

Resource Summary Report

Generated by [RRID](#) on Aug 21, 2026

CRISPRoff-v2.1

RRID:Addgene_167981

Type: Plasmid

Proper Citation

RRID:Addgene_167981

Plasmid Information

URL: <http://www.addgene.org/167981>

Proper Citation: RRID:Addgene_167981

Insert Name: CRISPRoff-v2.1 (DNMT3A-DNMT3L-dCas9-BFP-KRAB)

Organism: Homo sapiens

Bacterial Resistance: Ampicillin

Defining Citation: [PMID:33838111](#)

Vector Backbone Description: Backbone Size:4836; Vector Backbone:CAG expression plasmid; Vector Types:Mammalian Expression; Bacterial Resistance:Ampicillin

Plasmid Name: CRISPRoff-v2.1

Record Creation Time: 20220422T221943+0000

Record Last Update: 20260815T080949+0000

Ratings and Alerts

No rating or validation information has been found for CRISPRoff-v2.1.

No alerts have been found for CRISPRoff-v2.1.

Data and Source Information

Source: [Addgene](#)

Usage and Citation Metrics

We found 20 mentions in open access literature.

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

Lin J, et al. (2026) AAVLINK: A potent DNA-recombination method for large cargo delivery in gene therapy. *Cell*, 189(3), 969.

Waldo JJ, et al. (2025) Durable HTT silencing using non-evolved dCas9 epigenome editors in patient-derived cells. *Molecular therapy. Nucleic acids*, 36(2), 102561.

Liesenfelder S, et al. (2025) Epigenetic editing at individual age-associated CpGs affects the genome-wide epigenetic aging landscape. *Nature aging*, 5(6), 997.

Pattali RK, et al. (2025) CRISPRoff epigenome editing for programmable gene silencing in human cell lines and primary T cells. *Methods in enzymology*, 712, 517.

Guo D, et al. (2025) The ZBTB24-CDCA7-HELLS axis suppresses the totipotent 2C-like reprogramming by maintaining Dux methylation and repression. *Nucleic acids research*, 53(7).

Bell HW, et al. (2025) Removal of promoter CpG methylation by epigenome editing reverses HBG silencing. *Nature communications*, 16(1), 6919.

Gu S, et al. (2025) Elucidating the genetic mechanisms governing cytosine base editing outcomes through CRISPRi screens. *Nature communications*, 16(1), 4685.

Xu D, et al. (2025) Programmable epigenome editing by transient delivery of CRISPR epigenome editor ribonucleoproteins. *Nature communications*, 16(1), 7948.

Allen TP, et al. (2025) dFLASH; dual FLuorescent transcription factor activity sensor for histone integrated live-cell reporting and high-content screening. *Nature communications*, 16(1), 3298.

Kozlova A, et al. (2025) PICALM Alzheimer's risk allele causes aberrant lipid droplets in microglia. *Nature*, 646(8087), 1178.

Sarno F, et al. (2025) Epigenetic editing and epi-drugs: a combination strategy to simultaneously target KDM4 as a novel anticancer approach. *Clinical epigenetics*, 17(1), 105.

Weber R, et al. (2024) Epigenetic modification and characterization of the MGMT promoter region using CRISPRoff in glioblastoma cells. *Frontiers in oncology*, 14, 1342114.

He W, et al. (2024) Epigenetic editing of BRCA1 promoter increases cisplatin and olaparib sensitivity of ovarian cancer cells. *Epigenetics*, 19(1), 2357518.

Li X, et al. (2024) Chromatin context-dependent regulation and epigenetic manipulation of prime editing. *Cell*, 187(10), 2411.

Kozlova A, et al. (2024) Alzheimer's disease risk allele of PICALM causes detrimental lipid droplets in microglia. *Research square*.

Kawa Y, et al. (2024) Epigenome editing revealed the role of DNA methylation of T-DMR/CpG island shore on Runx2 transcription. *Biochemistry and biophysics reports*, 38, 101733.

Han X, et al. (2023) Downregulation of MGMT expression by targeted editing of DNA methylation enhances temozolomide sensitivity in glioblastoma. *Neoplasia* (New York, N.Y.), 44, 100929.

O'Geen H, et al. (2022) Determinants of heritable gene silencing for KRAB-dCas9 + DNMT3 and Ezh2-dCas9 + DNMT3 hit-and-run epigenome editing. *Nucleic acids research*, 50(6), 3239.

Sarno F, et al. (2022) Functional Validation of the Putative Oncogenic Activity of PLAU. *Biomedicines*, 11(1).

Shi Y, et al. (2022) Nuclear receptor ligand screening in an iPSC-derived in vitro blood-brain barrier model identifies new contributors to leptin transport. *Fluids and barriers of the CNS*, 19(1), 77.