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MUC1-CT regulates UDP: polypeptide N-Acetylgalactosaminyltransferases (ppGalNAc-Ts) expression in pancreatic cancer

Prakash Radhakrishnan and Michael A Hollingsworth

Eppley Institute for Research in Cancer and Allied Diseases, University of Nebraska Medical Center, Omaha, NE 68198-5950

MUC1 is a transmembrane glycoprotein overexpressed and aberrantly glycosylated in most of the human cancers including pancreatic cancer. Overexpression of MUC1 is associated with progression of pancreatic cancer. The MUC1 cytoplasmic tail (MUC1-CT) regulates a variety of genes that are implicated in the process of epithelial to mesenchymal transition (EMT), increased invasiveness and metastasis of cancer cells. In this study we show that overexpression of MUC1 downregulates one of the mucin type *O*-glycan initiating glycosyltransferases, UDP: polypeptide N-acetylgalactosyltransferase-5 (ppGalNAc-T5). The ppGalNAc-T5 is associated with tumor suppressor gene EXT-2. Further, down regulation of MUC1 by shRNA restores and up-regulates ppGalNAc-T5 expression. Our ChIP-on-chip and ChIP analysis reveals that binding of MUC-CT to the ppGalNAc-T5 promoter inhibits the transcription of ppGalNAc-T5. This study suggests that one oncogenic role of MUC1-CT is targeting the tumor suppressor glycosyltransferase GalNAc-T5 and it is the first study to show that MUC1-CT regulates expression of glycosyltransferases in pancreatic cancer cells.

Biography

Prakash Radhakrishnan obtained his Ph.D from University of Madras, India in 2006. His research work under Dr. Michael A Hollingsworth focused on role of Mucin glycans in pancreatic cancer growth and metastasis.