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Treatment may fuel cancer's spread, study finds

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By Maggie Fox, Health and Science Editor

WASHINGTON (Reuters) - Treating cancer with surgery, chemotherapy or radiation may sometimes cause tumors to spread and U.S. researchers said on Thursday they may have nailed down one of the causes -- a compound called TGF-beta.

Tests in mice show that using the chemotherapy drug doxorubicin or radiation both raised levels of TGF-beta, which in turn helped breast cancer tumors spread to the lung.

But using an antibody to block TGF-beta stopped the process, Dr. Carlos Arteaga and colleagues at Vanderbilt University in Tennessee reported.

Developing drugs that block TGF-beta might help prevent cancer from recurring, Arteaga's team reports in the May issue of the Journal of Clinical Investigation.

"The repopulation and progression of tumors after anti-cancer therapy is a well-recognized phenomenon," the researchers wrote. "It has been shown to occur following radiotherapy, chemotherapy, and surgery."

Cancer experts have wondered if the so-called primary tumor -- the first and biggest tumor -- might somehow suppress the growth of other tumors, and that removing or destroying the first tumor might allow other, undetectable, tumors to then grow.

TGF-beta, which is involved in both the growth and suppression of tumors, may hold part of the answer, Arteaga's team said.

When mice infected with human breast cancer cells were treated with radiation or doxorubicin, they had higher levels of TGF-beta in their blood. They also had more tiny tumor cells in their blood, and these cells metastasized, or spread, to the lungs.

When the mice were treated with an antibody that suppresses TGF-beta, the spread stopped. And this spreading process did not occur at all in mice bred to lack the TGF-beta protein.

"We wondered then if TGF-beta induced by anti-cancer therapies can serve as a survival signal for tumor cells, thus allowing them to withstand therapy and later recur," Arteaga said in a statement.

His team is now testing TGF-beta levels in the blood of breast cancer patients.

"We'll be looking to see in what proportion of patients the serum and tumor TGF-beta goes up, and whether the increase correlates with the inability of the therapy to eliminate the cancer in the breast," Arteaga said.

Higher levels of TGF-beta after treatment may be a way to predict which patients are likely to have their cancer come back after treatment, Arteaga said.

His team is also testing drugs that interfere with TGF-beta to see if they improve survival.

"It probably isn't just TGF-beta that is having this effect," Arteaga said. Many other compounds, including some immune system signaling chemicals, are also associated with tumor spread and growth.

"TGF-beta may be just the tip of the iceberg," Arteaga said.