

Resource Summary Report

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Computational Biology Center

RRID:SCR_002877

Type: Tool

Proper Citation

Computational Biology Center (RRID:SCR_002877)

Resource Information

URL: <http://cbio.mskcc.org/>

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Description: Computational biology research at Memorial Sloan-Kettering Cancer Center (MSKCC) pursues computational biology research projects and the development of bioinformatics resources in the areas of: sequence-structure analysis; gene regulation; molecular pathways and networks, and diagnostic and prognostic indicators. The mission of cBio is to move the theoretical methods and genome-scale data resources of computational biology into everyday laboratory practice and use, and is reflected in the organization of cBio into research and service components ~ the intention being that new computational methods created through the process of scientific inquiry should be generalized and supported as open-source and shared community resources. Faculty from cBio participate in graduate training provided through the following graduate programs: * Gerstner Sloan-Kettering Graduate School of Biomedical Sciences * Graduate Training Program in Computational Biology and Medicine Integral to much of the research and service work performed by cBio is the creation and use of software tools and data resources. The tools that we have created and utilize provide evidence of our involvement in the following areas: * Cancer Genomics * Data Repositories * iPhone & iPod Touch * microRNAs * Pathways * Protein Function * Text Analysis * Transcription Profiling

Synonyms: cBio

Resource Type: data or information resource, disease-related portal, portal, topical portal, training resource

Keywords: drug, evolution, experiment, gene, algorithm, bioinformatics, biology, cancer, clinical, computational, diagnostic, genome, human, initiation, kinetics, laboratory, leukemia, ligand, metastasis, microRNA, mirna, model, molecular, network, pathway, phenotype, prognostic, progression, protein, regulation, research, resistance, rna, sequence, stem cell, structure, t cell, therapy, treatment, tumor

Availability: Free, Freely available

Resource Name: Computational Biology Center

Resource ID: SCR_002877

Alternate IDs: nif-0000-25560

Record Creation Time: 20220129T080215+0000

Record Last Update: 20260829T182116+0000

Ratings and Alerts

No rating or validation information has been found for Computational Biology Center.

No alerts have been found for Computational Biology Center.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 71 mentions in open access literature.

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

Pozzobon D, et al. (2025) Pan-Cancer Upregulation of the FOXM1 Transcription Factor. Genes, 16(1).

Sad K, et al. (2025) Histone H3E50K remodels chromatin to confer oncogenic activity and support an EMT phenotype. NAR cancer, 7(1), zcaf002.

Zhang P, et al. (2025) Identification of CD66c as a potential target in gastroesophageal junction cancer for antibody-drug conjugate development. Gastric cancer : official journal of the International Gastric Cancer Association and the Japanese Gastric Cancer Association, 28(3), 422.

McGivney GR, et al. (2025) Somatic CRISPR tumorigenesis and multiomic analysis reveal a pentose phosphate pathway disruption vulnerability in MPNSTs. Science advances, 11(33), eadu2906.

Burnette H, et al. (2025) Chatbot assistance in precision oncology treatment decision-making. The oncologist, 30(10).

Alhamdan YR, et al. (2024) BRAF Expression and Copy Number Alterations Predict Unfavorable Tumor Features and Adverse Outcomes in Patients With Breast Cancer. International journal of breast cancer, 2024, 6373900.

Schulz-Mirbach H, et al. (2024) Engineering new-to-nature biochemical conversions by combining fermentative metabolism with respiratory modules. Nature communications,

15(1), 6725.

Bae SDW, et al. (2024) Constitutive androstane receptor (CAR) functions as a tumor suppressor via regulating stemness in liver cancer. *Scientific reports*, 14(1), 30926.

Sottnik JL, et al. (2024) Co-regulator activity of Mediator of DNA Damage Checkpoint 1 (MDC1) is associated with DNA repair dysfunction and PARP inhibitor sensitivity in lobular carcinoma of the breast. *bioRxiv : the preprint server for biology*.

Abecunas C, et al. (2023) Loss of NF1 in Melanoma Confers Sensitivity to SYK Kinase Inhibition. *Cancer research*, 83(2), 316.

Zhao M, et al. (2023) Epigenetically upregulating TROP2 and SLFN11 enhances therapeutic efficacy of TROP2 antibody drug conjugate sacituzumab govitecan. *NPJ breast cancer*, 9(1), 66.

Wang S, et al. (2023) Identifying prognostic subgroups of luminal-A breast cancer using deep autoencoders and gene expressions. *PLoS computational biology*, 19(5), e1011197.

Yin J, et al. (2023) The efficacy of immune checkpoint inhibitors is limited in elderly NSCLC: a retrospective efficacy study and meta-analysis. *Aging*, 15(24), 15025.

Cahuzac KM, et al. (2023) AKT activation because of PTEN loss upregulates xCT via GSK3 β /NRF2, leading to inhibition of ferroptosis in PTEN-mutant tumor cells. *Cell reports*, 42(5), 112536.

Auf der Maur P, et al. (2023) N-acetylcysteine overcomes NF1 loss-driven resistance to PI3K β inhibition in breast cancer. *Cell reports. Medicine*, 4(4), 101002.

da Costa BHB, et al. (2022) EGFL7 expression profile in IDH-wildtype glioblastomas is associated with poor patient outcome. *Journal of pathology and translational medicine*, 56(4), 205.

Li B, et al. (2021) Leptin Receptor Overlapping Transcript (LEPROT) Is Associated with the Tumor Microenvironment and a Prognostic Predictor in Pan-Cancer. *Frontiers in genetics*, 12, 749435.

Woolston A, et al. (2021) Mutational signatures impact the evolution of anti-EGFR antibody resistance in colorectal cancer. *Nature ecology & evolution*, 5(7), 1024.

Arakelyan A, et al. (2021) Transcriptome Patterns of BRCA1- and BRCA2- Mutated Breast and Ovarian Cancers. *International journal of molecular sciences*, 22(3).

James CD, et al. (2021) Restoring the DREAM Complex Inhibits the Proliferation of High-Risk HPV Positive Human Cells. *Cancers*, 13(3).