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New class of proteins allows breast cancer cells to evade tyrosine kinase inhibitors

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Aberrant regulation of cell growth pathways is required for normal cells to become cancerous, and in many types of cancer, cell growth is driven by a group of enzymes known as receptor tyrosine kinases (RTKs). The RTK epidermal growth factor receptor (EGFR) is overexpressed in over 30% of breast cancers; however, drugs that target RTKs, known as tyrosine kinase inhibitors (TKIs) have not been effective in treating breast cancer. Researchers believe that the cancer cells escape TKIs by circumventing the RTKs and utilizing other enzymes that are not TKI-sensitive.

In the current issue of the Journal of Clinical Investigation, two groups identify a pair of related oncogenes, FAM83A and B, which allow breast cancer cells to survive TKI treatment. Researchers led by Mina Bissell at the Lawrence Berkeley National Laboratory in Berkeley, CA performed a screen of human breast cancer cell lines to identify genes that make cancer cells resistant to EGFR TKIs.

Bissell and colleagues determined that increased expression of FAM83A increases proliferation and invasion, while decreased expression delays tumor growth in mice and renders cancer cells sensitive to TKIs.

At Case Western Reserve Medical School in Cleveland, OH, Mark Jackson and colleagues identified FAM83B as a gene that allows normal human mammary cells to become malignant. Further, expression of FAM83A and B in human tumors was correlated with decreased overall survival. Taken together, these studies identify two genes that may serve

as novel therapeutic targets.

In a companion piece, Steven Grant of the Medical College of Virginia discusses the impact of this research on the development of strategies to overcome resistance to currently available TKIs.

More information:

Identification of FAM83B as a novel intermediary in EGFR/RAS-mediated transformation, *Journal of Clinical Investigation*.

FAM83A confers EGFR-TKI resistance in breast cancer cells and in mice, *Journal of Clinical Investigation*.

FAM83A and FAM83B: candidate oncogenes and TKI resistance mediators, *Journal of Clinical Investigation*.

Provided by Journal of Clinical Investigation

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