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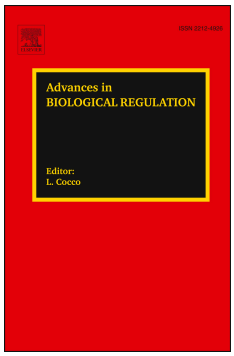

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Interaction of the Wnt/β-catenin and RAS-ERK pathways involving co-stabilization of both β-catenin and RAS plays important roles in the colorectal tumorigenesis

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Abstract

Cancer development is usually driven by multiple genetic and molecular alterations rather than by a single defect. In the human colorectal cancer (CRC), series of mutations of genes are involved in the different stages of tumorigenesis. For example, the *adenomatous polyposis coli* (*APC*) and *KRAS* mutations have been known to play roles in the initiation and progression of the tumorigenesis, respectively. However, many studies indicate that mutations of these two genes, which play roles in the Wnt/β-catenin and RAS-extra-cellular signal regulated kinase (ERK) pathways, respectively, cooperatively interact in the tumorigenesis in