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Summary

SLC26A9 PROMOTES LIPID METABOLISM BY INTERACTING WITH RRBP1 IN COLORECTAL CANCER

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Colorectal

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Slc26a9 interacts with RRBP1 to activate endoplasmic reticulum stress and drive lipid metabolic reprogramming in colorectal cancer progression.

KEY POINTS

- Slc26a9 was significantly upregulated in mouse colorectal cancer tissues compared to adjacent non-cancerous tissues, and RRBP1 showed the highest binding affinity with Slc26a9 among identified interacting proteins
- Slc26a9 overexpression in Colo201 cells promoted endoplasmic reticulum stress, mitochondrial energy metabolism, and lipid accumulation, with enhanced expression of markers including PERK-ATF4, IRE1α-XBP1, ATF6, Mitofilin, MFN1, COXIV, FASN, ACC, and SCD1
- Co-immunoprecipitation and immunofluorescence experiments demonstrated that Slc26a9 interacts with and co-localizes with RRBP1, with protein-protein docking revealing multiple hydrogen bonds between them
- Analysis of GEO dataset GSE39582 showed positive correlation between Slc26a9 and RRBP1 expression in human colorectal cancer tissues, with co-high expression predicting poor overall survival
- The authors propose that Slc26a9 and RRBP1 may serve as potential prognostic biomarkers and that the Slc26a9-RRBP1 axis represents a novel therapeutic target for colorectal cancer

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