

Resource Summary Report

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Cell Model Passports

RRID:SCR_027682

Type: Tool

Proper Citation

Cell Model Passports (RRID:SCR_027682)

Resource Information

URL: <https://cellmodelpassports.sanger.ac.uk/>

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Description: Hub for clinical, genetic and functional datasets of preclinical cancer models. Provides details of cell model relationships, patient and clinical information, as well as access to associated genetic and functional datasets. Passports database contains curated details and standardized annotation for cell models, including cancer organoid cultures. Users can navigate database via tissue, cancer-type, genetic feature and data availability to select model. REST-API provides programmatic data access and exploration.

Resource Type: catalog, data or information resource, database

Defining Citation: [PMID:30260411](#)

Keywords: clinical, genetic, functional, datasets, preclinical cancer models,

Funding: Wellcome Sanger Institute ;
Wellcome Trust

Availability: Free, Freely available

Resource Name: Cell Model Passports

Resource ID: SCR_027682

Record Creation Time: 20251120T080608+0000

Record Last Update: 20260821T194356+0000

Ratings and Alerts

No rating or validation information has been found for Cell Model Passports.

No alerts have been found for Cell Model Passports.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 15 mentions in open access literature.

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

Costa TGF, et al. (2026) Targeting cancer expressed EGFR with a humanized monoclonal antibody. Scientific reports, 16(1).

Laurenziello P, et al. (2026) Salinomycin as a death switch: how gastric cancer cells choose their demise. Cell death discovery, 12(1).

Popovi?? J, et al. (2026) Changes in EGFR activity following CRISPR/Cas9-editing of the EGF binding domain. Scientific reports, 16(1), 6797.

Rashad S, et al. (2026) G/C-ending and synonymous codon bias define functional translational programs that shape human tissue and cancer proteomes. NAR genomics and bioinformatics, 8(2), lqag033.

Gupta S, et al. (2025) Deciphering context-specific Axitinib escape pathways via multi-omics and explainable machine learning. Journal of translational medicine, 23(1), 1268.

Chauhan R, et al. (2025) Targeting ubiquitin-specific protease 14 reduces metastatic potential and metabolic activity in cervical cancer via direct modulation of monocarboxylate transporter-4. Journal of translational medicine, 24(1), 33.

Zhang P, et al. (2025) Integrated pathway analysis identifies prognostically relevant subtypes of glioblastoma characterized by abnormalities in multi-omics. Clinical and translational medicine, 15(12), e70517.

Scuderi C, et al. (2024) Gain-Type Aneuploidies Influence the Burden of Selective Long Non-Coding Transcripts in Colorectal Cancer. International journal of molecular sciences, 25(10).

Barresi V, et al. (2023) Temporary serine protease inhibition and the role of SPINK2 in human bone marrow. iScience, 26(6), 106949.

Liberal FDCG, et al. (2023) Characterization of Intrinsic Radiation Sensitivity in a Diverse Panel of Normal, Cancerous and CRISPR-Modified Cell Lines. International journal of molecular sciences, 24(9).

Croft D, et al. (2023) Effectively utilizing publicly available databases for cancer target evaluation. *NAR cancer*, 5(3), zcad035.

Wodi C, et al. (2023) SPHINX-Based Combination Therapy as a Potential Novel Treatment Strategy for Acute Myeloid Leukaemia. *British journal of biomedical science*, 80, 11041.

Zheng X, et al. (2022) A Novel Tongue Squamous Cell Carcinoma Cell Line Escapes from Immune Recognition due to Genetic Alterations in HLA Class I Complex. *Cells*, 12(1).

Ciucci A, et al. (2022) Preclinical models of epithelial ovarian cancer: practical considerations and challenges for a meaningful application. *Cellular and molecular life sciences* : CMLS, 79(7), 364.

Park BJ, et al. (2022) Utilization of Cancer Cell Line Screening to Elucidate the Anticancer Activity and Biological Pathways Related to the Ruthenium-Based Therapeutic BOLD-100. *Cancers*, 15(1).