

Resource Summary Report

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CHDI Foundation

RRID:SCR_004622

Type: Tool

Proper Citation

CHDI Foundation (RRID:SCR_004622)

Resource Information

URL: <http://www.chdifoundation.org/>

Proper Citation: CHDI Foundation (RRID:SCR_004622)

Description: A private, not-for-profit research organization that serves as an international collaborative enabler in order to discover drugs that slow the progression of Huntington's disease (HD). The activities of CHDI extend from exploratory biology to the identification and validation of therapeutic targets, and from drug discovery and development to clinical studies and trials. CHDI works with biotech and pharmaceutical companies and funds and works with academic HD researchers at universities.

Abbreviations: CHDI

Synonyms: CHDI Foundation Inc

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

Keywords: huntington's disease, antibody, collaborative enabler, international network, funding resource

Availability: Available to academic huntington's disease researchers

Resource Name: CHDI Foundation

Resource ID: SCR_004622

Alternate IDs: nlx_143844

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Record Creation Time: 20220129T080225+0000

Record Last Update: 20260805T044111+0000

Ratings and Alerts

No rating or validation information has been found for CHDI Foundation.

No alerts have been found for CHDI Foundation.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 26 mentions in open access literature.

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

Akkermans J, et al. (2024) Biodistribution and dosimetry of the PET radioligand [18F]CHDI-650 in mice for detection of mutant huntingtin aggregates. EJNMMI research, 14(1), 126.

Reindl W, et al. (2019) Meso scale discovery-based assays for the detection of aggregated huntingtin. PloS one, 14(3), e0213521.

Davis MI, et al. (2018) The cannabinoid-1 receptor is abundantly expressed in striatal striosomes and striosome-dendron bouquets of the substantia nigra. PloS one, 13(2), e0191436.

Schuldenzucker V, et al. (2017) Behavioral testing of minipigs transgenic for the Huntington gene-A three-year observational study. PloS one, 12(10), e0185970.

Coffey SR, et al. (2017) Peripheral huntingtin silencing does not ameliorate central signs of disease in the B6.HttQ111/+ mouse model of Huntington's disease. PloS one, 12(4), e0175968.

Hensman Moss DJ, et al. (2017) Quantification of huntingtin protein species in Huntington's disease patient leukocytes using optimised electrochemiluminescence immunoassays. PloS one, 12(12), e0189891.

Dietrich P, et al. (2017) Elimination of huntingtin in the adult mouse leads to progressive behavioral deficits, bilateral thalamic calcification, and altered brain iron homeostasis. PLoS genetics, 13(7), e1006846.

Bayram-Weston Z, et al. (2016) Comparison of mHTT Antibodies in Huntington's Disease Mouse Models Reveal Specific Binding Profiles and Steady-State Ubiquitin Levels with Disease Development. PloS one, 11(5), e0155834.

Kuljis DA, et al. (2016) Sex Differences in Circadian Dysfunction in the BACHD Mouse Model of Huntington's Disease. PloS one, 11(2), e0147583.

Valcárcel-Ocete L, et al. (2015) Exploring Genetic Factors Involved in Huntington Disease Age of Onset: E2F2 as a New Potential Modifier Gene. *PloS one*, 10(7), e0131573.

Ruzo A, et al. (2015) Discovery of novel isoforms of huntingtin reveals a new hominid-specific exon. *PloS one*, 10(5), e0127687.

Huang B, et al. (2015) Scalable production in human cells and biochemical characterization of full-length normal and mutant huntingtin. *PloS one*, 10(3), e0121055.

Nagy D, et al. (2015) Application of neurophysiological biomarkers for Huntington's disease: evaluating a phosphodiesterase 9A inhibitor. *Experimental neurology*, 263, 122.

Collins LM, et al. (2015) Novel Nut and Bolt Task Quantifies Motor Deficits in Premanifest and Manifest Huntington's Disease. *PLoS currents*, 7.

Larson E, et al. (2015) Age-, tissue- and length-dependent bidirectional somatic CAG•CTG repeat instability in an allelic series of R6/2 Huntington disease mice. *Neurobiology of disease*, 76, 98.

Miller JR, et al. (2015) Mutant Huntingtin Does Not Affect the Intrinsic Phenotype of Human Huntington's Disease T Lymphocytes. *PloS one*, 10(11), e0141793.

Toga AW, et al. (2015) Sharing big biomedical data. *Journal of big data*, 2.

Kolodziejczyk K, et al. (2014) Striatal synaptic dysfunction and hippocampal plasticity deficits in the Hu97/18 mouse model of Huntington disease. *PloS one*, 9(4), e94562.

Zhu LJ, et al. (2014) CRISPRseek: a bioconductor package to identify target-specific guide RNAs for CRISPR-Cas9 genome-editing systems. *PloS one*, 9(9), e108424.

Tourette C, et al. (2014) The Wnt receptor Ryk reduces neuronal and cell survival capacity by repressing FOXO activity during the early phases of mutant huntingtin pathogenicity. *PLoS biology*, 12(6), e1001895.