

A surprise 'spark' for pre-cancerous colon polyps

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Researchers at Huntsman Cancer Institute (HCI) at the University of Utah studied the events leading to colon cancer and found that an unexpected protein serves as the "spark" that triggers formation of colon polyps, the precursors to cancerous tumors.

"Our findings will certainly raise some eyebrows," says oncological sciences graduate student Reid Phelps, first author of the study, which will be published Friday, May 15 in the journal *Cell*. "We expect the conventional wisdom regarding colon tumor development to be reconsidered, together with some resistance to our alternative explanation."

The study in zebrafish and human cells discovered that a protein, known as C-terminal binding protein 1, or CTBP1, was the spark that initiated colon polyp formation, not the protein beta-catenin, as previously thought. With this new information, future treatments that prevent [tumor progression](#) can be developed.

The research centered on the mutation of a tumor-suppressor gene called APC - a mutation previously found to be present in 85 percent of all colon cancers. Since then, research labs around the world have developed theories about how the gene works in colon cancer development.

"Our work reveals new information about how the APC protein functions to prevent colon tumor formation. This new information opens

new possibilities treating and preventing colon cancer. " says David Jones, Ph.D., a professor of oncological sciences at the University of Utah and senior director of early translational research at HCI. "We want to know what happens immediately following mutation of the APC gene mutation as a way of understanding how we might intervene. If you're trying to match therapies with a specific genetic mutation, it helps to understand the earliest steps in tumor formation, as well as the downstream consequences."

APC stands for adenomatous polyposis coli. It is classified as a [tumor suppressor gene](#). Before the new study, scientists believed that following APC mutation, faulty cell communication caused by a particular [protein](#) known as beta-catenin resulted in colon polyp formation. Colon polyp formation precedes the development of colon cancer.

While considerable evidence implicates dysregulated Wnt/beta-catenin signaling as the initiating event underlying colon adenoma formation following loss of APC, several studies examining human FAP adenomas raised the possibility that APC loss alone is insufficient to promote aberrant Wnt/beta-catenin signaling. These findings suggest that beta-catenin dependent intestinal cell proliferation may contribute to adenoma progression, rather than initiation following loss of APC.

According to the National Cancer Institute, there are about 108,070 new cases of [colon cancer](#) and 40,740 new cases of rectal cancer each year in the United States. Colon and rectal cancers together claim 49,960 lives in the country annually.

Source: University of Utah ([news](#) : [web](#))

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