

Resource Summary Report

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PhosphoNET

RRID:SCR_013070

Type: Tool

Proper Citation

PhosphoNET (RRID:SCR_013070)

Resource Information

URL: <http://www.phosphonet.ca/>

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Description: PhosphoNET is an open-access, online knowledgebase developed by Kinexus Bioinformatics Corporation to foster the study of cell signaling systems to advance biomedical research in academia and industry. PhosphoNET is the world's largest repository of known and predicted information on human phosphorylation sites, their evolutionary conservation and the identities of protein kinases that may target these sites. Search by protein name, UniProt number, IPI number, or 15 AA P-site sequence. PhosphoNET presently holds data on over 650,000 known and putative phosphorylation sites (P-sites) in over 23,000 human proteins that have been collected from the scientific literature and other reputable websites. Over 14% of these phospho-sites have been experimentally validated. The rest have been predicted with a novel P-Site Predictor algorithm developed at Kinexus with academic partners at the University of British Columbia and Simon Fraser University. With the PhosphoNET Evolution module, this website also provides information about cognate proteins in over 20 other species that may share these human phospho-sites. This helps to define the most functionally important phospho-sites as these are expected to be highly conserved in nature. With the Kinase Predictor module, listings are provided for the top 50 human protein kinases that are likely to phosphorylate each of these phospho-sites using another proprietary kinase substrate prediction algorithm developed at Kinexus. Our kinase substrate predictions are based on deduced consensus phosphorylation site amino acid frequency scoring matrices that we have determined for each of ~500 different human protein kinases. The specificity matrices are generated directly from the primary amino acid sequences of the catalytic domains of these kinases, and when available, have proven to correlate strongly with substrate prediction matrices based on alignment of known substrates of these kinases. The higher the score, the better the prospect that a kinase will phosphorylate a given site. Over 30 million kinase-substrate phospho-site pairs are quantified in PhosphoNET. Kinexus Bioinformatics Corporation has the capability to test most of these putative interactions in vitro for our clients.

Synonyms: PhosphoNET - Human Phospho-Site KnowledgeBase

Resource Type: database, data or information resource, service resource

Keywords: FASEB list

Funding: National Research Council of Canada Industrial Assistance Program ;
Natural Sciences and Engineering Research Council of Canada

Resource Name: PhosphoNET

Resource ID: SCR_013070

Alternate IDs: nlx_98420

Record Creation Time: 20220129T080314+0000

Record Last Update: 20260805T044307+0000

Ratings and Alerts

No rating or validation information has been found for PhosphoNET.

No alerts have been found for PhosphoNET.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 103 mentions in open access literature.

Listed below are recent publications. The full list is available at [RRID](#) .

Oosterwijk-Wakka JC, et al. (2025) Kinomic profiling to predict sunitinib response of patients with metastasized clear cell Renal Cell Carcinoma. Neoplasia (New York, N.Y.), 60, 101108.

Nguyen JH, et al. (2025) Multi-Omic Analysis of Glutamate Excitotoxicity in Primary Neuronal Cultures. Journal of neurochemistry, 169(6), e70110.

Ren J, et al. (2025) C15ORF48 serves as a potential biomarker and therapeutic target in pan-cancer with implications for lung cancer. Scientific reports, 15(1), 32821.

Sahu U, et al. (2025) IDH status dictates oHSV mediated metabolic reprogramming affecting anti-tumor immunity. Nature communications, 16(1), 3874.

Zhang J, et al. (2025) Role of PHB2 as a potential biomarker in pan-cancer: a multi-database analysis. Scientific reports, 15(1), 23933.

Acharya D, et al. (2025) TRIM23 mediates cGAS-induced autophagy in anti-HSV defense. *Nature communications*, 16(1), 4418.

Raymond JH, et al. (2025) Targeting GRPR for sex hormone-dependent cancer after loss of E-cadherin. *Nature*, 643(8072), 801.

Qiu X, et al. (2025) Expression and immunological role of FUNDC2 in pan-cancer. *PloS one*, 20(4), e0319343.

Shi X, et al. (2025) Pan-cancer analysis of the oncogenic role of ZNF703 in regulating tumor immunity. *BMC cancer*, 25(1), 1437.

Liang L, et al. (2025) CD137L promotes immune surveillance in melanoma via HLTF regulation. *Nature communications*, 16(1), 8478.

Park IY, et al. (2025) A novel cardiomyopathy phenotype linked to a CHD7 missense variant. *Scientific reports*, 15(1), 19429.

Ma X, et al. (2025) ATR regulates OCT4 phosphorylation and safeguards human naïve pluripotency. *Scientific reports*, 15(1), 15274.

Zhang Y, et al. (2024) TrkA promotes MDM2-mediated AGPS ubiquitination and degradation to trigger prostate cancer progression. *Journal of experimental & clinical cancer research : CR*, 43(1), 16.

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Liu Y, et al. (2024) Phosphorylated FOXQ1, a novel substrate of JNK1, inhibits sorafenib-induced ferroptosis by activating ETHE1 in hepatocellular carcinoma. *Cell death & disease*, 15(6), 395.

Dollet L, et al. (2024) Exercise-induced crosstalk between immune cells and adipocytes in humans: Role of oncostatin-M. *Cell reports. Medicine*, 5(1), 101348.

Foster J, et al. (2024) Lipid- and phospho-regulation of CTP:Phosphocholine Cytidylyltransferase ? association with nuclear lipid droplets. *Molecular biology of the cell*, 35(3), ar33.

Lee CJ, et al. (2024) ELK3 destabilization by speckle-type POZ protein suppresses prostate cancer progression and docetaxel resistance. *Cell death & disease*, 15(4), 274.

Xiao J, et al. (2023) Control of human pancreatic beta cell kinome by glucagon-like peptide-1 receptor biased agonism. *Diabetes, obesity & metabolism*, 25(8), 2105.

Lopez-Mejia IC, et al. (2023) Oxidative stress-induced FAK activation contributes to uterine serous carcinoma aggressiveness. *Molecular oncology*, 17(1), 98.