

Resource Summary Report

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Autism Tissue Program

RRID:SCR_000651

Type: Tool

Proper Citation

Autism Tissue Program (RRID:SCR_000651)

Resource Information

URL: <http://www.nitrc.org/projects/atp>

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Description: Autism research program that makes available post-mortem brain tissue to qualified scientists all over the world. Working directly with tissue banks, organ procurement agencies, medical examiners and the general public, this is the largest program dedicated to increasing and enhancing the availability of post-mortem brain tissue for basic research in autism. To date, the ATP has collected and stored more than 170 brains in their repositories at Harvard (US) and Oxford (UK). These brains are processed by formalin fixation and/or snap frozen to properly provide high quality tissue of all brain regions, in support of biological research in autism. The ATP is unique in that they diligently pursue all available clinical data (pre and post mortem) on tissue donors in order to create the most biologically relevant brain repository for autism research. These data, together with tissue resources from both banks and associated repositories, are presented to all interested researchers through their extensive web-based data portal (login required). The ATP is not a brain bank, but works directly with the Harvard Brain Tissue Resource Center in Boston (HBTRC), Massachusetts to serve as its tissue repository. This program augments brain bank functions by: * Creating the most biologically relevant brain tissue repository possible * Fully covering all costs associated with brain extraction and transfer to the repositories at Harvard (US and Canada) and Oxford (UK). * Providing scientific oversight of tissue distributions * Overseeing and managing all tissue grants * Clinically phenotyping and acquiring extensive medical data on all of their donors * Providing continuing family support and communication to all of their donors * Directly supporting researchers to facilitate autism research * Maintaining a robust web based data management and secure on-line global interface system * Developing and supporting ATP established scientific initiatives * Actively providing public outreach and education The ATP is not a clinical organ procurement agency, but rather they facilitate the wishes of donors and families to donate their tissue to autism research. Through the ATP's established international infrastructure, they work with any accredited tissue bank, organ procurement agency, or medical examiner that receives a family's request to donate their loved one's tissue to the program. Once contacted, the ATP will insure that the family's

request to donate their loved one's tissue is faithfully met, covering all costs to the family and partnering agency as well as ensuring the tissues' proper and rapid transport to the ATP's repository at the Harvard Brain Tissue Resource Center (HBTRC) in Boston, Massachusetts.

Abbreviations: ATP

Resource Type: data or information resource, database, funding resource, disease-related portal, portal, topical portal

Defining Citation: [PMID:16933088](#)

Keywords: autism, brain, tissue, clinical data, post-mortem, brain tissue, donate, brain donation, autism spectrum disorder, pervasive development disorder, formalin fixation, snap frozen, tissue section, stained slide, dna, skin fibroblast culture, control, clinical, clinical neuroinformatics, imaging genomics, magnetic resonance, optical imaging, FASEB list

Related Condition: Autism, Autism spectrum disorder, Pervasive Development Disorder, Control

Funding: Autism Speaks

Availability: Free, Freely available

Resource Name: Autism Tissue Program

Resource ID: SCR_000651

Alternate IDs: nif-0000-10160

Alternate URLs: <http://www.brainbank.org/>,
<http://www.autismtissueprogram.org/site/c.nIKUL7MQIsG/b.5183271/k.BD86/Home.htm>

Record Creation Time: 20220129T080202+0000

Record Last Update: 20260820T054312+0000

Ratings and Alerts

No rating or validation information has been found for Autism Tissue Program.

No alerts have been found for Autism Tissue Program.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 28 mentions in open access literature.

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

Zikopoulos B, et al. (2018) Parallel trends in cortical gray and white matter architecture and connections in primates allow fine study of pathways in humans and reveal network disruptions in autism. PLoS biology, 16(2), e2004559.

Zhubi A, et al. (2017) Epigenetic regulation of RELN and GAD1 in the frontal cortex (FC) of autism spectrum disorder (ASD) subjects. International journal of developmental neuroscience : the official journal of the International Society for Developmental Neuroscience, 62, 63.

Pagan C, et al. (2017) Disruption of melatonin synthesis is associated with impaired 14-3-3 and miR-451 levels in patients with autism spectrum disorders. Scientific reports, 7(1), 2096.

Reilly J, et al. (2017) Bio-collections in autism research. Molecular autism, 8, 34.

Zhang Y, et al. (2016) Decreased Brain Levels of Vitamin B12 in Aging, Autism and Schizophrenia. PloS one, 11(1), e0146797.

Sun W, et al. (2016) Histone Acetylome-wide Association Study of Autism Spectrum Disorder. Cell, 167(5), 1385.

Ray B, et al. (2016) Finding novel distinctions between the sAPP β -mediated anabolic biochemical pathways in Autism Spectrum Disorder and Fragile X Syndrome plasma and brain tissue. Scientific reports, 6, 26052.

Wu YE, et al. (2016) Genome-wide, integrative analysis implicates microRNA dysregulation in autism spectrum disorder. Nature neuroscience, 19(11), 1463.

Mor M, et al. (2015) Hypomethylation of miR-142 promoter and upregulation of microRNAs that target the oxytocin receptor gene in the autism prefrontal cortex. Molecular autism, 6, 46.

Hu VW, et al. (2015) Investigation of sex differences in the expression of RORA and its transcriptional targets in the brain as a potential contributor to the sex bias in autism. Molecular autism, 6, 7.

Chandley MJ, et al. (2015) NTRK2 expression levels are reduced in laser captured pyramidal neurons from the anterior cingulate cortex in males with autism spectrum disorder. Molecular autism, 6, 28.

Lukose R, et al. (2015) Organization of the human superior olivary complex in 15q duplication syndromes and autism spectrum disorders. Neuroscience, 286, 216.

Nicolini C, et al. (2015) Decreased mTOR signaling pathway in human idiopathic autism and in rats exposed to valproic acid. Acta neuropathologica communications, 3, 3.

Ander BP, et al. (2015) Atypical miRNA expression in temporal cortex associated with dysregulation of immune, cell cycle, and other pathways in autism spectrum disorders. Molecular autism, 6, 37.

Li J, et al. (2014) Integrated systems analysis reveals a molecular network underlying autism spectrum disorders. *Molecular systems biology*, 10(12), 774.

Irimia M, et al. (2014) A highly conserved program of neuronal microexons is misregulated in autistic brains. *Cell*, 159(7), 1511.

Skefos J, et al. (2014) Regional alterations in purkinje cell density in patients with autism. *PloS one*, 9(2), e81255.

Gupta S, et al. (2014) Transcriptome analysis reveals dysregulation of innate immune response genes and neuronal activity-dependent genes in autism. *Nature communications*, 5, 5748.

Nardone S, et al. (2014) DNA methylation analysis of the autistic brain reveals multiple dysregulated biological pathways. *Translational psychiatry*, 4(9), e433.

Hutsler JJ, et al. (2013) Sigmoid fits to locate and characterize cortical boundaries in human cerebral cortex. *Journal of neuroscience methods*, 212(2), 242.