

Resource Summary Report

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BRASS

RRID:SCR_017091

Type: Tool

Proper Citation

BRASS (RRID:SCR_017091)

Resource Information

URL: <https://github.com/cancerit/BRASS>

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Description: Software tool for analysis of one or more related BAM files of paired end sequencing to determine potential rearrangement breakpoints. Identifies breaks and attempts to assemble rearrangements.

Resource Type: data analysis software, software resource, data processing software, software application

Keywords: analysis, BAM, file, paired, end, sequencing, determine, rearrangement, breakpoint, assemble

Availability: Free, Available for download, Freely available

Resource Name: BRASS

Resource ID: SCR_017091

License: GNU AGPL v3

Record Creation Time: 20220129T080333+0000

Record Last Update: 20260803T040740+0000

Ratings and Alerts

No rating or validation information has been found for BRASS.

No alerts have been found for BRASS.

Data and Source Information

Usage and Citation Metrics

We found 42 mentions in open access literature.

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

Rekhtman N, et al. (2025) Chromothripsis-Mediated Small Cell Lung Carcinoma. *Cancer discovery*, 15(1), 83.

Yost KE, et al. (2025) Three-dimensional genome landscape of primary human cancers. *Nature genetics*, 57(5), 1189.

Díaz-Gay M, et al. (2025) Geographic and age variations in mutational processes in colorectal cancer. *Nature*, 643(8070), 230.

Minello A, et al. (2025) A recurrent pathogenic BRCA2 truncating variant reveals a role for BRCA2-PCAF complex in modulating NF- κ B-driven transcription. *Nature communications*, 16(1), 10953.

Domenico D, et al. (2025) Enabling whole genome sequencing analysis from FFPE specimens in clinical oncology. *Nature communications*, 16(1), 10649.

Li YR, et al. (2025) Long-Term Latency of Highly Mutated Cells in Normal Mouse Skin Is Reversed by Exposure to Tumor Promoters and Chronic Tissue Damage. *Cancer discovery*, 15(6), 1115.

Lee-Six H, et al. (2025) High resolution clonal architecture of hypomutated Wilms tumours. *Nature communications*, 16(1), 4647.

Treger TD, et al. (2025) Predisposition Footprints in the Somatic Genome of Wilms Tumors. *Cancer discovery*, 15(2), 286.

Persaud N, et al. (2025) Clinical Significance of TERT Promoter Mutations in Neuroblastoma. *JCO precision oncology*, 9, e2500074.

Anselmino N, et al. (2024) Integrative Molecular Analyses of the MD Anderson Prostate Cancer Patient-derived Xenograft (MDA PCa PDX) Series. *Clinical cancer research : an official journal of the American Association for Cancer Research*, 30(10), 2272.

Vergara X, et al. (2024) Widespread chromatin context-dependencies of DNA double-strand break repair proteins. *Nature communications*, 15(1), 5334.

Ijaz J, et al. (2024) Haplotype-specific assembly of shattered chromosomes in esophageal adenocarcinomas. *Cell genomics*, 4(2), 100484.

Wu CC, et al. (2024) Whole genome and reverse protein phase array landscapes of patient derived osteosarcoma xenograft models. *Scientific reports*, 14(1), 19891.

Hu X, et al. (2024) The evolution of lung adenocarcinoma precursors is associated with chromosomal instability and transition from innate to adaptive immune response/evasion. Research square.

Keahi DL, et al. (2024) G-quadruplexes are a source of vulnerability in BRCA2 deficient granule cell progenitors and medulloblastoma. bioRxiv : the preprint server for biology.

Koh GCC, et al. (2024) The chemotherapeutic drug CX-5461 is a potent mutagen in cultured human cells. Nature genetics, 56(1), 23.

Nunes L, et al. (2024) Prognostic genome and transcriptome signatures in colorectal cancers. Nature, 633(8028), 137.

Andersen LVB, et al. (2023) Non-BRCA1/BRCA2 high-risk familial breast cancers are not associated with a high prevalence of BRCAness. Breast cancer research : BCR, 25(1), 69.

Gadd S, et al. (2022) Genetic changes associated with relapse in favorable histology Wilms tumor: A Children's Oncology Group AREN03B2 study. Cell reports. Medicine, 3(6), 100644.

Buhigas C, et al. (2022) The architecture of clonal expansions in morphologically normal tissue from cancerous and non-cancerous prostates. Molecular cancer, 21(1), 183.