

Resource Summary Report

Generated by [kravitz2](#) on Jul 31, 2026

canSAR

RRID:SCR_006794

Type: Tool

Proper Citation

canSAR (RRID:SCR_006794)

Resource Information

URL: <https://cansar.icr.ac.uk/>

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Description: canSAR is an integrated database that brings together biological, chemical, pharmacological (and eventually clinical) data. Its goal is to integrate this data and make it accessible to cancer research scientists from multiple disciplines, in order to help with hypothesis generation in cancer research and support translational research. This cancer research and drug discovery resource was developed to utilize the growing publicly available biological annotation, chemical screening, RNA interference screening, expression, amplification and 3D structural data. Scientists can, in a single place, rapidly identify biological annotation of a target, its structural characterization, expression levels and protein interaction data, as well as suitable cell lines for experiments, potential tool compounds and similarity to known drug targets. canSAR has, from the outset, been completely use-case driven which has dramatically influenced the design of the back-end and the functionality provided through the interfaces. The Web interface provides flexible, multipoint entry into canSAR. This allows easy access to the multidisciplinary data within, including target and compound synopses, bioactivity views and expert tools for chemogenomic, expression and protein interaction network data.

Abbreviations: canSAR

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

Defining Citation: [PMID:22013161](#)

Keywords: molecular target, expression, cell line, compound, molecule, protein, structure, ligand, drug, 3d, genomics, 3d complex, bioactivity, protein affinity, cell line sensitivity, pathway, annotation, bio.tools, FASEB list

Related Condition: Cancer

Funding: Cancer Research UK C309/A8274

Availability: CanSAR is freely available to all cancer researchers. By using canSAR you are agreeing to the Terms of Use, <https://cansar.icr.ac.uk/cansar/terms-of-use/>

Resource Name: canSAR

Resource ID: SCR_006794

Alternate IDs: biotools:cansar, nlx_149410

Alternate URLs: <https://bio.tools/cansar>

Record Creation Time: 20220129T080238+0000

Record Last Update: 20260731T042730+0000

Ratings and Alerts

No rating or validation information has been found for canSAR.

No alerts have been found for canSAR.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 54 mentions in open access literature.

Listed below are recent publications. The full list is available at [kravitz2](#) .

Ma A, et al. (2025) ENTR1 affects the progression of colon cancer by regulating energy metabolism under the influence of glycolysis. BMC cancer, 25(1), 992.

Zhang Y, et al. (2025) LEDGF Binds H3R17me2a Promoting De Novo Nucleotide Biosynthesis in SETD2 Mutant Clear Cell Renal Cell Carcinoma. Advanced science (Weinheim, Baden-Wurtemberg, Germany), 12(35), e16809.

Zhang S, et al. (2025) The role and application of bioinformatics techniques and tools in drug discovery. Frontiers in pharmacology, 16, 1547131.

Espinoza-Ferrao S, et al. (2025) Global analysis of actionable genomic alterations in thyroid cancer and precision-based pharmacogenomic strategies. Frontiers in pharmacology, 16, 1524623.

Silva L, et al. (2025) Efficient identification of new small molecules targeting succinate dehydrogenase in non-small cell lung cancer. Cancer cell international, 25(1), 362.

Kinnersley B, et al. (2025) Genomic landscape of diffuse glioma revealed by whole genome sequencing. *Nature communications*, 16(1), 4233.

Sánchez-Ortega M, et al. (2025) Double vulnerability of active-NRF2 lung squamous cell carcinoma to NRF2 and TRIM24. *Molecular cancer*, 24(1), 197.

Bapat J, et al. (2024) CASC4/GOLM2 drives high grade serous carcinoma anoikis resistance through the recycling of EGFR. *Cancer gene therapy*, 31(2), 300.

Wang B, et al. (2024) A bird's eye view of the potential role of NFKBIA in pan-cancer. *Heliyon*, 10(10), e31204.

López-Cortés A, et al. (2024) Unraveling druggable cancer-driving proteins and targeted drugs using artificial intelligence and multi-omics analyses. *Scientific reports*, 14(1), 19359.

Zhou Q, et al. (2024) Vemurafenib induces senescence in acute myeloid leukemia and myelodysplastic syndrome by activating the HIPPO signaling pathway: implications for potential targeted therapy. *Biology direct*, 19(1), 6.

Kinnersley B, et al. (2024) Analysis of 10,478 cancer genomes identifies candidate driver genes and opportunities for precision oncology. *Nature genetics*, 56(9), 1868.

Neeb A, et al. (2024) Thio-2 Inhibits Key Signaling Pathways Required for the Development and Progression of Castration-resistant Prostate Cancer. *Molecular cancer therapeutics*, 23(6), 791.

Zhu WZ, et al. (2023) An autophagy-related gene prognostic index predicting biochemical recurrence, metastasis, and drug resistance for prostate cancer. *Asian journal of andrology*, 25(2), 208.

Zhang Y, et al. (2023) Identification of the H3K36me3 reader LEDGF/p75 in the pancancer landscape and functional exploration in clear cell renal cell carcinoma. *Computational and structural biotechnology journal*, 21, 4134.

Acar A, et al. (2023) Pan-Cancer Analysis of the COVID-19 Causal Gene SLC6A20. *ACS omega*, 8(14), 13153.

Beach C, et al. (2023) Improving radiotherapy in immunosuppressive microenvironments by targeting complement receptor C5aR1. *The Journal of clinical investigation*, 133(23).

Candido MF, et al. (2023) Drugging Hijacked Kinase Pathways in Pediatric Oncology: Opportunities and Current Scenario. *Pharmaceutics*, 15(2).

Aftab F, et al. (2023) An intrinsic purine metabolite AICAR blocks lung tumour growth by targeting oncoprotein mucin 1. *British journal of cancer*, 128(9), 1647.

Sun W, et al. (2023) Catalytic domain-dependent and -independent transcriptional activities of the tumour suppressor histone H3K27 demethylase UTX/KDM6A in specific cancer types. *Epigenetics*, 18(1), 2222245.