

Resource Summary Report

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Allen Human Brain Atlas: BrainSpan (Atlas of the Developing Brain)

RRID:SCR_008083

Type: Tool

Proper Citation

Allen Human Brain Atlas: BrainSpan (Atlas of the Developing Brain) (RRID:SCR_008083)

Resource Information

URL: <http://brainspan.org/>

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Description: Atlas of developing human brain for studying transcriptional mechanisms involved in human brain development. Consists of RNA sequencing and exon microarray data profiling up to sixteen cortical and subcortical structures across full course of human brain development, high resolution neuroanatomical transcriptional profiles of about 300 distinct structures spanning entire brain for four midgestational prenatal specimens, in situ hybridization image data covering selected genes and brain regions in developing and adult human brain, reference atlas in full color with high resolution anatomic reference atlases of prenatal (two stages) and adult human brain along with supporting histology, magnetic resonance imaging (MRI) and diffusion weighted imaging (DWI) data.

Abbreviations: BrainSpan

Synonyms: BrainSpan - Atlas of the Developing Human Brain, BrainSpan: Atlas of the Developing Human Brain, NIMH Transcriptional Atlas of Human Brain Development

Resource Type: atlas, data or information resource, expression atlas, reference atlas

Keywords: anatomic, gene expression, molecular neuroanatomy, in situ hybridization, human, medial prefrontal cortex, primary visual cortex, hippocampus, amygdala, ventral striatum, postnatal, development, brain development, transcription, brain, rna sequencing, exon microarray, developmental stage, male, female, mrna transcript, developing human, adult human, fetal brain, fetus, histology, transcriptome, magnetic resonance imaging, diffusion tensor imaging, annotation, neuroanatomy, prenatal, development, fiber tract, microarray, mri, dti, methylation, microrna, mrf

Related Condition: Neurodevelopmental disorder, Neuropsychiatric disease, Schizophrenia, Epilepsy, Parkinson's disease, Alzheimer's disease, Neurological disease, Autism

Funding: NIMH RC2 MH089921;
NIMH RC2 MH089929;
NIMH RC2 MH090047

Availability: Free, Freely available

Resource Name: Allen Human Brain Atlas: BrainSpan (Atlas of the Developing Brain)

Resource ID: SCR_008083

Alternate IDs: nif-0000-10626

Alternate URLs: <http://www.developinghumanbrain.org/>

License URLs: http://www.brainspan.org/policies.html#terms_of_use

Record Creation Time: 20220129T080245+0000

Record Last Update: 20260903T234935+0000

Ratings and Alerts

No rating or validation information has been found for Allen Human Brain Atlas: BrainSpan (Atlas of the Developing Brain).

No alerts have been found for Allen Human Brain Atlas: BrainSpan (Atlas of the Developing Brain).

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 465 mentions in open access literature.

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

Liaci C, et al. (2026) ARHGEF6-dependent cytoskeletal regulation underlies a conserved program of forebrain interneuron development. bioRxiv : the preprint server for biology.

Liu Y, et al. (2026) Functional variants at 1p36.23 confer risk of schizophrenia through modulating RERE. Nature communications, 17(1), 1742.

Spirito G, et al. (2026) New insights into neurodevelopmental disorders by whole genome sequencing of 100 families from Italy. NPJ genomic medicine, 11(1), 11.

Stella C, et al. (2026) Biological underpinnings and genetic predisposition to schizophrenia within microrna-137 regulatory pathways across brain development. *Translational psychiatry*, 16(1).

He X, et al. (2026) Implication of genetic-dependent stage in the development of SCN8A-associated epilepsy: a case report. *Frontiers in human neuroscience*, 20, 1775909.

Sun L, et al. (2026) Transcriptomic signatures of synaptic loss in Alzheimer's disease. *iScience*, 29(7), 116336.

Eising E, et al. (2026) De novo protein-coding gene variants in developmental stuttering. *Molecular psychiatry*, 31(1), 104.

Doijad S, et al. (2026) Integrated genetic analysis reveals synaptic m6A enrichment in high-risk autism genes. *Frontiers in molecular neuroscience*, 19, 1767983.

Yang K, et al. (2026) In vivo base editing of Chd3 rescues behavioural abnormalities in mice. *Nature*, 651(8106), 785.

Musante L, et al. (2026) A novel spliceosomopathy caused by de novo SF3B3 variants. *Genome medicine*, 18(1).

Louis P, et al. (2026) Protracted Functional and Structural Reorganization of Human Prefrontal Cortex Supports Lateralized Category Geometries. *bioRxiv : the preprint server for biology*.

Demontis D, et al. (2026) Rare genetic variants confer a high risk of ADHD and implicate neuronal biology. *Nature*, 649(8098), 909.

Arcego DM, et al. (2026) Sex-specific interaction effects of Syntaxin 1A coexpression network and childhood trauma on adult depressive symptoms. *EBioMedicine*, 123, 106062.

Gonzalez-Peñas J, et al. (2026) Sulcal Widening in Schizophrenia Maps onto Sulcal Hubs and Energy-Synaptic Genes. *bioRxiv : the preprint server for biology*.

Varella-Branco E, et al. (2026) "SHANK3 deficiency alters early progenitor dynamics and reveals shared pathways with neurodegeneration". *Molecular psychiatry*, 31(6), 3033.

Baris S, et al. (2026) Biallelic Truncating DNAH14 Variant in Siblings with Neurodevelopmental Disorder and Predominant Ataxia: Clinical Report and Literature Review. *International journal of molecular sciences*, 27(2).

Liu J, et al. (2026) Recent developments in imaging transcriptomics for psychiatric disorders. *Psychoradiology*, 6, kkg010.

Dumanis G, et al. (2026) Medulloblastoma immune microenvironment resembles early brain development and reveals novel basophil- B cell- associated signatures. *Neuro-oncology advances*, 8(1), vdag134.

Epihova G, et al. (2026) The Fronto-Temporal Cortex Has Increased Subcortical Connectivity In Utero and Plasticity in Adulthood. *The Journal of neuroscience : the official journal of the Society for Neuroscience*, 46(7).

Pang T, et al. (2026) Transcriptomic analysis in autism spectrum disorder suggests three molecular subtypes with distinct phenotypic profiles and functional pathways. *Communications biology*, 9(1).