

Resource Summary Report

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Kinase Enrichment Analysis 3

RRID:SCR_023623

Type: Tool

Proper Citation

Kinase Enrichment Analysis 3 (RRID:SCR_023623)

Resource Information

URL: <https://maayanlab.cloud/kea3>

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Description: Web server application that infers overrepresentation of upstream kinases whose putative substrates are in user inputted list of proteins. Used to analyze data from phosphoproteomics and proteomics studies to predict upstream kinases responsible for observed differential phosphorylations.

Abbreviations: KEA3

Resource Type: data access protocol, software resource, web service

Defining Citation: [PMID:34019655](#)

Keywords: overrepresentation of upstream kinases, upstream kinases, upstream kinases substrates, user inputted list of proteins,

Funding: NCI U24 CA224260;
NHLBI U54 HL127624;
NIGMS T32 GM062754;
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Availability: Free, Freely available

Resource Name: Kinase Enrichment Analysis 3

Resource ID: SCR_023623

Record Creation Time: 20230527T050216+0000

Record Last Update: 20260829T182821+0000

Ratings and Alerts

No rating or validation information has been found for Kinase Enrichment Analysis 3.

No alerts have been found for Kinase Enrichment Analysis 3.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 16 mentions in open access literature.

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

Vijayakumar P, et al. (2026) Systems-Level Analysis of HPAI H5N1 Infection in Ducks: Integrating Transcriptomic, Proteomic, and Phosphoproteomic Data. International journal of molecular sciences, 27(6).

Yang H, et al. (2026) Pervasive phenotypic effects of FBXO42 are promoted by regulation of PP4 phosphatase. The EMBO journal, 45(4), 1332.

Shibue K, et al. (2025) Genome-wide CRISPR Screen Identifies Sec31A as a Key Regulator of Alpha Cell Survival. Nature communications, 16(1), 9159.

Wang H, et al. (2025) Isolation of Proteins on Chromatin Reveals Signaling Pathway-Dependent Alterations in the DNA-Bound Proteome. Molecular & cellular proteomics : MCP, 24(3), 100908.

Bahramibanan F, et al. (2025) Prognostic markers and molecular pathways in primary colorectal cancer with a high potential of liver metastases: a systems biology approach. Research in pharmaceutical sciences, 20(1), 121.

Krzystek TJ, et al. (2025) Opposing roles for GSK3?? and ERK1-dependent phosphorylation of huntingtin during neuronal dysfunction and cell death in Huntington's disease. Cell death & disease, 16(1), 328.

Nguyen JH, et al. (2025) Developmental pyrethroid exposure disrupts molecular pathways for MAP kinase and circadian rhythms in mouse brain. Physiological genomics, 57(4), 240.

Deng EZ, et al. (2024) Multiomics2Targets identifies targets from cancer cohorts profiled with transcriptomics, proteomics, and phosphoproteomics. Cell reports methods, 4(8), 100839.

Pichol-Thievend C, et al. (2024) VC-resist glioblastoma cell state: vessel co-option as a key driver of chemoradiation resistance. Nature communications, 15(1), 3602.

Arifin MZ, et al. (2024) BTLA and PD-1 signals attenuate TCR-mediated transcriptomic changes. iScience, 27(7), 110253.

Marino GB, et al. (2024) Protocol for using Multiomics2Targets to identify targets and driver kinases for cancer cohorts profiled with multi-omics assays. STAR protocols, 5(4), 103457.

Demir S, et al. (2024) Drug prioritization identifies panobinostat as a tailored treatment element for patients with metastatic hepatoblastoma. Journal of experimental & clinical cancer research : CR, 43(1), 299.

Nguyen JH, et al. (2023) Developmental pyrethroid exposure disrupts molecular pathways for circadian rhythms and MAP kinase in mouse brain. bioRxiv : the preprint server for biology.

Limsakul P, et al. (2023) Transcriptomic Analysis of Subtype-Specific Tyrosine Kinases as Triple Negative Breast Cancer Biomarkers. Cancers, 15(2).

Thom CS, et al. (2023) Phosphoproteomics reveals content and signaling differences between neonatal and adult platelets. bioRxiv : the preprint server for biology.

Gamradt P, et al. (2023) Stiffness-induced cancer-associated fibroblasts are responsible for immunosuppression in a platelet-derived growth factor ligand-dependent manner. PNAS nexus, 2(12), pgad405.