

Resource Summary Report

Generated by [nidm-terms](#) on Aug 9, 2026

pBabe puro HA PIK3CA

RRID:Addgene_12522

Type: Plasmid

Proper Citation

RRID:Addgene_12522

Plasmid Information

URL: <http://www.addgene.org/12522>

Proper Citation: RRID:Addgene_12522

Insert Name: PIK3CA

Organism: Homo sapiens

Bacterial Resistance: Ampicillin

Defining Citation: [PMID:16339315](#)

Vector Backbone Description: Backbone Marker:Available at Addgene (plasmid #1764); Backbone Size:5150; Vector Backbone:pBabe puro; Vector Types:Mammalian Expression, Retroviral; Bacterial Resistance:Ampicillin

Comments: To construct a hemagglutinin (HA)-tagged allele of p110 alpha, a cDNA clone of human PIK3CA (obtained from the FLEXGene Repository of the Harvard Institute of Proteomics [HIP]) was subcloned into pBabepuro by PCR with primers containing HA-epitope coding sequence with subsequent restriction and ligation. Please note that the original cDNA from HIP differed from the sequence posted on NCBI. This cDNA has an A->G alteration at the 424th nucleotide, which changes the amino acid from Ile -> Val. It could be a SNP. No functional changes have been observed so far in signaling assays.

Plasmid Name: pBabe puro HA PIK3CA

Record Creation Time: 20220422T221656+0000

Record Last Update: 20260808T080652+0000

Ratings and Alerts

No rating or validation information has been found for pBabe puro HA PIK3CA.

No alerts have been found for pBabe puro HA PIK3CA.

Data and Source Information

Source: [Addgene](#)

Usage and Citation Metrics

We found 21 mentions in open access literature.

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

Hassenrück F, et al. (2023) Functional impact and molecular binding modes of drugs that target the PI3K isoform p110?. Communications biology, 6(1), 603.

Lin TY, et al. (2023) Epinephrine inhibits PI3K? via the Hippo kinases. Cell reports, 42(12), 113535.

Candido S, et al. (2022) The PIK3CA H1047R Mutation Confers Resistance to BRAF and MEK Inhibitors in A375 Melanoma Cells through the Cross-Activation of MAPK and PI3K-Akt Pathways. Pharmaceuticals, 14(3).

Prieto-Garcia C, et al. (2022) USP28 enables oncogenic transformation of respiratory cells, and its inhibition potentiates molecular therapy targeting mutant EGFR, BRAF and PI3K. Molecular oncology, 16(17), 3082.

Gjelaj E, et al. (2021) Distinct epithelial-to-mesenchymal transitions induced by PIK3CAH1047R and PIK3CB. Journal of cell science, 134(4).

Nieuwenhuis B, et al. (2020) PI 3-kinase delta enhances axonal PIP3 to support axon regeneration in the adult CNS. EMBO molecular medicine, 12(8), e11674.

Ghezzi C, et al. (2019) A high-throughput screen identifies that CDK7 activates glucose consumption in lung cancer cells. Nature communications, 10(1), 5444.

Schaefer T, et al. (2019) Regulation of glioma cell invasion by 3q26 gene products PIK3CA, SOX2 and OPA1. Brain pathology (Zurich, Switzerland), 29(3), 336.

Razavi P, et al. (2018) The Genomic Landscape of Endocrine-Resistant Advanced Breast Cancers. Cancer cell, 34(3), 427.

McNeill RS, et al. (2018) PIK3CA missense mutations promote glioblastoma pathogenesis, but do not enhance targeted PI3K inhibition. PloS one, 13(7), e0200014.

Romano G, et al. (2018) A Preexisting Rare PIK3CAE545K Subpopulation Confers Clinical Resistance to MEK plus CDK4/6 Inhibition in NRAS Melanoma and Is Dependent on S6K1 Signaling. Cancer discovery, 8(5), 556.

Lin R, et al. (2018) The Dietary Supplement Chondroitin-4-Sulfate Exhibits Oncogene-Specific Pro-tumor Effects on BRAF V600E Melanoma Cells. Molecular cell, 69(6), 923.

Blakely CM, et al. (2017) Evolution and clinical impact of co-occurring genetic alterations in advanced-stage EGFR-mutant lung cancers. *Nature genetics*, 49(12), 1693.

Wang C, et al. (2016) Activated mutant forms of PIK3CA cooperate with RasV12 or c-Met to induce liver tumour formation in mice via AKT2/mTORC1 cascade. *Liver international : official journal of the International Association for the Study of the Liver*, 36(8), 1176.

Gámez B, et al. (2016) Class I PI-3-Kinase Signaling Is Critical for Bone Formation Through Regulation of SMAD1 Activity in Osteoblasts. *Journal of bone and mineral research : the official journal of the American Society for Bone and Mineral Research*, 31(8), 1617.

Henry WS, et al. (2016) LINC00520 is induced by Src, STAT3, and PI3K and plays a functional role in breast cancer. *Oncotarget*, 7(50), 81981.

Nakayama M, et al. (2015) Attenuation of the phosphatidylinositol 3-kinase/Akt signaling pathway by *Porphyromonas gingivalis* gingipains RgpA, RgpB, and Kgp. *The Journal of biological chemistry*, 290(8), 5190.

Bieniasz M, et al. (2015) Preclinical Efficacy of Ron Kinase Inhibitors Alone and in Combination with PI3K Inhibitors for Treatment of sfRon-Expressing Breast Cancer Patient-Derived Xenografts. *Clinical cancer research : an official journal of the American Association for Cancer Research*, 21(24), 5588.

Leavenworth JW, et al. (2015) A p85 α -osteopontin axis couples the receptor ICOS to sustained Bcl-6 expression by follicular helper and regulatory T cells. *Nature immunology*, 16(1), 96.

Brady SW, et al. (2014) Enhanced PI3K p110 α signaling confers acquired lapatinib resistance that can be effectively reversed by a p110 α -selective PI3K inhibitor. *Molecular cancer therapeutics*, 13(1), 60.