Resource Summary Report

Generated by RRID on Sep 9, 2026

OCI-AML-3

RRID:CVCL_1844
Type: Cell Line

Proper Citation

RRID:CVCL_1844

Cell Line Information

URL: https://web.expasy.org/cellosaurus/CVCL_1844

Proper Citation: RRID:CVCL_1844

Description: Cell line OCI-AML-3 is a Cancer cell line with a species of origin Homo sapiens (Human)

Sex: Male

Disease: Adult acute myelomonocytic leukemia

Defining Citation: PMID:2538684, PMID:12393637, PMID:16079892, PMID:16408098, PMID:21552520, PMID:22460905, PMID:25485619, PMID:25877200, PMID:25984343, PMID:26434589, PMID:26589293, PMID:27397505, PMID:28109323, PMID:29491412, PMID:30285677, PMID:30629668, PMID:30894373, PMID:30971826, PMID:31068700, PMID:31160637, PMID:31739141, PMID:36982453

Comments: Population: Caucasian., Part of: LL-100 blood cancer cell line panel., Part of: COSMIC cell lines project., Part of: Cancer Dependency Map project (DepMap) (includes Cancer Cell Line Encyclopedia - CCLE).

Category: Cancer cell line

Organism: Homo sapiens (Human)

Name: OCI-AML-3

Synonyms: OCI-Aml-3, OCI/AML-3, OCI-AML3, OCI/AML3, OCI AML3, OCIAML3, Ontario Cancer Institute-Acute Myeloid Leukemia-3

Cross References: BTO:BTO_0004620, CLO:CLO_0009853, EFO:EFO_0006289, ArrayExpress:E-MTAB-2706, ArrayExpress:E-MTAB-2770, ArrayExpress:E-MTAB-3610, ArrayExpress:E-MTAB-7721, ArrayExpress:E-MTAB-7722, BioGRID_ORCS_Cell_line:214, BioSample:SAMN03473107, BioSample:SAMN10988470,

cancercelllines:CVCL_1844, CCTCC:GDC0216, Cell_Model_Passport:SIDM00462, ChEMBL-Cells:CHEMBL4295392, ChEMBL-Targets:CHEMBL4296479, Cosmic:787467, Cosmic:975289, Cosmic:1012110, Cosmic:1127258, Cosmic:1245831, Cosmic:1319546, Cosmic:1557602, Cosmic:1582387, Cosmic:1696140, Cosmic:1779129, Cosmic:2131552, Cosmic:2306232, Cosmic:2750869, Cosmic-CLP:1290455, DepMap:ACH-000336, DSMZ:ACC-582, DSMZCellDive:ACC-582, EGA:EGAS00001000610, EGA:EGAS00001000978, GDSC:1290455, GEO:GSM482512, GEO:GSM887469, GEO:GSM888549, GEO:GSM1446756, GEO:GSM1525022, GEO:GSM1670296, GEO:GSM2716711, GEO:GSM2716712, GEO:GSM2716774, GEO:GSM2716775, GEO:GSM2716776, GEO:GSM2716777, IARC_TP53:30132, LiGeA:CCLE_569, LINCS_LDP:LCL-1066, Lonza:530, PharmacoDB:OCIAML3_1184_2019, Progenetix:CVCL_1844, PubChem_Cell_line:CVCL_1844, Ubigene:YC-C022, Wikidata:Q54931742

ID: CVCL_1844

Record Creation Time: 20220427T221355+0000

Record Last Update: 20260905T181949+0000

Ratings and Alerts

No rating or validation information has been found for OCI-AML-3.

No alerts have been found for OCI-AML-3.

Data and Source Information

Source: Cellosaurus

Usage and Citation Metrics

We found 291 mentions in open access literature.

Listed below are recent publications. The full list is available at RRID .

Barral L, et al. (2026) Targeting BMP and TAZ/TEAD mechanotransduction pathways impairs acute myeloid leukemia chemoresistance. Leukemia, 40(6), 1151.

López-Hernández L, et al. (2026) BCLAF1 links RNA splicing to ATF4-dependent metabolic adaptation in acute myeloid leukemia. bioRxiv : the preprint server for biology.

Jones L, et al. (2026) A structure-based modeling approach identifies effective drug combinations for RAS-mutant acute myeloid leukemia. iScience, 29(8), 116875.

Uible EE, et al. (2026) Scaffolding-dependent CASP1 constrains excessive cell-intrinsic inflammatory signaling in leukemia. Cell chemical biology, 33(1), 59.

Pereira-Martins DA, et al. (2026) ?Np73 isoform defines a TP53-mutant-like poor-risk subgroup of acute myeloid leukemia. Cell reports. Medicine, 102540.

Adhikary U, et al. (2026) Cancer Susceptibility to Stapled Oncolytic Peptides Is Dictated by Membrane Cholesterol and Inflammatory Signaling. Cancer research, 86(11), 2810.

Lewis AC, et al. (2026) Inhibition of heme biosynthesis triggers cuproptosis in acute myeloid leukemia. Cell, 189(1), 215.

Clayfield L, et al. (2026) Retinal determination network reactivation drives chemoresistance and blocks myeloid differentiation in acute myeloid leukemia. Cell reports, 45(2), 116875.

Zhang W, et al. (2026) The interaction between NPMc+ and Orai1 induces abnormal calcium influx to facilitate leukemogenesis. The FEBS journal.

Fang C, et al. (2026) Base Editing of TIGIT Reprograms CD155 Signaling in Natural Killer Cells to Enhance Cancer Immunotherapy Efficacy. Cancer research, 86(8), 1956.

Waclawiczek A, et al. (2026) Leukemic stem cell subtypes determine venetoclax resistance and therapeutic vulnerabilities in AML. Cell stem cell, 33(6), 982.

Jin K, et al. (2026) LDLRAD2 drives glycolysis and angiogenesis to promote extramedullary infiltration in acute myeloid leukemia. iScience, 29(6), 115987.

Tin E, et al. (2025) SOCS1 Protects Acute Myeloid Leukemia against Allogeneic T Cell-Mediated Cytotoxicity. Blood cancer discovery, 6(3), 217.

Devlin JR, et al. (2025) A CDK11-dependent RNA polymerase II pause-checkpoint precedes CDK9-mediated transition to transcriptional elongation. Molecular cell, 85(17), 3256.

Giovannini S, et al. (2025) Thioredoxin-interacting protein (TXNIP) is a substrate of the NEDD4-like E3 ubiquitin-protein ligase WWP1 in cellular redox state regulation of acute myeloid leukemia cells. Molecular oncology, 19(1), 133.

Peled A, et al. (2025) BKT300: A Novel Anti-Leukemic Small Molecule Targeting the Protein Regulator of Cytokinesis 1 (PRC1) Pathway. Research square.

Fiskus W, et al. (2025) Superior preclinical efficacy of co-treatment with BRG1/BRM and FLT3 inhibitor against AML cells with FLT3 mutations. Blood cancer journal, 15(1), 40.

Shang K, et al. (2025) CD97-directed CAR-T cells with enhanced persistence eradicate acute myeloid leukemia in diverse xenograft models. Cell reports. Medicine, 6(6), 102148.

Schmitt N, et al. (2025) The bispecific innate cell engager AFM28 eliminates CD123+ leukemic stem and progenitor cells in AML and MDS. Nature communications, 16(1), 7793.

Hong J, et al. (2025) PSPC1 exerts an oncogenic role in AML by regulating a leukemic transcription program in cooperation with PU.1. Cell stem cell, 32(3), 463.