

# Long non-coding RNA modulates colorectal cancer metabolism

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Long non-coding RNAs (lncRNA) are unusual in that they don't encode proteins like normal RNA. Yet they do play a role in regulating cellular functions and interest cancer researchers.

Scientists at The University of Texas MD Anderson Cancer Center have found that a specific lncRNA called CCAT2 regulates cancer metabolism both in vivo and in vitro. The data was presented on April 20 at the 2015 American Association for Cancer Research (AACR) Annual Meeting in Philadelphia.

"Altered [energy metabolism](#) is a cancer hallmark as malignant cells tailor their metabolic pathways to meet their energy requirements," said George Calin, M.D., Ph.D., professor of Experimental Therapeutics. "In our study, we found that CCAT2 regulated cancer metabolism in an allele-specific manner that appears to match a known risk associated with [colon cancer](#). This study was novel in that it uncovered complex mechanisms of cancer metabolism and regulation controlled by a long non-coding RNA."

Alleles are gene forms that cause genetic traits, and can be specific for disease development. Some alleles are associated with a higher risk for cancer. To better understand how lncRNAs play a role in [cancer metabolism](#), Calin's team, including Roxana Redis, Ph.D., postdoctoral fellow, looked at glutamine, one of the major nutrients that fuel [cellular metabolism](#).

"The pathways that use these nutrients are often changed in cancer," said Redis. "The long non-coding RNA CCAT2 resulted in a newly formed RNA protein complex that regulated the metabolic enzyme glutaminase 1 (GLS1)."

The lncRNA, located near a DNA sequence called SNP known to be associated with an increased risk of colon cancer, accomplishes this by binding to a protein complex called CFIm that regulates genetic splicing of GLS1. This binding displays similarities to known genetic risks tied to SNP, indicating that this stronger interaction may be associated with the higher-risk allele.

"While we are not saying that an increased risk for [cancer](#) results from this interaction, it may contribute to it," said Calin. "More studies are needed to learn more about this new development."

The findings were supported by data in human colorectal samples from multiple cohorts, including data from The Cancer Genome Atlas and multiple animal models.

Provided by University of Texas M. D. Anderson Cancer Center

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