Resource Summary Report

Generated by PRECISE-TBI on Aug 12, 2026

MSCV JMJD3 mutant

RRID:Addgene_21214
Type: Plasmid

Proper Citation

RRID:Addgene_21214

Plasmid Information

URL: http://www.addgene.org/21214

Proper Citation: RRID:Addgene_21214

Insert Name: JMJD3

Organism: Homo sapiens

Bacterial Resistance: Ampicillin

Defining Citation: PMID:18628393

Vector Backbone Description: Backbone Size:6300; Vector Backbone:pMSCVpuro; Vector Types:Mammalian Expression, Retroviral; Bacterial Resistance:Ampicillin

Plasmid Name: MSCV JMJD3 mutant

Relevant Mutation: H1390A

Record Creation Time: 20220422T222054+0000

Record Last Update: 20260808T081408+0000

Ratings and Alerts

No rating or validation information has been found for MSCV JMJD3 mutant.

No alerts have been found for MSCV JMJD3 mutant.

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 PRECISE-TBI .

Mitra S, et al. (2024) A Novel Role for the Histone Demethylase JMJD3 in Mediating Heroin-Induced Relapse-Like Behaviors. Biological psychiatry.

Yang M, et al. (2022) KDM6B promotes PARthanatos via suppression of O6-methylguanine DNA methyltransferase repair and sustained checkpoint response. Nucleic acids research, 50(11), 6313.

van Gils N, et al. (2022) Targeting histone methylation to reprogram the transcriptional state that drives survival of drug-tolerant myeloid leukemia persisters. iScience, 25(9), 105013.

Yang L, et al. (2019) Histone demethylase KDM6B has an anti-tumorigenic function in neuroblastoma by promoting differentiation. Oncogenesis, 8(1), 3.

Al Labban D, et al. (2018) Notch-effector CSL promotes squamous cell carcinoma by repressing histone demethylase KDM6B. The Journal of clinical investigation, 128(6), 2581.

Sherry-Lynes MM, et al. (2017) Regulation of the JMJD3 (KDM6B) histone demethylase in glioblastoma stem cells by STAT3. PloS one, 12(4), e0174775.

Benyoucef A, et al. (2016) UTX inhibition as selective epigenetic therapy against TAL1-driven T-cell acute lymphoblastic leukemia. Genes & development, 30(5), 508.