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MiR-155-5p controls colon cancer cell migration via post-transcriptional regulation of Human Antigen R (HuR)

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Abstract

Colorectal cancer (CRC) is the third most common cancer and a significant cause of cancer-related deaths worldwide. Metastasis is the worst prognostic factor for patients with CRC. HuR (ELAVL1) is overexpressed in CRC and has been reported to **promote** colon cancer growth **by targeting RNA in the cell cytoplasm**. Herein, the role of miR-155-5p in regulating HuR expression and cell migration was examined in colon cancer cells. MiR-155-5p knockdown in **serum-starved** colon cancer cells decreased both colon cancer cell chemotaxis and cytoplasmic expression of HuR. Bioinformatics analysis predicted two putative binding sites in the AU-rich elements (AREs) at the 3′-UTR of HuR mRNA. MiR-155-5p binding to HuR was verified using specific target site blockers and functionally validated by use of RNA immunoprecipitation assays, showing that miR-155-5p-dependent regulation of HuR expression is mediated by AREs. Targeting AREs with a specific blocker inhibited colon cancer cell migration. Taken together, these novel findings demonstrate that AREs mediate miR-155-5p positive regulation of HuR mRNA levels and translation as well as migration in colon cancer cells, suggesting that targeting miR-155-5p and/or Hur might be useful therapeutic strategies against colon cancer metastasis.

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