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PubMed is also available via pubmed.gov but access the database through the open web will not grant the user access to full text through the USF library.

### SCOPE AND COVERAGE:

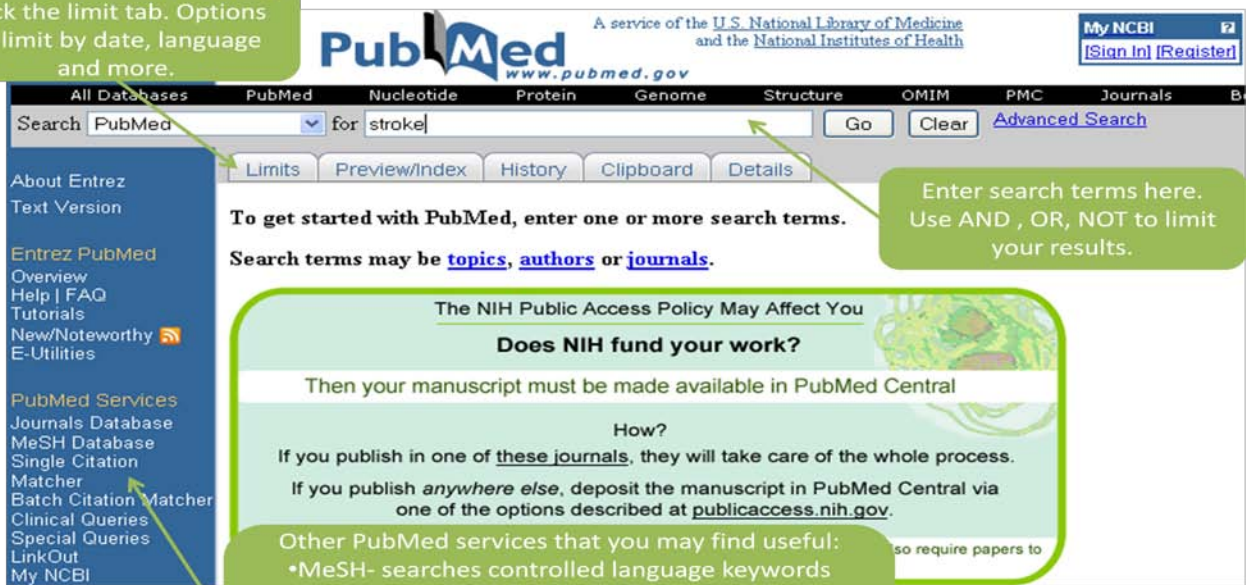
PubMed is a clinical and biomedical database that contains journal articles, consumer health information, clinical trial information, government guidelines, article reviews and systematic reviews and more. It is one of

many databases run by the National Library of Medicine (NLM) and the National Institutes of Health (NIH). The most popular services of PubMed are the simple search, shown below, the Single Citation Matcher, and a MeSH search, described on another handout.

PubMed is only an abstracting and indexing service, *NO FULL TEXT IS AVAILABLE THROUGH PUBMED*. However, the Find It button is found on the abstract page of each item and occasionally there will be links to free full text through PubMed or BioMed Central. Other links advertising full text availability are usually ads and the full text will be available only through purchase or subscription.

## PUBMED MAIN HOME PAGE

To limit your search further, click the limit tab. Options to limit by date, language and more.



The screenshot shows the PubMed main home page with the following elements and annotations:

- Search Bar:** Contains the text "PubMed" and "for stroke". A green callout box points to the search input area with the text: "Enter search terms here. Use AND , OR, NOT to limit your results."
- Navigation Tabs:** Includes "Limits", "Preview/Index", "History", "Clipboard", and "Details". A green callout box points to the "Limits" tab with the text: "To limit your search further, click the limit tab. Options to limit by date, language and more."
- Left Sidebar:** Contains links for "About Entrez", "Entrez PubMed", "PubMed Services", and "My NCBI". A green callout box points to the "Single Citation Matcher" link with the text: "Other PubMed services that you may find useful: •MeSH- searches controlled language keywords supplied by authors. •Single Citation Matcher- enter part of a citation and be taken directly to the article. •My NCBI- save searches and be updated when new items matching your search are available."
- Main Content:** Features a section titled "The NIH Public Access Policy May Affect You" with sub-sections "Does NIH fund your work?" and "Then your manuscript must be made available in PubMed Central".
- Top Right:** Includes a "My NCBI" button with "Sign In" and "Registered" links.

