

The right combination: Overcoming drug resistance in cancer

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Overactive epidermal growth factor receptor (EGFR) signaling has been linked to the development of cancer. Several drug therapies have been developed to treat these EGFR-associated cancers; however, many patients have developed resistance to these drugs and are therefore no longer responsive to drug treatment.

In a recent research article published in the [Journal of Clinical Investigation](#), Goutham Narla and colleagues at Case Western Reserve University sought to better understand the molecular players in the EGFR signaling pathway in hopes of finding new drug targets for EGFR-associated cancers. Using cancerous human lung tissue and a mouse model of EGFR-associated lung cancer, The Narla team discovered that two [tumor suppressor genes](#), KLF6 and FOXO1, function to disrupt overactive EGFR signaling.

After treating the cancerous lung tissue and cancer-prone mice with an FDA-approved drug called trifluoperazine hydrochloride (TFP), which increases the activity of FOXO1, they restored the effectiveness of the anti-EGFR drug erlotinib and reduced tumor growth.

Their work identified new drug targets for EGFR-associated cancers and suggests that combinatorial drug therapy regimens may improve treatment outcome.

More information: Targeting the FOXO1/KLF6 axis regulates EGFR signaling and treatment response, *Journal of Clinical Investigation*, 2012.

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