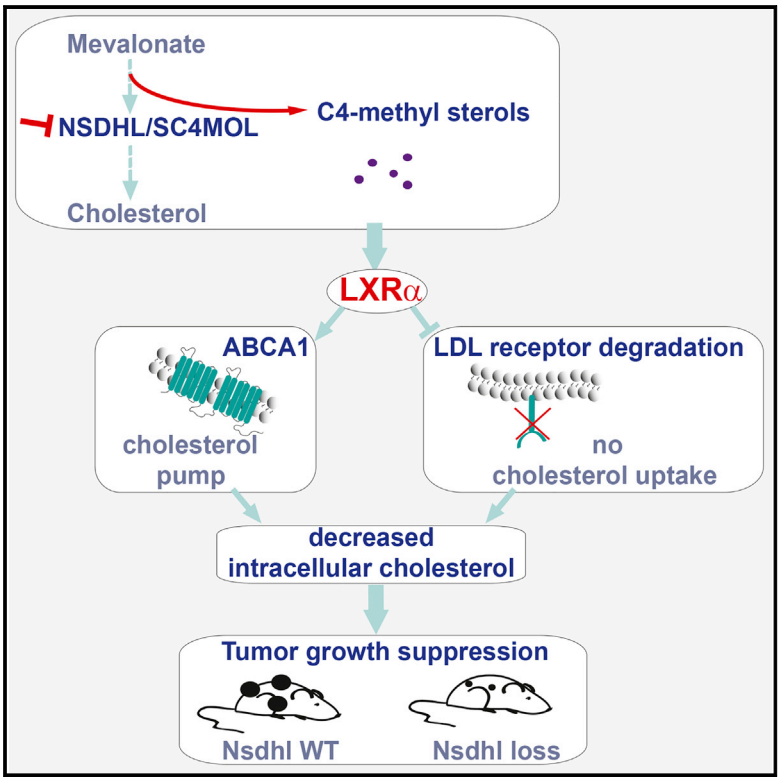
Article

Cell Reports

Endogenous Sterol Metabolites Regulate Growth of EGFR/KRAS-Dependent Tumors via LXR

Graphical Abstract



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In Brief

Cholesterol is a prerequisite for growth of cancer cells. Gabitova et al. show that blockade of cholesterol biosynthesis at the C4-demethylation step results in suppression of tumor growth. Cholesterol blockade leads to the accumulation of sterol metabolites that activate nuclear receptor LXRα and its transcriptional targets, leading to an uncompensated loss of cholesterol.

Highlights

- *NSDHL/SC4MOL* loss induces the expression of LXRα transcriptional targets
- *Nsdhl* inactivation antagonizes the growth of $KRas^{G12D}$-induced mouse skin papillomas
- EGFR inhibitors and LXR agonists synergistically suppress cancer cell growth

Gabitova et al., 2015, Cell Reports *12*, 1927–1938
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