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Controlling Ras stability via the Wnt/β-catein signaling: Implication in cancer therapy

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Three decades after the identification of *Ras* as an oncogene, the field remains as dynamic and important as ever. Ras controls a wide variety of biological processes including cell growth, survival, and differentiation and is also involved in a number of diseases, including cancer and developmental disorders. However, despite the significant progress that has been made, our understanding of Ras is still incomplete.

As adding importance of *K-Ras* mutation in cancer biology we recently found that oncogenic *K-Ras* progress tumorigenesis and metastasis of colorectal cancer haboring *APC* mutations via activating cancer stem cells. Initial activation of β-catenin by *APC* loss and further enhancement through *K-Ras* mutation induces CD44, CD133 and CD166 expression (1).

We also provide convincing evidence for a new Ras regulatory mechanism that provides a potential approach for the direct control of Ras instead of the well-known Ras regulation mechanisms of GDP/GTP exchange and the lipid-directed post-translational modification involved in membrane trafficking. We not only present a detailed mechanism for Ras degradation involving its phosphorylation by negative Wnt/β-catenin signaling via GSK3β but also provide critical pathophysiological evidence related to human colorectal cancer (2). The *in vivo* role of the regulation of Ras stability and the involvement of Ras stabilization in colorectal tumorigenesis were further demonstrated using *Adenomatous polyposis coli* (*Apc*)-defective $Apc^{Min/+}$ and Apc^{1638N} mouse tumours and human colon cancers in various stages, as well as specimens of familial adenomatous polyposis (FAP) caused by *Apc* mutations. In this meeting, I will also discuss on our current status of the development of anti-cancer drugs controlling stability of β-catenin and Ras in control of colorectal cancer.

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