The effect of endogenous Gαq activation on expression of some β-Catenin target genes in SW480 cells

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Abstract

Wnt signal transduction pathways are fundamental mechanisms that direct cell proliferation, cell polarity and cell fate determination during embryonic development and cellular homeostasis. The canonical Wnt signaling pathway functions by regulating the protein stabilization of β-Catenin, a transcriptional co activator that controls the expression of some important cellular genes. Upregulation of β-Catenin transcriptional activity can lead to numerous forms of human cancer, most notably hereditary and sporadic forms of colon cancer. Our laboratory has already presented some evidence that there is a positive relationship between activity of the Gαq class of Gα proteins and cellular β-Catenin protein levels. In order to see the effect of endogenous Gαq signaling on β-Catenin transcriptional activity, SW480 colon cancer cells were cultured under standard conditions and treated with carbachol, thrombin, and trypsin: three agonists known to bind receptors that couple and activate Gαq signaling. Then total cellular RNA was extracted and used for measuring expression of three β-Catenin target genes (*C-MYC*, *CCND1* and *FGF20*) by reverse transciptase-pcr and real time-pcr. Interestingly, treatment of SW480 cells with the Gαq agonists did not lead to a significant change in expression of the β-Catenin target genes used in our study. We are currently investigating the mechanism of the results presented in this study.

Key words: β-Catenin, Gαq, Wnt signaling, Colon cancer