

Resource Summary Report

Generated by [RRID](#) on Aug 31, 2026

pHAGE-PIK3CA-H1047R

RRID:Addgene_116500

Type: Plasmid

Proper Citation

RRID:Addgene_116500

Plasmid Information

URL: <http://www.addgene.org/116500>

Proper Citation: RRID:Addgene_116500

Insert Name: PIK3CA

Organism: Homo sapiens

Bacterial Resistance: Ampicillin

Defining Citation: [PMID:29533785](#)

Vector Backbone Description: Vector Backbone:pHAGE; Vector Types:Lentiviral;
Bacterial Resistance:Ampicillin

Plasmid Name: pHAGE-PIK3CA-H1047R

Relevant Mutation: H1047R

Record Creation Time: 20220422T221610+0000

Record Last Update: 20260829T075232+0000

Ratings and Alerts

No rating or validation information has been found for pHAGE-PIK3CA-H1047R.

No alerts have been found for pHAGE-PIK3CA-H1047R.

Data and Source Information

Source: [Addgene](#)

Usage and Citation Metrics

We found 7 mentions in open access literature.

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

Tandon V, et al. (2025) Dyr726, a brain-penetrant inhibitor of PI3K??, Type III receptor tyrosine kinases, and WNT signaling. bioRxiv : the preprint server for biology.

Marohl T, et al. (2025) PCSK5M452I is a recessive hypomorph exclusive to MCF10DCIS.com cells. Molecular cancer research : MCR.

Kohler KT, et al. (2024) Oncogene activated human breast luminal progenitors contribute basally located myoepithelial cells. Breast cancer research : BCR, 26(1), 183.

Chan CH, et al. (2023) PAK and PI3K pathway activation confers resistance to KRASG12C inhibitor sotorasib. British journal of cancer, 128(1), 148.

Jauhiainen S, et al. (2023) ErbB signaling is a potential therapeutic target for vascular lesions with fibrous component. eLife, 12.

Goldhammer N, et al. (2022) Myoepithelial progenitors as founder cells of hyperplastic human breast lesions upon PIK3CA transformation. Communications biology, 5(1), 219.

Zhou J, et al. (2022) PIK3CA hotspot mutations p. H1047R and p. H1047L sensitize breast cancer cells to thymoquinone treatment by regulating the PI3K/Akt1 pathway. Molecular biology reports, 49(3), 1799.