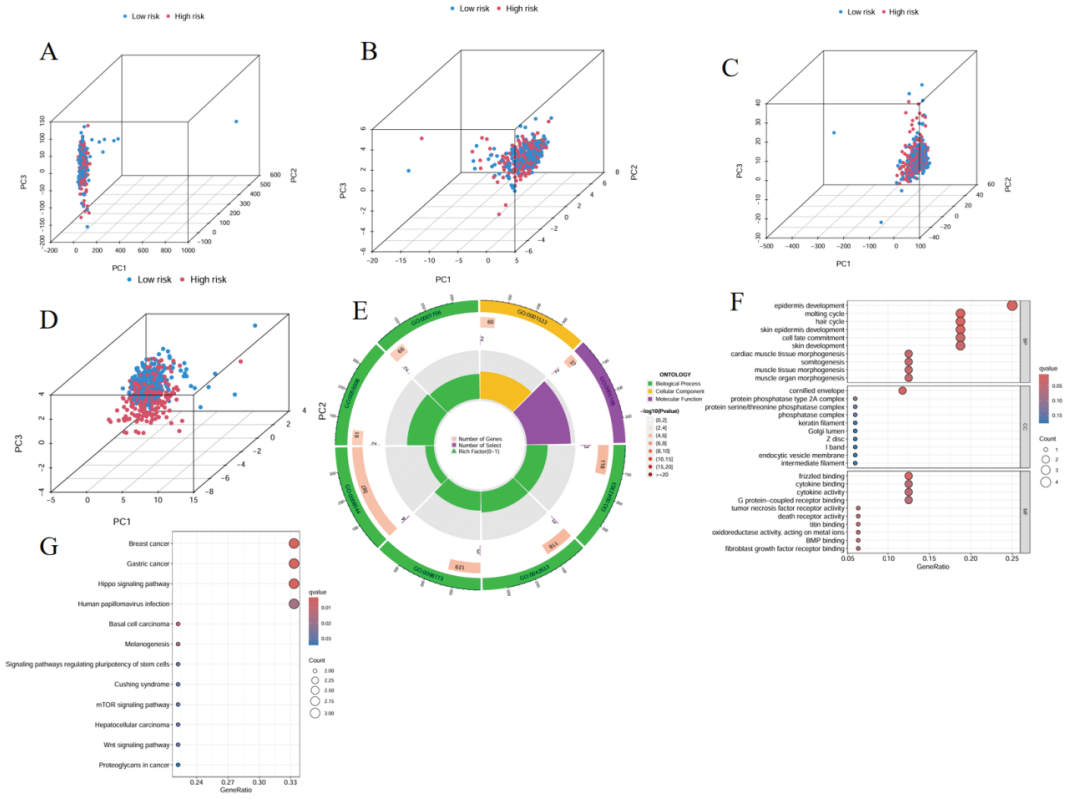
3.4 Principal Component Analysis and Functional Enrichment Analysis

To evaluate whether the LncRNAs incorporated into the prognostic model could effectively distinguish between high-risk and low-risk samples, principal component analysis (PCA) was performed based on the CMiSlncRNA dataset. When PCA was applied to the risk model, the CMiSlncRNAs, migrasome-related genes, and all genes, the results demonstrated that the model effectively differentiated high-risk samples from low-risk samples (Figure 4A–D).

Differential pathway analysis between high-risk and low-risk groups was conducted using the TCGA cohort. GO analysis revealed that CMiSLncRNAs were associated with biological processes including epidermis development, the molting cycle,

and the hair cycle (Figure 4E, F). KEGG analysis showed significant enrichment in pathways such as breast cancer, gastric cancer, the Hippo signaling pathway, pathways regulating pluripotency of stem cells, and the mTOR signaling pathway (Figure 4G). These findings suggest that CMiSLncRNAs may influence the invasion and metastasis of COAD by modulating cell differentiation and tissue remodeling.

