The IncRNA NEAT1 activates Wnt/β-catenin signaling and promotes colorectal cancer progression

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NEAT1 activated Wnt signaling to promote colorectal cancer progression and metastasis

Here, we report that NEAT1 interacted with DDX5 and activated wnt/β-catenin signaling in CRC cells. NEAT1 expression was significantly upregulated in CRC tissues compared with that in normal tissues. Altered NEAT1 expression led to marked changes in proliferation, migration and invasion of CRC cells both in vitro and in vivo. Mechanistically, we found that NEAT1 directly bound to DDX5 and regulated its stability and sequentially activated Wnt signaling. Our study showed that NEAT1 indirectly activated the Wnt/β-catenin signaling pathway via DDX5 and NEAT1 fulfilled its oncogenic functions in a DDX5-mediated manner. Clinically, concomitant NEAT1 and DDX5 protein levels negatively correlated with overall survival and disease free survival of CRC patients. The NEAT1/DDX5/Wnt/β-catenin axis could be a potential therapeutic target in pharmacological strategies.

Introduction

The long non-coding RNA nuclear-enriched abundant transcript 1 (NEAT1) has been reported overexpressed in colorectal cancer (CRC). However, its underlying mechanisms to the progression of CRC has been little studied. NEAT1 is upregulated in human CRC patients and predicted poor prognosis.

In the future, we will confirm whether NEAT1 will activate Wnt/β-catenin signaling by targeting DDX5, and find the clinical associations between NEAT1 and DDX5 in human CRC sample.