Warring molecules keep the colon cancer-free

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A molecular battle is going on inside your colon, and University of Kansas researchers want neither side to win.

KU associate professor of molecular biosciences Kristi Neufeld and her graduate student Erick Spears study how a molecule, a protein called APC, suppresses colon cancer. In a recent article in the Journal of Biological Chemistry, they explain how a drug might someday treat the disease by blocking the action of one of APC's molecular opponents.

Currently, no drug specifically treats colon cancer. The vast majority of cases derive from a faulty gene in intestinal cells that produces a defective APC protein. APC — whose hefty full name is Adenomatous Polyposis Coli — is named after the intestinal polyps it helps prevent. Polyps can turn malignant if not removed by surgery.

"Many researchers are trying to figure out now why this protein is so critical for preventing polyp formation," Neufeld said. "Mine has been one of those labs."

Neufeld's work concerns the health of an astonishingly sophisticated organ. The last part of the digestive system, the colon absorbs water, salt, some nutrients, and keeps symbiotic bacteria in check. Key to its success are stem cells in its lining. These cells reproduce or mature to take different jobs, and then shed when they wear out.

Inside the cells, a governing board of proteins decides whether more cells should reproduce — divide — or take on different jobs —

differentiate. Scientists have previously determined that APC always advises differentiation. At the same time, another protein board member pushes for division. It is named Musashi after a renowned samurai swordsman.

APC and Musashi not only have opposite agendas in the colon, Neufeld and Spears now find, but also actively sabotage each other: behind the scenes, APC controls how many Musashi proteins get made and vice versa. When APC is absent, Musashi in a sense shouts louder, causing cells to proliferate out of control and form polyps and tumors.

The health of the colon requires both Musashi and APC. Restoring APC to people who lack a proper copy of its gene is still out of reach. But a designer drug may be able to subdue Musashi.

“Eighty percent of colon cancers will have a nonfunctioning APC protein. Technology doesn’t allow us to fix that,” Neufeld said. “Keeping Musashi controlled — we can try to do that in another way.”

Next, the team will look for a drug that will inhibit Musashi and will test their hypothesis in mice. The current work was done in cultures of human colon cells.

“Talking to different people, I am struck by how prevalent the disease is,” Neufeld said. “The research that I do is still years away from something that would benefit patients directly. But we’re getting closer. And I do think about how great it would be if something we found in the lab could be translated into a real therapy.”

Provided by University of Kansas

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