Resource Summary Report

Generated by RRID on Aug 5, 2026

CDC37 (D11A3) XP Rabbit mAb

RRID:AB_10695539
Type: Antibody

Proper Citation

Cell Signaling Technology Cat# 4793, RRID:AB_10695539

Antibody Information

URL: http://antibodyregistry.org/AB_10695539

Proper Citation: Cell Signaling Technology Cat# 4793, RRID:AB_10695539

Target Antigen: CDC37 (D11A3) XP Rabbit mAb

Host Organism: rabbit

Clonality: monoclonal

Comments: Applications: W, IP, IF-IC. Consolidation: AB_10698884.

Antibody Name: CDC37 (D11A3) XP Rabbit mAb

Description: This monoclonal targets CDC37 (D11A3) XP Rabbit mAb

Target Organism: rat, h, m, mouse, r, non-human primate, human, mk

Antibody ID: AB_10695539

Vendor: Cell Signaling Technology

Catalog Number: 4793

Alternative Catalog Numbers: 4793S, 4793P

Record Creation Time: 20250820T003703+0000

Record Last Update: 20250820T084432+0000

Ratings and Alerts

No rating or validation information has been found for CDC37 (D11A3) XP Rabbit mAb.

No alerts have been found for CDC37 (D11A3) XP Rabbit mAb.

Data and Source Information

Source: Antibody Registry

Usage and Citation Metrics

We found 15 mentions in open access literature.

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

Bay S, et al. (2025) Systems-Level Interactome Mapping Reveals Actionable Protein Network Dysregulation Across the Alzheimer's Disease Spectrum. Research square.

Bay S, et al. (2024) Synthesis and Characterization of Click Chemical Probes for Single-Cell Resolution Detection of Epichaperomes in Neurodegenerative Disorders. Biomedicines, 12(6).

McNutt SW, et al. (2024) Phosphorylation-Driven Epichaperome Assembly: A Critical Regulator of Cellular Adaptability and Proliferation. Research square.

Kumagai H, et al. (2024) MOTS-c modulates skeletal muscle function by directly binding and activating CK2. iScience, 27(11), 111212.

Sharma S, et al. (2023) Unraveling the Mechanism of Epichaperome Modulation by Zelavespib: Biochemical Insights on Target Occupancy and Extended Residence Time at the Site of Action. Biomedicines, 11(10).

Backe SJ, et al. (2023) Activation of autophagy depends on Atg1/Ulk1-mediated phosphorylation and inhibition of the Hsp90 chaperone machinery. Cell reports, 42(7), 112807.

Rodina A, et al. (2023) Systems-level analyses of protein-protein interaction network dysfunctions via epichaperomics identify cancer-specific mechanisms of stress adaptation. Nature communications, 14(1), 3742.

Li L, et al. (2021) Discovery of a covalent inhibitor of heat shock protein 90 with antitumor activity that blocks the co-chaperone binding via C-terminal modification. Cell chemical biology, 28(10), 1446.

Joshi S, et al. (2021) Pharmacologically controlling protein-protein interactions through epichaperomes for therapeutic vulnerability in cancer. Communications biology, 4(1), 1333.

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

Lee SB, et al. (2020) Proline Hydroxylation Primes Protein Kinases for Autophosphorylation and Activation. Molecular cell, 79(3), 376.

Lackie RE, et al. (2020) Modulation of hippocampal neuronal resilience during aging by the Hsp70/Hsp90 co-chaperone STI1. Journal of neurochemistry, 153(6), 727.

Rahnel H, et al. (2017) A Selective Biligand Inhibitor of CK2 Increases Caspase-3 Activity in Cancer Cells and Inhibits Platelet Aggregation. ChemMedChem, 12(20), 1723.

Woodford MR, et al. (2016) The FNIP co-chaperones decelerate the Hsp90 chaperone cycle and enhance drug binding. Nature communications, 7, 12037.

Lee MN, et al. (2013) Identification of regulators of the innate immune response to cytosolic DNA and retroviral infection by an integrative approach. Nature immunology, 14(2), 179.