

Resource Summary Report

Generated by [RRID](#) on Jul 31, 2026

WRN (8H3) Mouse mAb

RRID:AB_10692114

Type: Antibody

Proper Citation

Cell Signaling Technology Cat# 4666, RRID:AB_10692114

Antibody Information

URL: http://antibodyregistry.org/AB_10692114

Proper Citation: Cell Signaling Technology Cat# 4666, RRID:AB_10692114

Target Antigen: WRN (8H3) Mouse mAb

Host Organism: mouse

Clonality: monoclonal

Comments: Applications: W

Antibody Name: WRN (8H3) Mouse mAb

Description: This monoclonal targets WRN (8H3) Mouse mAb

Target Organism: h, m, mouse, human

Antibody ID: AB_10692114

Vendor: Cell Signaling Technology

Catalog Number: 4666

Record Creation Time: 20250819T205207+0000

Record Last Update: 20250819T210510+0000

Ratings and Alerts

No rating or validation information has been found for WRN (8H3) Mouse mAb.

No alerts have been found for WRN (8H3) Mouse mAb.

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](#) .

Ortega R, et al. (2026) PARP1 and PARP2 are dispensable for DNA repair by microhomology-mediated end-joining at double-ended DSBs. Nucleic acids research, 54(1).

Ortega R, et al. (2025) PARP1 and PARP2 are dispensable for DNA repair by microhomology-mediated end-joining during mitosis. bioRxiv : the preprint server for biology.

Xiang S, et al. (2024) Identification of ATP-Competitive Human CMG Helicase Inhibitors for Cancer Intervention that Disrupt CMG-Replisome Function. Molecular cancer therapeutics, 23(11), 1568.

Xiang S, et al. (2023) Identification of Selective ATP-Competitive CMG Helicase Inhibitors for Cancer Intervention that Disrupt CMG-Replisome Function. Research square.

Lieb S, et al. (2019) Werner syndrome helicase is a selective vulnerability of microsatellite instability-high tumor cells. eLife, 8.

Pal D, et al. (2017) TGF-?? reduces DNA ds-break repair mechanisms to heighten genetic diversity and adaptability of CD44+/CD24- cancer cells. eLife, 6.