

Study pinpoints microRNA tied to colon cancer tumor growth

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Researchers at the University of Minnesota have identified microRNAs that may cause colon polyps from turning cancerous. The finding could help physicians provide more specialized, and earlier, treatment before colon cancer develops.

The findings are published today in *The Journal of Pathology*.

The American Cancer Society estimates over 134,000 people will be diagnosed with [colon cancer](#) in 2014, despite the expanded screening processes now available. This year alone, about 50,000 people will die because of the disease.

Research was led by Subbaya Subramanian, Ph.D., assistant professor in the Division of Basic and Translational Research in the Department of Surgery in the University of Minnesota Medical School and member of the Masonic Cancer Center, University of Minnesota.

"With the advanced screenings we now have available, why are so many people still being diagnosed with colon cancer? We really wanted to understand if there was a way to stop the disease before it starts, before benign polyps became [cancerous tumors](#)," said Subramanian.

By looking at microRNA, Subramanian and his colleagues hoped to unlock what pieces were present in [colon polyps](#) that developed into cancer. They found miR-182 and miR-503 work together to transform a benign polyp to a cancerous tumor by holding down the cell's ability to

create the tumor suppressing protein FBXW7.

This was determined by looking at a benign polyp cell line. In this line, miR-182 was present and appeared as a feature of the creation of adenomas, or polyps. Researchers then introduced miR-503 to the cell line and noted the partnership limited the tumor suppressing protein and [polyps](#) had a much higher potential for becoming cancerous.

Armed with this knowledge, the researchers then took a closer look at actual patient data. They examined the expression of miR-182 and miR-503 in colon cancer patients with a 12-year survival outcome data. When both microRNAs were present at higher levels, decreased patient survival was clearly correlated.

"It suggests a biomarker for colon cancer patients, something ideally physicians can one day screen for as a diagnostic and prognostic tool," said Subramanian.

Subramanian believes the next step will be determining if drugs are able to target miR-182 and -503, as well as what miR-182 and -503 do after suppressing FBXW7. He hopes to develop a clinical test as well as a translational target for treatments to be utilized in a clinical setting.

Provided by University of Minnesota

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