

Resource Summary Report

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DCTD

RRID:SCR_004196

Type: Tool

Proper Citation

DCTD (RRID:SCR_004196)

Resource Information

URL: <http://dctd.cancer.gov/>

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Description: Division of NCI that takes prospective cancer detection and treatment leads, facilitates their paths to clinical application, and expedites the initial and subsequent large-scale testing of new agents, biomarkers, imaging tests, and other therapeutic interventions (radiation, surgery, immunotherapy) in patients. DCTD, like all of NCI, supports many programs that could not be done without government funding - investigators supported by the division engage in scientifically sound, high-risk research that may yield great benefits for patients with cancer, but are too difficult or risky for industry or academia to pursue. This includes a particular emphasis on the development of distinct molecular signatures for cancer, refined molecular assays, and state-of-the-art imaging techniques that will guide oncologic therapy in the future. The division has eight major programs that work together to bring unique molecules, diagnostic tests, and therapeutic interventions from the laboratory bench to the patient bedside: * Cancer Diagnosis Program * Cancer Imaging Program * Cancer Therapy Evaluation Program * Developmental Therapeutics Program * Radiation Research Program * Translational Research Program * Biometrics Research Branch * Office of Cancer Complementary and Alternative Medicine

Abbreviations: DCTD

Synonyms: Division of Cancer Treatment and Diagnosis

Resource Type: data or information resource, funding resource, portal

Keywords: treatment, diagnosis, molecule, diagnostic test, therapeutic intervention

Related Condition: Cancer

Funding: NCI

Resource Name: DCTD

Resource ID: SCR_004196

Alternate IDs: OMICS_01537

Record Creation Time: 20220129T080223+0000

Record Last Update: 20260912T200054+0000

Ratings and Alerts

No rating or validation information has been found for DCTD.

No alerts have been found for DCTD.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 20 mentions in open access literature.

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

Cahn D, et al. (2026) PEGylation strategies for enhanced nanoparticle delivery to tumor-associated immune cells. *Bioengineering & translational medicine*, 11(3), e70098.

Mestareehi A, et al. (2026) Integrated Global Phosphoproteomic, Bioinformatic, and Machine Learning Analysis Reveals Regulatory Networks of TOP1, TOP2A, TOP2B, and C1orf35 in Hepatocellular Carcinoma (HCC). *Oncology research*, 34(5), 18.

Moreno CAM, et al. (2025) Clinical and molecular spectrum of TK2-deficiency: a large Brazilian cohort. *Scientific reports*, 15(1), 9013.

Wang Y, et al. (2025) Induced clustering of SHP2-depleted tumor cells in vascular islands restores sensitivity to MEK/ERK inhibition. *The Journal of clinical investigation*, 135(10).

Cahn D, et al. (2025) PEGylation strategies for enhanced nanoparticle delivery to tumor associated immune cells. *bioRxiv : the preprint server for biology*.

Ahluwalia MS, et al. (2024) Evaluating the Base Excision Repair Inhibitor TRC102 and Temozolomide for Patients with Recurrent Glioblastoma in the Phase 2 Adult Brain Tumor Consortium Trial BERT. *Clinical cancer research : an official journal of the American Association for Cancer Research*, 30(15), 3167.

Nduom EK, et al. (2023) Clinical protocol: Feasibility of evaluating abemaciclib neuropharmacokinetics of diffuse midline glioma using intratumoral microdialysis. *PLoS one*, 18(9), e0291068.

- Kang S, et al. (2022) Transition of Dephospho-DctD to the Transcriptionally Active State via Interaction with Dephospho-IIAGlc. *mBio*, 13(2), e0383921.
- Lee JW, et al. (2021) A comparative study on the unified model based multifactor dimensionality reduction methods for identifying gene-gene interactions associated with the survival phenotype. *BioData mining*, 14(1), 17.
- Gilmore AC, et al. (2021) An in vitro tumorigenesis model based on live-cell-generated oxygen and nutrient gradients. *Communications biology*, 4(1), 477.
- Dominguez-Gonzalez C, et al. (2020) Growth Differentiation Factor 15 is a potential biomarker of therapeutic response for TK2 deficient myopathy. *Scientific reports*, 10(1), 10111.
- Blázquez-Bermejo C, et al. (2019) Age-related metabolic changes limit efficacy of deoxynucleoside-based therapy in thymidine kinase 2-deficient mice. *EBioMedicine*, 46, 342.
- Lopez-Gomez C, et al. (2019) Bioavailability and cytosolic kinases modulate response to deoxynucleoside therapy in TK2 deficiency. *EBioMedicine*, 46, 356.
- Yadak R, et al. (2017) Mitochondrial Neurogastrointestinal Encephalomyopathy Caused by Thymidine Phosphorylase Enzyme Deficiency: From Pathogenesis to Emerging Therapeutic Options. *Frontiers in cellular neuroscience*, 11, 31.
- Murai J, et al. (2016) Resistance to PARP inhibitors by SLFN11 inactivation can be overcome by ATR inhibition. *Oncotarget*, 7(47), 76534.
- Ligasová A, et al. (2016) Dr Jekyll and Mr Hyde: a strange case of 5-ethynyl-2'-deoxyuridine and 5-ethynyl-2'-deoxycytidine. *Open biology*, 6(1), 150172.
- Almqvist H, et al. (2016) CETSA screening identifies known and novel thymidylate synthase inhibitors and slow intracellular activation of 5-fluorouracil. *Nature communications*, 7, 11040.
- Fernández I, et al. (2015) Snapshots of Conformational Changes Shed Light into the NtrX Receiver Domain Signal Transduction Mechanism. *Journal of molecular biology*, 427(20), 3258.
- Martín Sánchez C, et al. (2015) Disruption of the mevalonate pathway induces dNTP depletion and DNA damage. *Biochimica et biophysica acta*, 1851(9), 1240.
- Davies KM, et al. (2009) The HupR receiver domain crystal structure in its nonphospho and inhibitory phospho states. *Journal of molecular biology*, 385(1), 51.