UCSD Cancer Researchers Provide First Direct Evidence Of 2-way Conversations Between Malignant, Normal Cells

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For more than seven decades, scientists have had tantalizing clues that cancer cells and neighboring non-cancerous cells in the body communicate with one another. It now emerges that this dialog may explain the clinical observation that cancer cells grow to make secondary tumors (metastasize) in some organs of the body and not others. Findings published today (Feb. 15) suggest that this may also have therapeutic implications

With the aid of gene chip technology and other powerful new tools, researchers at the Moores Cancer Center at University of California, San Diego (UCSD) have shown clearly that there are two-way conversations taking place that are essential for metastatic cancer cells to form new tumors in distant organs. Further, they have been able to distinguish messages generated by the metastatic cells from those produced by the neighboring non-cancerous cells.

"We now know that metastatic tumor cells do not act alone. They must find the right neighborhood, whose resident cells speak their language and are able to provide the support system necessary for the metastatic cells to survive and form secondary tumors," said David Tarin, M.D., Ph.D., professor of pathology and member of the Moores Cancer Center at University of California, San Diego. Tarin is the principal investigator of the study, published February 15 as an EarlyView article on the *International Journal of Cancer* web site at Wiley Interscience (www.wiley.interscience.com). The DOI (digital object identifier) is 10.1002/ijc.21757.

This new insight into the biology of cancer may alter the way scientists think about how best to attack cancer, suggesting that hindering or blocking a support system provided by the host cells may be more effective than a direct hit to the cancer cells.

Tarin explained that the tumor cell is inherently genetically unstable – or highly variable in its genetic makeup – which means that only certain tumor cells are susceptible to powerful cancer drugs. Surviving cells then multiply more rapidly because they are not competing with as many other tumor cells for oxygen and nutrients. On the other hand, host cells are more genetically stable, or less variable in their genetic makeup, and therefore more vulnerable to attack.

The researchers explored intercellular signaling by grafting human breast cancer cells labeled with a green fluorescent protein, for easy recognition, into the mammary pads of laboratory mice. After the cells formed a breast tumor, the researchers were able to track and study cells that had broken away from the primary tumor to form new tumors in specific, predictable sites – namely the lungs, and lymph nodes.

They also observed single cells in organs where there were no secondary tumor growths (metastases). Using special gene chips constructed to analyze 22,000 genes at once, and specially designed probes, the researchers were able to distinguish the signals generated by normal mouse cells from those generated by the human breast cancer cells, and to document changes in the gene expression pattern in both cell types during the metastatic process. (A probe is a piece of labeled DNA or RNA, or an antibody, used to detect the function of a gene).

"We've known for some time that cancer cells spread everywhere in the body and get into every single organ, but we've not had direct evidence until now as to why they only multiply to make secondary tumors in certain organs," said Tarin. "They do not grow in places where the communication between the tumor cells and the host organ is not favorable and this suggests an 'Achilles heel' that could be targeted. It may sound heretical to suggest attacking normal cells to control the spread of a cancer, but, these new findings suggest that this may be an effective strategy. If we can develop a therapy that targets the support system that the host provides to the metastatic cells, it follows that the metastatic cells will stop growing with no further intervention."

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Co-authors are Valerie Montel and Evangeline Sari Mose, both of the Department of Pathology and the Moores Cancer Center, University of California, San Diego. The research was funded by a Lopiccola Research Fellowship Award.

Founded in 1979, the Moores UCSD Cancer Center is one of just 39 centers in the United States to hold a National Cancer Institute (NCI) designation as a Comprehensive Cancer Center. As such, it ranks among the top centers in the nation conducting basic, translational and clinical cancer research, providing advanced patient care and serving the community through outreach and education programs.

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