Low Expression of Tumor Suppressor ARID1A Correlates with Reduced Expression of E-cadherin in Colorectal Cancer

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Abstract

Metastasis is a major cause of death in Colorectal cancer (CRC) patients, and the Epithelial mesenchymal transition (EMT) has been known to be a crucial event in cancer metastasis. Downregulated expression of AT-rich interaction domain-containing protein 1A (ARID1A), a bona fide tumor suppressor gene, plays an important role in promoting EMT and CRC metastasis, but the underlying molecular mechanisms remain poorly understood. Here, we evaluated the correlation between ARID1A expression and EMT-associated markers, *E-cadherin* and *β-catenin*, in human CRC. We measured the transcription levels of ARID1A, E-cadherin and β-Catenin via real time quantitative PCR (qPCR) in 30 pairs of colorectal cancer tissues and their matched non-tumor adjacent tissues. Interestingly, we found an obvious correlation between the expression of ARID1A and E-cadherin in colorectal cancer tissue samples, however, the correlation coefficient was not perfect ($r = -0.526$). β-Catenin transcription levels was not found to correlate with ARID1A. Thus, ARID1A downregulation may promote CRC metastasis through decreasing EMT-related protein E-cadherin and promoting epithelial cell movement.

Keywords Colorectal cancer, ARID1A, E-cadherin, β-catenin