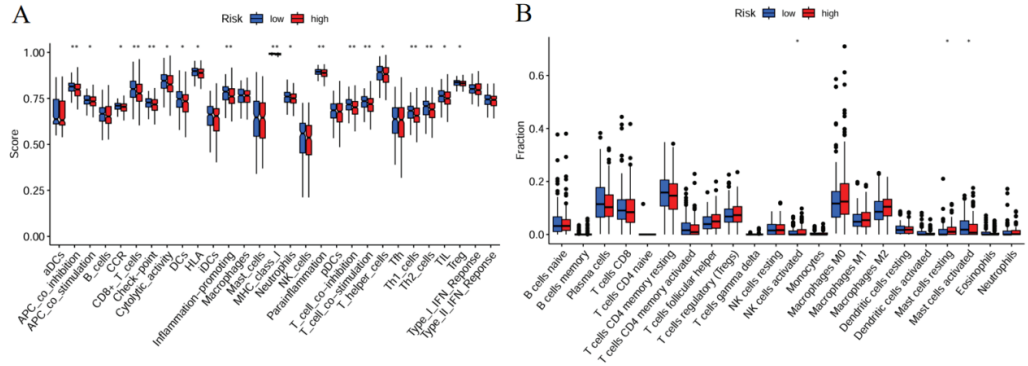
Figure 4: A-D PCA analysis of all genes, migration-associated genes, migration-related LncRNAs, and the risk model we constructed.E-F GO analysis.G KEGG analysis.



3.5 Differences in Tumor Immune Microenvironment (TIME) Between High-Risk and Low-Risk Groups

Immune function scores, assessed via CIBERSORT, were significantly higher in the low-risk group than in the high-risk group, indicating an immunosuppressive state in the latter (Figure 5A). Analysis of 22 immune cell subtypes showed that activated NK cells and activated mast cells were more abundant in the low-risk group, while resting mast cells predominated in the high-risk group (Figure 5B). These findings suggest that the low-risk group maintains a more active immune microenvironment conducive to tumor suppression, whereas the high-risk group exhibits a relatively suppressed immune state that may promote tumor progression.

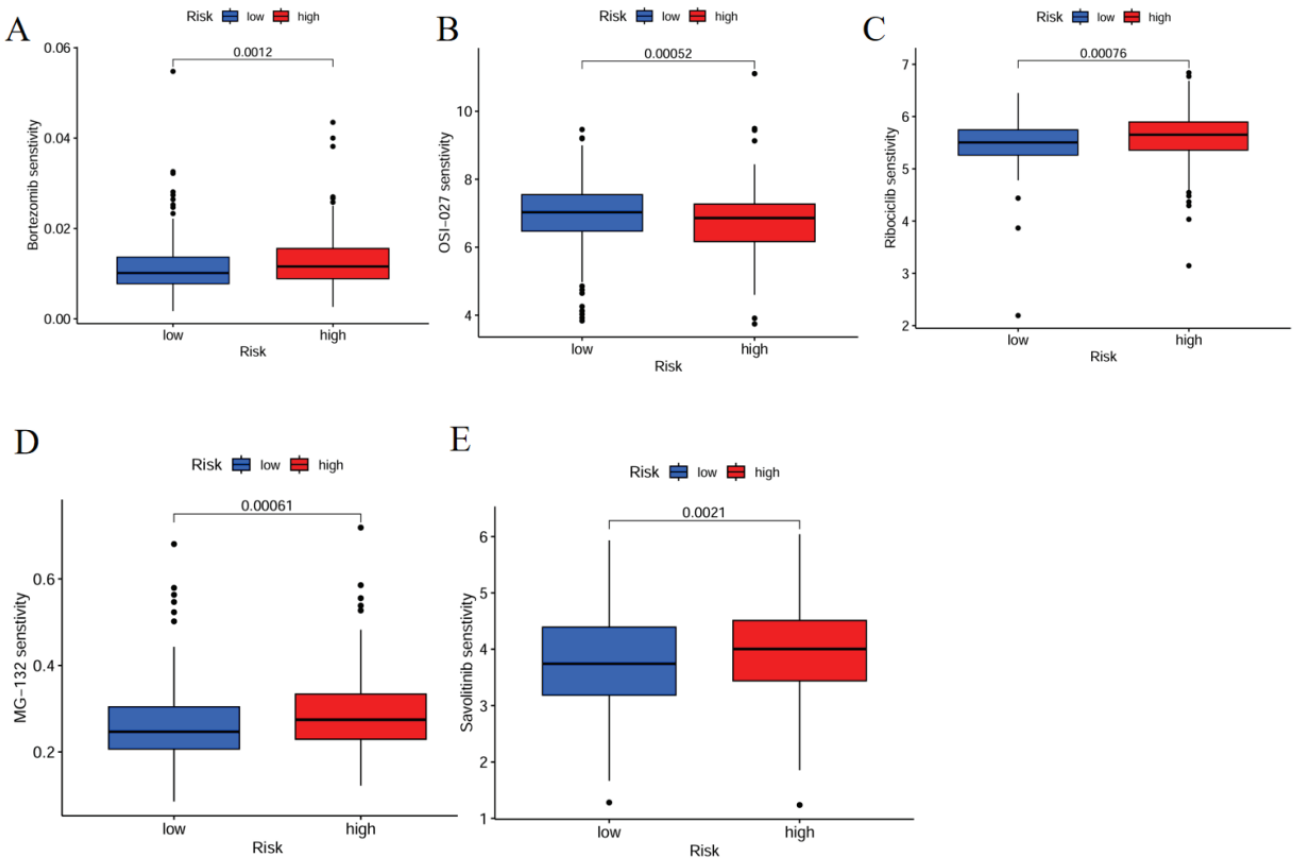
Figure 5: A Differential analysis of immune-related activities between high and low-risk groups("": P<0.05, "**": P<0.01). B Immune cell infiltration analysis in the tumor microenvironment between high and low-risk groups. ("*": P<0.05,"**": P<0.01).*



