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EGFR SORTING IN LUNG CANCER: "SMELLS LIKE A SORTILIN SPIRIT"

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The aim of the present project is to obtain a better understanding of EGFR deactivation in lung cancer. To accomplish this we investigated the role of sortilin in EGFR regulation following EGF-induced EGFR internalization. The study also provides evidence that sortilin expression represents a favorable prognostic marker in lung adenocarcinoma patients.

Tyrosine kinase receptors such as the epidermal growth factor receptor (EGFR) transduce information from the microenvironment into the cell and activate homeostatic signaling pathways. Internalization and degradation of EGFR after ligand binding limits the intensity of proliferative signaling, thereby helping to maintain cell integrity. In cancer cells, deregulation of EGFR trafficking has a variety of effects on tumor progression. Here we report that sortilin is a key regulator of EGFR internalization. Loss of sortilin in tumor cells promoted cell proliferation by sustaining EGFR signaling at the cell surface, ultimately accelerating tumor growth. In lung cancer patients, sortilin expression decreased with increased pathologic grade, and expression of sortilin was strongly correlated with survival, especially in patients with high EGFR expression. Sortilin is therefore a regulator of EGFR intracellular trafficking that promotes receptor internalization and limits signaling, which in turn impacts tumor growth.

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