

Fusobacterium nucleatum manipulates host autophagy to promote its intracellular survival and treatment resistance in nasopharyngeal carcinoma

Molecular Cancer

IF 33.9

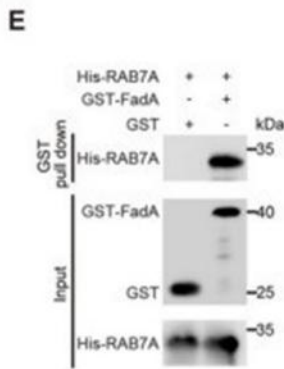
Intratumoral *Fusobacterium nucleatum*, specifically the Fna C2 clade, persists intracellularly in nasopharyngeal carcinoma (NPC) by inhibiting host autophagy. Its virulence factor FadA enhances TRIM28-mediated RAB7A ubiquitination and degradation, disrupting autophagosome-lysosome fusion. This impairs autophagy flux, enabling bacterial survival and conferring treatment resistance. Clinically, high intratumoral *F. nucleatum* correlates with tumor relapse and poor prognosis in NPC patients, underscoring its pathogenic role and potential as a target for microbiota-directed anticancer therapy.

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Cited Products

[YHE80201] Recombinant Human RAB7A Protein, N-His



GST pull-down assays revealing a direct physical interaction between FadA and RAB7A.

Associated Products

[YME80201] Recombinant Mouse RAB7A Protein, N-His
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