3.6 Assessment of Drug Responsiveness Based on a Prognostic Risk Model

Sensitivity to 30 of 198 drugs differed significantly between risk groups. Five key drugs (Figure 6A–E) were identified: bortezomib, ribociclib, MG-132, and savolitinib were more sensitive in the low-risk group, whereas OSI-027 showed greater efficacy in the high-risk group. The above findings indicate that patients in different risk groups exhibit distinct drug sensitivity profiles, suggesting that the prognostic risk model holds potential value for guiding personalized therapeutic strategies.

Figure 6: Drug Sensitivity Analysis of High and Low-Risk Groups. A Bortezomib.B OSI-027.C Ribociclib.D MG-132. E Savolitinib.



4. Discussion

CRC ranks among the most prevalent malignancies worldwide [13]. In 2020, approximately 147,950 new cases of CRC were reported globally, with an estimated 36% of diagnosed patients succumbing to the disease. The annual incidence of colon and rectal tumors is increasing at a rate of 1.8% and is progressively affecting younger populations [14, 15]. Consequently, there is an urgent need for the development of novel therapeutic strategies for colon tumors.

LncRNAs have been identified as promoters of invasion and metastasis in various tumor types, including breast cancer [16], non-small cell lung cancer [17], and CRC [18]. Research has also demonstrated that lncRNAs play critical roles in conferring resistance to cisplatin in head and neck squamous cell carcinoma [19] and to oxaliplatin in colorectal cancer [20]. As a result, targeting LncRNAs has emerged as a growing focus in cancer research. For instance, Qu et al. reported that locked nucleic acid antisense oligonucleotides (LNA ASOs) can effectively overcome resistance in advanced renal cell carcinoma [21].

Migrasomes, which are extracellular vesicles resembling exosomes, have been observed to form during cell migration [22, 23]. Given the highly migratory nature of tumor cells, migrasomes may play a significant role in tumor metastasis [23]. Studies have shown that exosomes secreted by metastatic cancer cells can induce epithelial–mesenchymal transition (EMT) in adjacent cells, thereby facilitating cancer dissemination [24]. Furthermore, migrasomes possess the capacity to transport exosomes, which may also contribute to enhanced tumor cell infiltration and metastasis[24].

In this study, we comprehensively utilized CMiSLncRNAs to construct a prognostic risk model for colorectal cancer

patients based on data from TCGA database. Our analysis revealed the potential roles of these LncRNAs in tumor migration, prognostic prediction, and regulation of the tumor immune microenvironment.We further validated their value across different clinical stages and in relation to drug sensitivity. By analyzing co-expression relationships between migrasome-related genes and LncRNAs, we identified LncRNAs associated with colorectal cancer migration. Using LASSO regression and multivariate Cox regression models, we selected 13 CMiSLncRNAs with prognostic significance: AC009315.1, AC010207.1, AC011815.1, AC013652.1, AC064836.3, AC083967.1, AL096865.1, AL512603.2, ALKBH3-AS1, LASTR, LINC00513, PRKAR1B-AS2, and UBA6-AS1.

