Resource Summary Report

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MRC London Neurodegenerative Diseases Brain Bank

RRID:SCR_013839
Type: Tool

Proper Citation

MRC London Neurodegenerative Diseases Brain Bank (RRID:SCR_013839)

Resource Information

URL: http://www.kcl.ac.uk/ioppn/depts/cn/research/MRC-London-Neurodegenerative-Diseases-Brain-Bank/MRC-London-Neurodegenerative-Diseases-Brain-Bank.aspx

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Description: A biomaterial supply resource which provides high quality, clinically and neuropathologically well-characterised human brain and spinal cord tissue. The Brain Bank focuses on neurodegenerative diseases such as Alzheimer's Disease (AD), Frontotemperal dementias (FTD) and Motor Neurone Disease (MND). However, it also contains tissues for the study of HIV, Autism and Schizophrenia, and movement disorders.

Synonyms: The Brain Bank

Resource Type: biomaterial supply resource, material resource

Keywords: biomaterial supply resource, brain, brain tissue, brain bank, neurodegenerative disorder, neurodegenerative disease, spinal cord tissue

Funding: Medical Research Council

Availability: Available to the research community

Resource Name: MRC London Neurodegenerative Diseases Brain Bank

Resource ID: SCR_013839

License URLs: http://www.kcl.ac.uk/terms/index.aspx

Record Creation Time: 20220129T080318+0000

Record Last Update: 20250419T055404+0000

Ratings and Alerts

No rating or validation information has been found for MRC London Neurodegenerative Diseases Brain Bank.

No alerts have been found for MRC London Neurodegenerative Diseases Brain Bank.

Data and Source Information

Source: SciCrunch Registry

Usage and Citation Metrics

We found 31 mentions in open access literature.

Listed below are recent publications. The full list is available at NIF.

Baek JH, et al. (2019) GRP78 Level Is Altered in the Brain, but Not in Plasma or Cerebrospinal Fluid in Parkinson's Disease Patients. Frontiers in neuroscience, 13, 697.

Li P, et al. (2019) Epigenetic dysregulation of enhancers in neurons is associated with Alzheimer's disease pathology and cognitive symptoms. Nature communications, 10(1), 2246.

Ashton NJ, et al. (2019) Increased plasma neurofilament light chain concentration correlates with severity of post-mortem neurofibrillary tangle pathology and neurodegeneration. Acta neuropathologica communications, 7(1), 5.

An H, et al. (2019) ALS-linked FUS mutations confer loss and gain of function in the nucleus by promoting excessive formation of dysfunctional paraspeckles. Acta neuropathologica communications, 7(1), 7.

Kattuah W, et al. (2019) Heterogeneous Nuclear Ribonucleoprotein E2 (hnRNP E2) Is a Component of TDP-43 Aggregates Specifically in the A and C Pathological Subtypes of Frontotemporal Lobar Degeneration. Frontiers in neuroscience, 13, 551.

Hainsworth AH, et al. (2018) Super-resolution imaging of subcortical white matter using stochastic optical reconstruction microscopy (STORM) and super-resolution optical fluctuation imaging (SOFI). Neuropathology and applied neurobiology, 44(4), 417.

Shelkovnikova TA, et al. (2018) Protective paraspeckle hyper-assembly downstream of TDP-43 loss of function in amyotrophic lateral sclerosis. Molecular neurodegeneration, 13(1), 30.

Spreafico M, et al. (2018) Multiple Layers of CDK5R1 Regulation in Alzheimer's Disease Implicate Long Non-Coding RNAs. International journal of molecular sciences, 19(7).

Montibeller L, et al. (2018) Amyotrophic lateral sclerosis (ALS) and Alzheimer's disease (AD) are characterised by differential activation of ER stress pathways: focus on UPR target genes. Cell stress & chaperones, 23(5), 897.

Alghamdi A, et al. (2017) Reduction of RPT6/S8 (a Proteasome Component) and Proteasome Activity in the Cortex is Associated with Cognitive Impairment in Lewy Body Dementia. Journal of Alzheimer's disease : JAD, 57(2), 373.

Sproviero W, et al. (2017) ATXN2 trinucleotide repeat length correlates with risk of ALS. Neurobiology of aging, 51, 178.e1.

Viana J, et al. (2017) Schizophrenia-associated methylomic variation: molecular signatures of disease and polygenic risk burden across multiple brain regions. Human molecular genetics, 26(1), 210.

Ditsworth D, et al. (2017) Mutant TDP-43 within motor neurons drives disease onset but not progression in amyotrophic lateral sclerosis. Acta neuropathologica, 133(6), 907.

Napolitano F, et al. (2017) Decreased Rhes mRNA levels in the brain of patients with Parkinson's disease and MPTP-treated macaques. PloS one, 12(7), e0181677.

Lee YB, et al. (2017) C9orf72 poly GA RAN-translated protein plays a key role in amyotrophic lateral sclerosis via aggregation and toxicity. Human molecular genetics, 26(24), 4765.

Parviainen L, et al. (2017) Glial cells are functionally impaired in juvenile neuronal ceroid lipofuscinosis and detrimental to neurons. Acta neuropathologica communications, 5(1), 74.

Koss DJ, et al. (2016) Soluble pre-fibrillar tau and ?-amyloid species emerge in early human Alzheimer's disease and track disease progression and cognitive decline. Acta neuropathologica, 132(6), 875.

Kurbatskaya K, et al. (2016) Upregulation of calpain activity precedes tau phosphorylation and loss of synaptic proteins in Alzheimer's disease brain. Acta neuropathologica communications, 4, 34.

Mirza A, et al. (2016) The Identification of Aluminum in Human Brain Tissue Using Lumogallion and Fluorescence Microscopy. Journal of Alzheimer's disease : JAD, 54(4), 1333.

Niblock M, et al. (2016) Retention of hexanucleotide repeat-containing intron in C9orf72 mRNA: implications for the pathogenesis of ALS/FTD. Acta neuropathologica communications, 4, 18.