Cracking the code: How what you eat might affect your cancer risk

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There is an unresolved debate about the extent to which the environment contributes to cancer risk. Although epidemiological studies suggest that environmental factors such as diet can certainly contribute, especially for

colon cancer, how dietary factors could tip the scale in favor of cancer is not known.

In a study published in *Cancer Research Communications*, a team led by researchers at Baylor College of Medicine reveals a mechanism by which dietary folate enhances colon cancer risk in an animal model.

The findings also highlight the need for monitoring the long-term safety of folate food fortification and resulting cancer-promoting effects, particularly given the rising incidence of early-onset colon cancer in the United States over the past two decades.

"In this study we show a mechanistic pathway from diet to colon cancer in an animal model," said corresponding author Dr. Lanlan Shen, professor of pediatrics—nutrition at Baylor and a member of the Dan L Duncan Comprehensive Cancer Center.

"We investigated whether this pathway involved epigenetics, a system of bookmarking DNA that determines which genes will or will not be expressed in a cell. Epigenetics is one way cells can control the activities of their genes without altering the DNA sequence and is closely linked to the environment."

Cells bookmark genes by adding small chemical modifications to the DNA. Methyl groups are one of these chemical modifications, and folate and other associated nutrients are directly involved in the metabolic pathway leading to DNA methylation. "Understanding this link between our meals and how our genes work is a big deal. It's like finding a missing piece of a puzzle we're getting closer to solving about how to keep our bodies healthy," Shen said.

In the current study, the researchers tested the effect of dietary folate on colon cancer development in their animal model. The team found that

animals on the folate-supplemented diet had significantly shortened overall survival and more tumors as well as larger tumors compared to the animals on the non-supplemented diet.

A closer look at the tumors revealed the presence of tumor-associated macrophages, a type of immune cell infiltration that is clinically associated with immunosuppression and poor prognosis in colorectal cancer patients. The tumors also were highly proliferative.

"Importantly, we observed substantially increased epigenetic methylation of gene p16—a gene involved in colon cancer development—in animals on the supplemented diet compared to controls," Shen said. "These findings illuminate a direct link between dietary folate and accelerated tumor development in the colon."

This study provides valuable insights into how the environment can influence cancer risk, opening new avenues to treat or prevent colon cancer, one of the most common cancers and the second leading cause of cancer death in the United States.

Provided by Baylor College of Medicine

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