Resource Summary Report

Generated by NIF on Apr 24, 2025

PhosphoSitePlus: Protein Modification Site

RRID:SCR 001837

Type: Tool

Proper Citation

PhosphoSitePlus: Protein Modification Site (RRID:SCR_001837)

Resource Information

URL: http://www.phosphosite.org

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Description: A freely accessible on-line systems biology resource devoted to all aspects of protein modification, as well as other post-translational modifications. It provides valuable and unique tools for both cell biologists and mass spectroscopists. PhosphoSite is a human-and mouse-centric database. It includes features such as: viewing the locations of modified residues on molecular models; browsing and searching MS2 records by disease, tissue, and cell line; submitting lists of peptides to identify previously reported genes; searching by subcellular localization, treatment, tissues, cell types, cell lines and diseases, and protein types and protein domains; searching for experimentally-verified kinase substrates and viewing preferred substrate motifs; and viewing MS2 spectra for peptides and sites not previously published.

Abbreviations: PSP

Synonyms: PhosphoSitePlus, PhosphoSite

Resource Type: data or information resource, knowledge environment resource, portal

Defining Citation: PMID:22135298

Keywords: portal, mass spectroscopist, molecular model, mouse, post translational, subcellular localization, protein modification, post-translational modification, protein phosphorylation, protein structure, protein function, ubiquitinylation, acetylation, cellular component, cell type, visualization, data repository, bio.tools, FASEB list

Funding: NCI;

NIAAA R44 AA014848;

NIGMS R43 GM65768

Availability: Public, Free, The community can contribute to this resource

Resource Name: PhosphoSitePlus: Protein Modification Site

Resource ID: SCR_001837

Alternate IDs: biotools:phosphositeplus, nif-0000-10399

Alternate URLs: https://bio.tools/phosphositeplus

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Record Creation Time: 20220129T080209+0000

Record Last Update: 20250424T064511+0000

Ratings and Alerts

No rating or validation information has been found for PhosphoSitePlus: Protein Modification Site.

No alerts have been found for PhosphoSitePlus: Protein Modification Site.

Data and Source Information

Source: SciCrunch Registry

Usage and Citation Metrics

We found 825 mentions in open access literature.

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

Stastna M, et al. (2025) Post-translational modifications of proteins in cardiovascular diseases examined by proteomic approaches. The FEBS journal, 292(1), 28.

Shen Y, et al. (2025) GC-derived exosomal circMAN1A2 promotes cancer progression and suppresses T-cell antitumour immunity by inhibiting FBXW11-mediated SFPQ degradation. Journal of experimental & clinical cancer research: CR, 44(1), 24.

Hu Y, et al. (2025) Cross-Species Epitope Sequence Analysis for Discovery of Existing Antibodies Useful for Phospho-Specific Protein Detection in Model Species. International journal of molecular sciences, 26(2).

Souza-Silva IM, et al. (2025) Phosphoproteomics for studying signaling pathways evoked by

hormones of the renin-angiotensin system: A source of untapped potential. Acta physiologica (Oxford, England), 241(2), e14280.

Ngoi P, et al. (2025) Structural mechanism for recognition of E2F1 by the ubiquitin ligase adaptor Cyclin F. bioRxiv: the preprint server for biology.

Zou Y, et al. (2025) Sonic hedgehog restrains the ubiquitin-dependent degradation of SP1 to inhibit neuronal/glial senescence associated phenotypes in chemotherapy-induced peripheral neuropathy via the TRIM25-CXCL13 axis. Journal of advanced research, 68, 387.

Pollin G, et al. (2025) Emergent properties of the lysine methylome reveal regulatory roles via protein interactions and histone mimicry. Epigenomics, 17(1), 5.

Sundararajan R, et al. (2025) Loss of correlated proteasomal subunit expression selectively promotes the 20SHigh state which underlies luminal breast tumorigenicity. Communications biology, 8(1), 55.

Guo C, et al. (2025) LEDGF/p75 promotes transcriptional pausing through preventing SPT5 phosphorylation. Science advances, 11(3), eadr2131.

Matsumoto M, et al. (2025) Missense mutations of the ephrin receptor EPHA1 associated with Alzheimer's disease disrupt receptor signaling functions. The Journal of biological chemistry, 301(2), 108099.

Koo H, et al. (2025) Anti-proteolytic regulation of KRAS by USP9X/NDRG3 in KRAS-driven cancer development. Nature communications, 16(1), 628.

Li L, et al. (2024) Comprehensive Proteogenomic Profiling Reveals the Molecular Characteristics of Colorectal Cancer at Distinct Stages of Progression. Cancer research, 84(17), 2888.

Darling S, et al. (2024) The C-terminal disordered loop domain of Apc8 unlocks APC/C mitotic activation. Cell reports, 43(6), 114262.

Phung TK, et al. (2024) CURTAIN-A unique web-based tool for exploration and sharing of MS-based proteomics data. Proceedings of the National Academy of Sciences of the United States of America, 121(7), e2312676121.

Liu MY, et al. (2024) ATR phosphorylates DHX9 at serine 321 to suppress R-loop accumulation upon genotoxic stress. Nucleic acids research, 52(1), 204.

Zhang Y, et al. (2024) O-GlcNAcylation promotes malignancy and cisplatin resistance of lung cancer by stabilising NRF2. Clinical and translational medicine, 14(10), e70037.

Zuo S, et al. (2024) Mitochondria-Associated Gene SLC25A32 as a Novel Prognostic and Immunotherapy Biomarker: From Pan-Cancer Multiomics Analysis to Breast Cancer Validation. Analytical cellular pathology (Amsterdam), 2024, 1373659.

Farrokhi Yekta R, et al. (2024) Deciphering the potential role of post-translational

modifications of histones in gastrointestinal cancers: a proteomics-based review with therapeutic challenges and opportunities. Frontiers in oncology, 14, 1481426.

Wei L, et al. (2024) Systems-level reconstruction of kinase phosphosignaling networks regulating endothelial barrier integrity using temporal data. NPJ systems biology and applications, 10(1), 134.

Liu J, et al. (2024) MDM4 inhibits ferroptosis in p53 mutant colon cancer via regulating TRIM21/GPX4 expression. Cell death & disease, 15(11), 825.