Poor survival among colorectal cancer patients tied to biomarker CSN6

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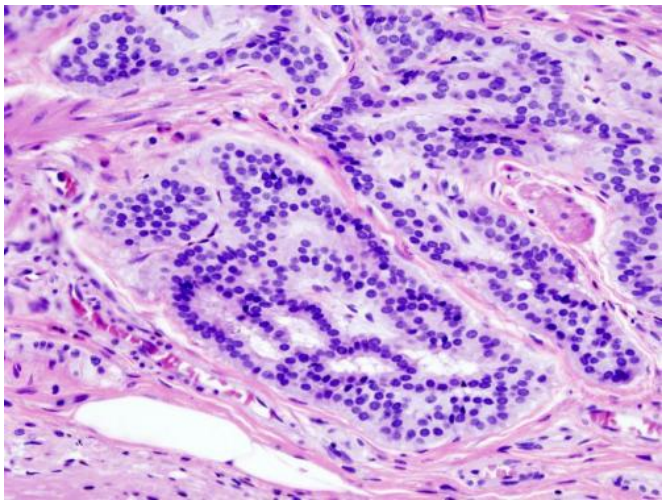

Cancer — Histopathologic image of colonic carcinoid.
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A protein called CSN6 has been found to be correlated with poor survival among patients with colorectal cancer, according to a study at The University of Texas MD Anderson Cancer Center.

The study revealed that CSN6, a subunit of a protein complex known as COP9 signalsome, is overexpressed in colorectal cancer tissue samples. The finding could be significant in the search for alternative treatment strategies for colorectal cancer.

"CSN6 is a biomarker that is elevated in colon cancer and leads to worse recurrence-free survival," said Mong-Hong Lee, Ph.D., professor of Molecular and Cellular Oncology. "This occurs when CSN6 is deregulated through a series of cellular signaling pathways."

The biomarker is normally regulated through signaling pathways called EGFR and ERK. When CSN6 is overexpressed via ERK2 in particular, however, it can lead to deregulation of another protein, beta-catenin, a transcription factor known to be linked to cancer development.

"Our findings indicated that deregulation of CSN6 by ERK2 resulted in stabilization and activation of beta-catenin, which is important for colorectal cancer development," said Lee. "We further defined the mechanism by which beta-catenin is regulated in this cancer."

The team's study findings were published in the Aug. 10, 2015 issue of *Cancer Cell*.

Currently, the standard treatment for colorectal cancer patients at high risk of developing recurrent or metastatic cancer includes surgery, chemotherapy and targeted therapies.

"The molecular alterations in colorectal cancer have been studied extensively," said Lee. "However, a more detailed picture of the pathways deregulated in this cancer has yet to emerge. Defining those molecular alterations can help guide treatment and improve clinical care."

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