

Resource Summary Report

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pdCas9-DNMT3A-EGFP (ANV)

RRID:Addgene_71685

Type: Plasmid

Proper Citation

RRID:Addgene_71685

Plasmid Information

URL: <http://www.addgene.org/71685>

Proper Citation: RRID:Addgene_71685

Insert Name: S. pyogenes dCas9 fused with the inactivated (E756A) catalytic domain of human DNMT3A (amino acids P602-V912) and T2A-EGFP

Organism: Homo sapiens

Bacterial Resistance: Ampicillin

Defining Citation: [PMID:26969735](#)

Vector Backbone Description: Backbone Size:4246; Vector Backbone:PX462; Vector Types:Mammalian Expression, CRISPR; Bacterial Resistance:Ampicillin

Comments: The catalytic domain of human DNMT3A (amino acids P602-V912) was derived from the plasmid pcDNA3/Myc-DNMT3A (Addgene, plasmid #35521) (Chen et al., 2005, J Cell Biochem 95: 902-917). Undesired BbsI restriction site was removed by site-directed mutagenesis, without affecting the amino acid sequence. The DNMT3A active site motif ENV was mutated to ANV (E756A). Plasmid pSpCas9n(BB)-2A-Puro (PX462) (Addgene, plasmid #48141) (Ran et al., 2013, Nat Protoc 8: 2281-2308) was used as a backbone. Additional H840A mutation was introduced into Cas9n D10A nickase. T2A-PuroR EcoRI fragment was replaced with T2A-EGFP fragment from pSpCas9n(BB)-2A-GFP (PX461) (Addgene, plasmid #48140) (Ran et al., 2013, Nat Protoc 8: 2281-2308).

Plasmid Name: pdCas9-DNMT3A-EGFP (ANV)

Relevant Mutation: D10A and H840A in S.pyogenes Cas9; E756A inactivating mutation in H. sapiens DNMT3A

Record Creation Time: 20220422T222441+0000

Record Last Update: 20260829T080945+0000

Ratings and Alerts

No rating or validation information has been found for pdCas9-DNMT3A-EGFP (ANV).

No alerts have been found for pdCas9-DNMT3A-EGFP (ANV).

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 [nidm-terms](#) .

Kehayova YS, et al. (2023) Mediation of the Same Epigenetic and Transcriptional Effect by Independent Osteoarthritis Risk-Confering Alleles on a Shared Target Gene, COLGALT2. Arthritis & rheumatology (Hoboken, N.J.), 75(6), 910.

Truong VA, et al. (2022) Bi-directional gene activation and repression promote ASC differentiation and enhance bone healing in osteoporotic rats. Molecular therapy : the journal of the American Society of Gene Therapy, 30(1), 92.

Singh K, et al. (2022) Genome-wide DNA hypermethylation opposes healing in patients with chronic wounds by impairing epithelial-mesenchymal transition. The Journal of clinical investigation, 132(17).

Li M, et al. (2021) Regulation of MYB by distal enhancer elements in human myeloid leukemia. Cell death & disease, 12(2), 223.

Qian P, et al. (2018) Retinoid-Sensitive Epigenetic Regulation of the Hoxb Cluster Maintains Normal Hematopoiesis and Inhibits Leukemogenesis. Cell stem cell, 22(5), 740.

Galonska C, et al. (2018) Genome-wide tracking of dCas9-methyltransferase footprints. Nature communications, 9(1), 597.

Xiong T, et al. (2017) Targeted DNA methylation in human cells using engineered dCas9-methyltransferases. Scientific reports, 7(1), 6732.