Resource Summary Report

Generated by NIF on Sep 2, 2026

NeuroMab

RRID:SCR_003086
Type: Tool

Proper Citation

NeuroMab (RRID:SCR_003086)

Resource Information

URL: http://neuromab.ucdavis.edu/

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Description: A national mouse monoclonal antibody generating resource for biochemical and immunohistochemical applications in mammalian brain. NeuroMabs are generated from mice immunized with synthetic and recombinant immunogens corresponding to components of the neuronal proteome as predicted from genomic and other large-scale cloning efforts. Comprehensive biochemical and immunohistochemical analyses of human, primate and non-primate mammalian brain are incorporated into the initial NeuroMab screening procedure. This yields a subset of mouse mAbs that are optimized for use in brain (i.e. NeuroMabs): for immunocytochemical-based imaging studies of protein localization in adult, developing and pathological brain samples, for biochemical analyses of subunit composition and post-translational modifications of native brain proteins, and for proteomic analyses of native brain protein networks. The NeuroMab facility was initially funded with a five-year U24 cooperative grant from NINDS and NIMH. The initial goal of the facility for this funding period is to generate a library of novel NeuroMabs against neuronal proteins, initially focusing on membrane proteins (receptors/channels/transporters), synaptic proteins, other neuronal signaling molecules, and proteins with established links to disease states. The scope of the facility was expanded with supplements from the NIH Blueprint for Neuroscience Research to include neurodevelopmental targets, the NIH Roadmap for Medical Research to include epigenetics targets, and NIH Office of Rare Diseases Research to include rare disease targets. These NeuroMabs will then be produced on a large scale and made available to the neuroscience research community on an inexpensive basis as tissue culture supernatants or purified immunoglobulin by Antibodies Inc. The UC Davis/NIH NeuroMab Facility makes NeuroMabs available directly to end users and is unable to accommodate sales to distributors for third party distribution. Note, NeuroMab antibodies are now offered through antibodiesinc.

Abbreviations: NeuroMab

Synonyms: UCDavis/NIH NeuroMab Facility, antibodies.inc, antibodiesinc.com, antibodiesinc

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

Keywords: antibody, brain, channel, disease-related protein, k channel subunit, mab, mammalian, membrane protein, monoclonal antibody, mouse, neuronal monoclonal antibody, neuronal protein, neuronal signaling molecule, reagent, receptor, research reagent, synaptic protein, transporter

Funding: NINDS ;
NIMH ;
NIH Blueprint for Neuroscience Research ;
NIH Roadmap for Medical Research ;
Office of Rare Diseases Research ;
Antibodies Inc.

Availability: Free, Freely available

Resource Name: NeuroMab

Resource ID: SCR_003086

Alternate IDs: grid.482686.6, nif-0000-00175

Alternate URLs: https://ror.org/00fyrp007

Record Creation Time: 20220129T080217+0000

Record Last Update: 20260829T182834+0000

Ratings and Alerts

No rating or validation information has been found for NeuroMab.

No alerts have been found for NeuroMab.

Data and Source Information

Source: SciCrunch Registry

Usage and Citation Metrics

We found 1859 mentions in open access literature.

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

Kovács ZJ, et al. (2026) Nanoscale CaV channel reorganization links ?-synuclein pathology to calcium-dependent transcriptional dysregulation. bioRxiv : the preprint server for biology.

Wang D, et al. (2026) Systemic delivery of synapsin-promoted caveolin-1 overexpression ameliorates pathological TDP-43-induced cognitive decline and neurodegenerative changes. Alzheimer's & dementia : the journal of the Alzheimer's Association, 22(5), e71450.

Wang J, et al. (2026) Electrophysiological Characterization of Murine Vestibular Efferent Neurons and Modulation by Acute Peripheral Vestibular Deprivation. Neuroscience bulletin, 42(3), 589.

Llovera G, et al. (2026) Blocking microglial reactivity via purinergic receptors prevents subacute cognitive deficits after TIA. EMBO molecular medicine, 18(4), 1150.

Osaki T, et al. (2026) Early differential impact of MeCP2 mutations on functional networks in Rett syndrome patient-derived human cortical organoids. Nature communications, 17(1).

Foote MK, et al. (2026) Non-Synaptic Mechanism of Ocular Dominance Plasticity. ASN neuro, 18(1), 2616376.

Perdigão C, et al. (2026) Encephalopathy-linked UFM1 variants impede neuronal protein translation, development, and function. EMBO molecular medicine, 18(4), 1265.

Chatragadda B, et al. (2026) Exploring the neuroprotective effects of phytocannabinoids on oxygen-glucose deprived neurons in an in vitro model of stroke. Journal of cannabis research, 8(1), 29.

Wyeth M, et al. (2026) Numbers of Granule Cells and GABAergic Boutons Are Correlated in Shrunken Sclerotic Hippocampi of Sea Lions with Temporal Lobe Epilepsy. eNeuro, 13(3).

Vu DT, et al. (2026) Multi-cohort, cross-species urinary proteomics reveals signatures of LRRK2 dysfunction in Parkinson's disease. Molecular systems biology, 22(5), 712.

Wang Y, et al. (2026) The axon initial segment-associated microglia regulate neuronal activity and visual perception. Cell research, 36(4), 249.

Chen R, et al. (2026) Pathological TDP-43 filaments accumulate at synapses and cause synaptic dysfunction. bioRxiv : the preprint server for biology.

Hanes CM, et al. (2026) A new mouse mutant with a discrete mutation in Pcdhgc5 reveals that the Protocadherin ?C5 isoform is not essential for dendrite arborization in the cerebral cortex. PloS one, 21(3), e0344863.

Yang B, et al. (2026) Ccdc57 regulates cilia and left-right patterning in Xenopus. Biology open, 15(2).

Thakur V, et al. (2026) Glycine receptors in circulating white blood cells regulated by neuroinflammation. Frontiers in immunology, 17, 1749275.

Athanasopoulos PS, et al. (2026) Regulation between LRRK2 and PP2A signaling in cellular models of Parkinson's disease. The Biochemical journal, 483(6), 1097.

Aldahabi M, et al. (2026) Selective, genetically induced increase in synaptic vesicle priming. Science advances, 12(15), eaee6848.

Zhu J, et al. (2026) Neurofascin stabilization by contactin-associated protein-like 2 and CTCF alleviates mitochondrial dysfunction in Schwann cells during facial nerve injury. The Journal of biological chemistry, 302(2), 111090.

Benske TM, et al. (2026) A GluN2B disease-associated variant promotes the degradation of NMDA receptors via autophagy. The Journal of biological chemistry, 111147.

Coceano G, et al. (2026) Quantitative optical nanoscopy of mitochondrial-derived vesicles in neurons classifies pre-peroxisomal and clearing organelles. Nature communications, 17(1), 419.