

# Study shows how dietary supplement may block cancer cells

29 June 2010

Researchers at the Ohio State University Comprehensive Cancer Center-Arthur G. James Cancer Hospital and Richard J. Solove Research Institute (OSUCCC-James) have discovered how a substance that is produced when eating broccoli and Brussels sprouts can block the proliferation of cancer cells.

Compelling evidence indicates that the substance, indole-3-carbinol (I3C), may have anticancer effects and other health benefits, the researchers say. These findings show how I3C affects cancer cells and normal cells.

The laboratory and animal study discovered a connection between I3C and a molecule called Cdc25A, which is essential for cell division and proliferation. The research showed that I3C causes the destruction of that molecule and thereby blocks the growth of [breast cancer cells](#).

The study was published online June 29 in the journal *Cancer Prevention Research*.

"Cdc25A is present at abnormally high levels in about half of breast cancer cases, and it is associated with a poor prognosis," says study leader Xianghong Zou, assistant professor of pathology at the Ohio State University Medical Center.

The molecule also occurs at abnormally high levels in cancers of the breast, prostate, liver, esophagus, endometrium and colon, and in [non-Hodgkin lymphoma](#), and in other diseases such as Alzheimer's disease, he noted.

"For this reason, a number of anti-Cdc25 agents have been identified, but they have not been successful for cancer prevention or treatment due to concerns about their safety or efficacy," says Zou, who is also a member of the OSUCCC-James Molecular Carcinogenesis and Chemoprevention program.

"I3C can have striking effects on [cancer cells](#)," he explains, "and a better understanding of this mechanism may lead to the use of this dietary supplement as an effective and safe strategy for treating a variety of cancers and other human diseases associated with the overexpression of Cdc25A," Zou says.

For this study, Zou and his colleagues exposed three [breast cancer](#) cell lines to I3C. These experiments revealed that the substance caused the destruction of Cdc25A. They also pinpointed a specific location on that molecule that made it susceptible to I3C, showing that if that location is altered (because of a gene mutation), I3C no longer causes the molecule's destruction.

Last, the investigators tested the effectiveness of I3C in breast tumors in a mouse model. When the substance was given orally to the mice, it reduced tumor size by up to 65 percent. They also showed that I3C had no effect on breast-cell tumors in which the Cdc25A molecule had a mutation in that key location.

Provided by The Ohio State University

APA citation: Study shows how dietary supplement may block cancer cells (2010, June 29) retrieved 21 November 2017 from <https://medicalxpress.com/news/2010-06-dietary-supplement-block-cancer-cells.html>

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