

Researchers report a new target to treat prostate cancer

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The drug Gefitinib is used to treat breast, lung, and other cancers by inhibiting epidermal growth factor receptor (EGFR) signaling, but it has only a limited effect on prostate cancer. EGFR, present on the cell membrane, is involved in cell proliferation and the development of dermis, lung, and digestive tissues. When a mutation causes its over-activation, it can lead to increased cell proliferation and tumor formation.

Tadashi Matsuda of Hokkaido University and his colleagues in Japan investigated human [prostate cancer cells](#) to determine if there is an unknown up-regulation mechanism in the EGFR pathway.

When EGFR is attached to a small protein called ubiquitin, it is given "the kiss of death" and tagged for degradation inside the cell. This tagging process is facilitated by a protein called c-CBL. The degradation of EGFR leads to less signaling from the receptor and reduced [cell proliferation](#).

Matsuda and his team found that signal-transducing adaptor protein-2 (STAP-2) stabilizes EGFR by inhibiting its c-CBL-mediated ubiquitination. Furthermore, when the team suppressed STAP-2, the prostate cancer cells showed reduced proliferation and did not form a tumor when transplanted into mice.

"STAP-2 inhibitors could play a role in treating Gefitinib-resistant prostate cancers. Further studies on STAP-2 will provide new insights

into cancer physiology and support the development of anticancer therapies," says Tadashi Matsuda. The study was published in the *Journal of Biological Chemistry*.

More information: Yuichi Kitai et al. STAP-2 protein promotes prostate cancer growth by enhancing epidermal growth factor receptor stabilization, *Journal of Biological Chemistry* (2017). [DOI: 10.1074/jbc.M117.802884](https://doi.org/10.1074/jbc.M117.802884)

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