

Resource Summary Report

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NIMH Intramural Research Program Clinical Brain Disorders Branch

RRID:SCR_008728

Type: Tool

Proper Citation

NIMH Intramural Research Program Clinical Brain Disorders Branch (RRID:SCR_008728)

Resource Information

URL: <http://cbdb.nimh.nih.gov/>

Proper Citation: NIMH Intramural Research Program Clinical Brain Disorders Branch (RRID:SCR_008728)

Description: THIS RESOURCE IS NO LONGER IN SERVICE, documented on February 07, 2013. A multidisciplinary neuroscience laboratory in which basic and clinical scientists work side by side exploring neural mechanisms and models of mental and cognitive function and of neuropsychiatric illness. Experiments are performed at many levels of inquiry, from basic molecular biology of the gene to clinical examinations of patients. A major area of investigation of this laboratory is the genetic mechanisms implicated in the pathogenesis of schizophrenia and its treatment. The laboratory is organized as a multi-disciplinary team of investigators with a common mission: to identify and fully characterize basic genetic and neurobiological mechanisms of schizophrenia and related cognitive and emotional disorders. The various components of this effort are centered various different units or divisions represented by groups of investigators, at various levels of training and experience, working on related experiments. The Director of the Branch and of the Genes, Cognition and Psychosis Program (GCAP) is Daniel R. Weinberger, M.D. The CBDB is the principle research laboratory in the created (2003) Genes, Cognition, and Psychosis Program (GCAP) of the NIMH. After twelve years of residing on the pastoral grounds of St. Elizabeths Hospital, in Southeast Washington, CBDB moved back to the main NIH campus in Bethesda, Maryland in 1998. While the unique setting of St. Elizabeths is irreplaceable, we have occupied beautiful new laboratories and clinic spaces that were created for us, and we are in the mainstream of NIH life., THIS RESOURCE IS NO LONGER IN SERVICE. Documented on September 16,2025.

Abbreviations: CBDB

Synonyms: NIMH Clinical Brain Disorders Branch, Clinical Brain Disorders Branch

Resource Type: topical portal, data or information resource, portal

Keywords: mental function, cognitive function, gene, clinical, treatment, pathogen

Related Condition: Schizophrenia, Neuropsychiatric illness, Cognitive disorder, Emotional disorder

Funding: NIMH

Availability: THIS RESOURCE IS NO LONGER IN SERVICE

Resource Name: NIMH Intramural Research Program Clinical Brain Disorders Branch

Resource ID: SCR_008728

Alternate IDs: nlx_143685

Record Creation Time: 20220129T080249+0000

Record Last Update: 20260731T042801+0000

Ratings and Alerts

No rating or validation information has been found for NIMH Intramural Research Program Clinical Brain Disorders Branch.

No alerts have been found for NIMH Intramural Research Program Clinical Brain Disorders Branch.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 13 mentions in open access literature.

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

Pretta A, et al. (2025) Early-onset colorectal cancer patients exhibit a distinct molecular fingerprint: insights from a large-scale NGS study of 1209 patients. ESMO open, 10(9), 105756.

Wei SM, et al. (2024) Altered pubertal timing in 7q11.23 copy number variations and associated genetic mechanisms. iScience, 27(3), 109113.

Na PJ, et al. (2021) Prevalence, risk and protective factors associated with suicidal ideation during the COVID-19 pandemic in U.S. military veterans with pre-existing psychiatric conditions. Journal of psychiatric research, 137, 351.

Na PJ, et al. (2021) Factors associated with post-traumatic growth in response to the COVID-19 pandemic: Results from a national sample of U.S. military veterans. Social science & medicine (1982), 289, 114409.

Deep-Soboslay A, et al. (2019) African-American and Caucasian participation in postmortem human brain donation for neuropsychiatric research. *PloS one*, 14(10), e0222565.

Savage SA, et al. (2013) Genome-wide association study identifies two susceptibility loci for osteosarcoma. *Nature genetics*, 45(7), 799.

Greenwood TA, et al. (2013) Genome-wide association study of irritable vs. elated mania suggests genetic differences between clinical subtypes of bipolar disorder. *PloS one*, 8(1), e53804.

Oskvig DB, et al. (2012) Maternal immune activation by LPS selectively alters specific gene expression profiles of interneuron migration and oxidative stress in the fetus without triggering a fetal immune response. *Brain, behavior, and immunity*, 26(4), 623.

Lehmann ML, et al. (2010) NF-kappaB activity affects learning in aversive tasks: possible actions via modulation of the stress axis. *Brain, behavior, and immunity*, 24(6), 1008.

Kakiuchi C, et al. (2005) Functional polymorphisms of HSPA5: possible association with bipolar disorder. *Biochemical and biophysical research communications*, 336(4), 1136.

Kassed CA, et al. (2004) NF-kappaB p50-deficient mice show reduced anxiety-like behaviors in tests of exploratory drive and anxiety. *Behavioural brain research*, 154(2), 577.

Dick DM, et al. (2003) Genomewide linkage analyses of bipolar disorder: a new sample of 250 pedigrees from the National Institute of Mental Health Genetics Initiative. *American journal of human genetics*, 73(1), 107.

Hohmann AG, et al. (1999) Localization of central cannabinoid CB1 receptor messenger RNA in neuronal subpopulations of rat dorsal root ganglia: a double-label in situ hybridization study. *Neuroscience*, 90(3), 923.