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A pro-inflammatory role for vimentin in the colon

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Inflammatory Bowel Disease (IBD) is most frequently manifested by ulceration and chronic inflammation of the gastrointestinal tract and is associated with increased risk of colorectal cancer. IBD is believed to be caused by an abnormal immune response to commensal bacteria, as well as environmental factors and genetic predisposition. Recent treatments for IBD include anti-inflammatory drugs, which may increase the risk of secondary infections and cancer, thereby increasing the need for preventative care and improved treatment modalities. Two distinct studies of human tissue of gene expression during the course of IBD have shown increased vimentin, expression especially in ulcerative colitis. In addition, interaction between vimentin and nucleotide binding oligomerization domain-containing protein 2 (NOD-2) has been found. Despite these findings,vimentin's function and its role in IBD remain elusive. We now have evidence that suggests a new role for the vimentin protein in intestinal homeostasis. Using *E. coli* and dextran sodium sulfate (DSS)-induced colitis mouse models, we have found that Vim KO mice show significant resistance to the development of acute and chronic colitis, while under steady state conditions Vim KO mice have a remarkable normal phenotype and very subtle abnormalities. Our results suggest that vimentin can suppress the production of reactive oxygen species (ROS) and autophagy. Since ROS production and defects in autophagy are linked tothe pathophysiology of IBD, we concluded that vimentin might contribute to IBD development, and may promote cancer.

Biography

Nirit Mor-Vaknin has completed her PhD at the age of 30 years from the Ben Gurion University of the Negev in Israel and Postdoctoral studies from the University of Michigan School of Medicine. She has published more than 20 papers in reputed journals.

Short activating RNA (saRNA) targeting C/EBPA significantly inhibits cell proliferation of undifferentiated cancer cells

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In general, 'poorly' differentiated tumours have a worse prognosis when compared to more 'well' differentiated ones. Therefore, the use of a biological agent that could promote differentiation might have a therapeutic advantage. CCAAT enhancer binding protein alpha (C/EBPα) is a transcription factor known to be involved in the regulation of cell differentiation in a number of tissue types and has reported to. inhibit the development of hepatocellular carcinoma. Here we report the effect of stimulation of C/EBPα expression by a specific small activating RNA (saRNA) on a panel of cell lines representing both well-differentiated and poorly-differentiated cancer cell types. The C/EBPα-saRNA inhibited proliferation of poorly differentiated small cell lung cancer and pancreatic cancer cell lines compared to treatment with scrambled double-stranded RNA controls. However C/EBPα-saRNA was not as effective in suppressing proliferation in well-differentiated insulinoma and breast cancer (MCF7) cell lines. Comparison of endogenous levels of C/EBPα, using qPCR and Western blots, showed that undifferentiated tumour cell lines expressed lower levels of C/EBPα when compared to the well-differentiated tumour types. Our results suggest that saRNA mediated stimulation of C/EBPα, could be of potential therapeutic value, especially in poorly differentiated cancers. Furthermore, intracellular expression levels of C/EBPα could be an important prognostic factor for predicting the therapeutic response in poor or un-differentiated tumours.