

Resource Summary Report

Generated by [kravitz2](#) on Aug 11, 2026

pQCXIH BRGI-Flag

RRID:Addgene_19148

Type: Plasmid

Proper Citation

RRID:Addgene_19148

Plasmid Information

URL: <http://www.addgene.org/19148>

Proper Citation: RRID:Addgene_19148

Insert Name: SWI/SNF related, matrix associated, actin dependent regulator of chromatin, subfamily a, member 4

Organism: Homo sapiens

Bacterial Resistance: Ampicillin

Defining Citation: [PMID:18003620](#)

Vector Backbone Description: Backbone Marker:Clontech; Backbone Size:7200; Vector Backbone:pQCXIH; Vector Types:Mammalian Expression, Retroviral; Bacterial Resistance:Ampicillin

Comments: human BRGI with Flag at C-terminus PCR product cut with SalI then blunted and ligated into blunt BamHI site in vector

Plasmid Name: pQCXIH BRGI-Flag

Record Creation Time: 20220422T222044+0000

Record Last Update: 20260808T081349+0000

Ratings and Alerts

No rating or validation information has been found for pQCXIH BRGI-Flag.

No alerts have been found for pQCXIH BRGI-Flag.

Data and Source Information

Source: [Addgene](#)

Usage and Citation Metrics

We found 5 mentions in open access literature.

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

Yu C, et al. (2024) MEF2B C-terminal mutations enhance transcriptional activity and stability to drive B cell lymphomagenesis. Nature communications, 15(1), 7195.

Li J, et al. (2021) Chromatin Remodelers Interact with Eya1 and Six2 to Target Enhancers to Control Nephron Progenitor Cell Maintenance. Journal of the American Society of Nephrology : JASN, 32(11), 2815.

Gupta M, et al. (2020) BRG1 Loss Predisposes Lung Cancers to Replicative Stress and ATR Dependency. Cancer research, 80(18), 3841.

Fillmore CM, et al. (2015) EZH2 inhibition sensitizes BRG1 and EGFR mutant lung tumours to TopoII inhibitors. Nature, 520(7546), 239.

Ahmed M, et al. (2012) EYA1 and SIX1 drive the neuronal developmental program in cooperation with the SWI/SNF chromatin-remodeling complex and SOX2 in the mammalian inner ear. Development (Cambridge, England), 139(11), 1965.