The roles of these LncRNAs in promoting tumor progression have been documented in the literature. For example, the LncRNA LASTR regulates SART3 activity to promote cancer cell adaptation [25]. Manhui Xia et al. demonstrated that LASTR promotes lung cancer progression through the miR-137/TGFA/PI3K/AKT axis [26]. ALKBH3-AS1 enhances the growth and metastasis of hepatocellular carcinoma cells [27]. PRKAR1B-AS2 promotes tumorigenesis, survival, and chemoresistance via the PI3K/AKT/mTOR pathway [28]. UBA6-AS1 enhances proliferation in glioblastoma and triple-negative breast cancer [29, 30]. LINC00513 accelerates the malignant progression of colorectal cancer by stabilizing connective tissue growth factor (CTGF) mRNA [31].

In this model, the risk score derived from these LncRNAs demonstrated strong discriminatory ability across the training set, testing set, and the entire cohort, with low-risk patients exhibiting significantly higher survival rates than those classified as high-risk. Multivariate Cox analysis further confirmed that the CMiSlncRNA-derived risk score serves as an independent prognostic factor, irrespective of age and tumor stage. Calibration curves for the nomogram predicting 1-, 3-, and 5year survival rates demonstrated moderate prognostic predictive capability. This may be attributed to the fact that migrasome formation is dependent on cell migration status, potentially playing a critical role during specific stages of tumor progression—such as invasion or hematogenous metastasis—while exerting limited effects at other stages. Consequently, a migrasome activity-based signature may show stronger predictive value in certain subgroups, such as patients with established metastases, while appearing moderate in the overall population..PCA further revealed that the CMiSLncRNA-based risk model distinguished high-risk from low-risk patients more clearly than models based on migrasome-related genes, all genes, or other gene sets. Pathway enrichment analysis of differentially expressed genes between the high- and low-risk groups indicated that CMiSLncRNAs are predominantly enriched in pathways such as the Hippo signaling pathway (involved in regulating stem cell pluripotency), the mTOR signaling pathway, and the Wnt signaling pathway. These findings suggest that CMiSLncRNAs may contribute to tumor initiation and progression by influencing cancer cell differentiation, proliferation, and key signaling pathways.

LncRNAs play important roles in the metabolic reprogramming of both tumor and immune cells, thereby influencing antitumor immunity, reshaping the tumor immune microenvironment (TIME), and promoting oncogenesis [32-35]. In our examination of the immune microenvironment, we observed that the low-risk group exhibited significantly higher immune function scores compared to the high-risk group, indicating a more robust immune response. The increased infiltration of natural killer (NK) cells and activated mast cells in the low-risk group may contribute to tumor control [36]. In contrast, the immune system of the high-risk group appeared to be in a suppressed state, characterized by significant infiltration of resting mast cells, which may facilitate immune evasion and tumor progression [37]. Through CIBERSORT analysis and assessment of immune cell proportions, we further validated the marked differences in immune regulation between the high-risk and low-risk groups.

Drug sensitivity analysis demonstrated distinct drug response profiles between risk groups: patients in the low-risk group showed higher sensitivity to bortezomib and ribociclib, while those in the high-risk group exhibited stronger responsiveness to OSI-027. These findings provide a theoretical basis for drug selection in personalized treatment strategies and underscore the critical role of CMiSLncRNAs in guiding therapeutic decisions.

However, several limitations of this study should be considered. The data were obtained from the TCGA database, necessitating further validation of the model's stability and clinical applicability using independent multicenter cohorts. Moreover, additional in vitro and in vivo studies are required to elucidate the molecular mechanisms through which CMiSLncRNAs influence colorectal cancer development and progression, thereby facilitating the identification of potential therapeutic

targets. Collectively, the CMiSlncRNA-based prognostic model developed in this study provides novel perspectives for risk stratification and personalized treatment in colorectal cancer patients.

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Conflict of Interests

The authors declare that there is no conflict of interest regarding the publication of this paper.

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