

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,

cancer cell lines: 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, PharmacDB:OCIAML3_1184_2019, Progenetix:CVCL_1844, PubChem_Cell_line:CVCL_1844, Ubigen: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.

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*.

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

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.

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

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

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.

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

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.

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.

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.

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.