Resource Summary Report

Generated by RRID on Aug 10, 2026

Rabbit Anti-NUP98 (L205) Polyclonal Antibody, Unconjugated

RRID:AB_561204
Type: Antibody

Proper Citation

Cell Signaling Technology Cat# 2288, RRID:AB_561204

Antibody Information

URL: http://antibodyregistry.org/AB_561204

Proper Citation: Cell Signaling Technology Cat# 2288, RRID:AB_561204

Target Antigen: NUP98 (L205)

Host Organism: rabbit

Clonality: polyclonal

Comments: Applications: W. Consolidation on 10/2018: AB_10693602, AB_561204.

Antibody Name: Rabbit Anti-NUP98 (L205) Polyclonal Antibody, Unconjugated

Description: This polyclonal targets NUP98 (L205)

Target Organism: monkey, rat, simian, mouse, human

Antibody ID: AB_561204

Vendor: Cell Signaling Technology

Catalog Number: 2288

Record Creation Time: 20250820T004553+0000

Record Last Update: 20250820T090834+0000

Ratings and Alerts

No rating or validation information has been found for Rabbit Anti-NUP98 (L205) Polyclonal Antibody, Unconjugated.

No alerts have been found for Rabbit Anti-NUP98 (L205) Polyclonal Antibody, Unconjugated.

Data and Source Information

Source: Antibody Registry

Usage and Citation Metrics

We found 6 mentions in open access literature.

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

Dubey SK, et al. (2025) Aberrant nuclear pore complex degradation contributes to neurodegeneration in VCP disease. Neuron.

Kirkiz E, et al. (2025) Harnessing the E3 ligase SPOP for targeted degradation of the NUP98::KDM5A fusion oncoprotein. Cell reports, 44(12), 116602.

Umeda M, et al. (2025) Fusion oncoproteins and cooperating mutations define disease phenotypes in NUP98-rearranged leukemia. medRxiv : the preprint server for health sciences.

Wang HC, et al. (2024) Kmt2c restricts G-CSF-driven HSC mobilization and granulocyte production in a methyltransferase-independent manner. Cell reports, 43(8), 114542.

Dubey SK, et al. (2022) Nucleoporins are degraded via upregulation of ESCRT-III/Vps4 complex in Drosophila models of C9-ALS/FTD. Cell reports, 40(12), 111379.

Chen R, et al. (2021) Kmt2c mutations enhance HSC self-renewal capacity and convey a selective advantage after chemotherapy. Cell reports, 34(7), 108751.