

Resource Summary Report

Generated by [kravitz2](#) on Aug 23, 2026

HA-AMOT p130

RRID:Addgene_32821

Type: Plasmid

Proper Citation

RRID:Addgene_32821

Plasmid Information

URL: <http://www.addgene.org/32821>

Proper Citation: RRID:Addgene_32821

Insert Name: AMOT p130

Organism: Homo sapiens

Bacterial Resistance: Ampicillin

Defining Citation: [PMID:21205866](#)

Vector Backbone Description: Backbone Marker:Guan lab; Backbone Size:5400; Vector Backbone:pcDNA3-HA; Vector Types:Mammalian Expression; Bacterial Resistance:Ampicillin

Comments: E5D is present in all Guan lab AMOT p130 cDNAs. According to the depositing lab, it has no effect on function. AMOT p130 contains G858D compared to NP_001106962.1.

Plasmid Name: HA-AMOT p130

Relevant Mutation: E5D

Record Creation Time: 20220422T222142+0000

Record Last Update: 20260815T081344+0000

Ratings and Alerts

No rating or validation information has been found for HA-AMOT p130.

No alerts have been found for HA-AMOT p130.

Data and Source Information

Source: [Addgene](#)

Usage and Citation Metrics

We found 6 mentions in open access literature.

Listed below are recent publications. The full list is available at [kravitz2](#) .

Hennigan RF, et al. (2023) Merlin tumor suppressor function is regulated by PIP2-mediated dimerization. PloS one, 18(2), e0281876.

Park SJ, et al. (2022) Vinexin contributes to autophagic decline in brain ageing across species. Cell death and differentiation, 29(5), 1055.

Kantar D, et al. (2021) MAGI1 inhibits the AMOTL2/p38 stress pathway and prevents luminal breast tumorigenesis. Scientific reports, 11(1), 5752.

Hennigan RF, et al. (2019) Proximity biotinylation identifies a set of conformation-specific interactions between Merlin and cell junction proteins. Science signaling, 12(578).

Van Quickelberghe E, et al. (2018) A protein-protein interaction map of the TNF-induced NF- κ B signal transduction pathway. Scientific data, 5, 180289.

Wang Y, et al. (2017) PKC γ regulates nuclear YAP1 localization and ovarian cancer tumorigenesis. Oncogene, 36(4), 534.