## PUBMED RESULTS PAGE

History tab allows you to see previous searches and combine them to create a narrower search.

Change the format of the results page in the "display" "show" and "sort by" drop down menus.

Number of items matching your search.

Number of literature or systematic reviews matching your search.

The PMID is a unique identifier for each item. Type it in the search box to go directly to the item's record.

Click a title to be taken to the abstract.

Also try:

- ischemic **stroke**
- acute **stroke**
- stroke** rehabilitation
- stroke** prevention
- stroke** risk

Recent Activity

- stroke (12) MeSH
- stroke (141160) PubMed

Search PubMed for stroke

Display Summary Show 20 Sort By Send to

All: 141160 Review: 18959

1 - 20 of 141160

1: [Rethinking a drug treatment failure on a traditional ALS target.](#)  
Maragakis NJ.  
Exp Neurol. 2009 Apr;216(2):254-7.  
PMID: 19309797 [PubMed - in process]  
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2: [Arrestin3 mediates D\(2\) dopamine receptor internalization.](#)  
Skinbjerg M, Ariano MA, Thorsell A, Hellig M, Halldin C, Innis RB, Sibley DR.  
Synapse. 2009 Mar 23;63(7):621-624. [Epub ahead of print] No abstract available.  
PMID: 19309759 [PubMed - as supplied by publisher]  
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3: [Inadvertent subclavian artery catheter placement complicated by stroke. Endovascular management and review.](#)  
Jahromi BS, Tummala RP, Levy EI.  
Catheter Cardiovasc Interv. 2008 Nov 7;73(5):706-711

## PUBMED INDIVIDUAL ITEM PAGE

A "0" in the review tab means the article is not any kind of review.

Someone published an editorial comment regarding this article. View it here.

Links to other articles that have similar subject terms.

Click the author's name to see all his or her works in PubMed.

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Related Articles

- Nordihydroguaiaretic acid increases glutamate uptake in vitro and in vivo: therapeutic implications for amyotrophic lateral sclerosis [Exp Neurol. 2008]
- Increased internalisation and degradation of GLT-1 oligal glutamate transporter in a cell model for familial amyotrophic lateral sclerosis [J Cell Sci. 2004]
- Review: Update on the glutamatergic neurotransmitter system and the role of excitotoxicity in amyotrophic lateral sclerosis [Muscle Nerve. 2002]
- Review: Dementia of Alzheimer's disease and other neurodegenerative disorders--memantine, a review [Pharmacol Res. 2005]
- Loss of metabotropic glutamate receptor-mediated regulation of glutamate transport in chemically activated astrocytes [J Neurochem. 2006]

Recent Activity

- Rethinking a drug treatment failure on a traditional ALS target.
- Arrestin3 mediates D(2) dopamine receptor internalization.
- stroke (141160)
- ((aphasia)) AND ((Stroke... (723)
- aphasia (10848) PubMed

All: 1 Review: 0

1: [Exp Neurol.](#) 2009 Apr;216(2):254-7.  
Comment on:  
[Exp Neurol.](#) 2008 Sep;213(1):229-37.

**Rethinking a drug treatment failure on a traditional ALS target.**

**Maragakis NJ.**  
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In a recent issue of Experimental Neurology, Boston-Howes and colleagues used an assay of glutamate transport to screen 1040 FDA approved drugs in an attempt to identify compounds that would increase glutamate transport, a central function of astrocytes, and a potential biological target for neuroprotection for a variety of neurological disorders. They identified the compound nordihydroguaiaretic acid (NDGA) as a particularly good candidate for inducing glutamate transport. Pharmacological increases in glutamate transport could have a number of potential applications to diseases of the nervous system where glutamate excitotoxicity is thought to be a contributing factor to pathogenesis including Amyotrophic Lateral Sclerosis, Alzheimer's disease, Parkinson's disease, stroke, and epilepsy among others. They chose to test this compound in a model of Amyotrophic Lateral Sclerosis (ALS)--the SOD1G93A mouse. In both human ALS and rodent models of the disease, glutamate excitotoxicity and abnormalities in glutamate transporter biology more specifically, have been implicated in ALS disease propagation. Interestingly, while the authors nicely demonstrate that NDGA has a biological effect on glutamate transport in normal (wild type) central nervous system tissues both in vitro and in vivo, it was the somewhat unexpected (and often overlooked) findings in the ALS mouse model that makes this manuscript notable and suggests that rethinking translational approaches to drug therapies in ALS may be on the horizon.

PMID: 19309797 [PubMed - in process]

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