Resource Summary Report

Generated by RRID on Aug 11, 2026

Bcl-w (31H4) Rabbit mAb

RRID:AB_10691557
Type: Antibody

Proper Citation

Cell Signaling Technology Cat# 2724, RRID:AB_10691557

Antibody Information

URL: http://antibodyregistry.org/AB_10691557

Proper Citation: Cell Signaling Technology Cat# 2724, RRID:AB_10691557

Target Antigen: BCL2L2

Host Organism: rabbit

Clonality: monoclonal

Comments: Applications: W. Consolidation on 11/2018: AB_10691557, AB_2259215.

Antibody Name: Bcl-w (31H4) Rabbit mAb

Description: This monoclonal targets BCL2L2

Target Organism: rat, mouse, human

Antibody ID: AB_10691557

Vendor: Cell Signaling Technology

Catalog Number: 2724

Record Creation Time: 20250820T045850+0000

Record Last Update: 20250820T202723+0000

Ratings and Alerts

No rating or validation information has been found for Bcl-w (31H4) Rabbit mAb.

No alerts have been found for Bcl-w (31H4) Rabbit mAb.

Data and Source Information

Source: Antibody Registry

Usage and Citation Metrics

We found 13 mentions in open access literature.

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

Perurena N, et al. (2026) EZH2 Inhibitors Sensitize Breast Cancer to HER2 Kinase Inhibitors through Cooperative Effects on YAP and Proapoptotic Regulators. Cancer research, 86(6), 1467.

Uprety A, et al. (2026) SETD2 Deficiency Drives Mitochondrial DNA Leakage and Creates a Druggable Dependency on BCL-xL in Clear Cell Renal Cell Carcinoma. Cancer research, 86(15), 3838.

Hirani R, et al. (2025) BCL2 drives castration resistance in castration-sensitive prostate cancer by orchestrating reciprocal crosstalk between oncogenic pathways. Cell reports, 44(6), 115779.

Qin X, et al. (2025) Paclitaxel-induced mitotic arrest results in a convergence of apoptotic dependencies that can be safely exploited by BCL-XL degradation to overcome cancer chemoresistance. bioRxiv : the preprint server for biology.

Newman CE, et al. (2025) Inhibition of oligomeric BAX by an anti-apoptotic dimer. Cell, 188(26), 7397.

Tang SX, et al. (2024) Dissecting the neuroprotective interaction between the BH4 domain of BCL-w and the IP3 receptor. Cell chemical biology, 31(10), 1815.

Neault M, et al. (2023) CBFA2T3-GLIS2-dependent pediatric acute megakaryoblastic leukemia is driven by GLIS2 and sensitive to navitoclax. Cell reports, 42(9), 113084.

Ming Z, et al. (2023) IFN-?? Signaling Sensitizes Melanoma Cells to BH3 Mimetics. The Journal of investigative dermatology, 143(7), 1246.

Bolaender A, et al. (2021) Chemical tools for epichaperome-mediated interactome dysfunctions of the central nervous system. Nature communications, 12(1), 4669.

Vigneswaran K, et al. (2021) YAP/TAZ Transcriptional Coactivators Create Therapeutic Vulnerability to Verteporfin in EGFR-mutant Glioblastoma. Clinical cancer research : an official journal of the American Association for Cancer Research, 27(5), 1553.

Kurppa KJ, et al. (2020) Treatment-Induced Tumor Dormancy through YAP-Mediated Transcriptional Reprogramming of the Apoptotic Pathway. Cancer cell, 37(1), 104.

Morrison K, et al. (2019) The Oncogenic Kaposi's Sarcoma-Associated Herpesvirus Encodes a Mimic of the Tumor-Suppressive miR-15/16 miRNA Family. Cell reports, 29(10), 2961.

Pease-Raissi SE, et al. (2017) Paclitaxel Reduces Axonal Bclw to Initiate IP3R1-Dependent Axon Degeneration. Neuron, 96(2), 373.