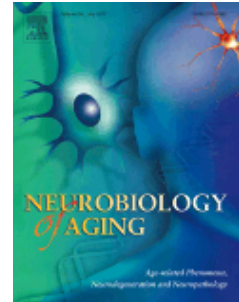




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DESCRIPTION

Neurobiology of Aging publishes the results of studies in **behavior, biochemistry, cell biology, endocrinology, molecular biology, morphology, neurology, neuropathology, pharmacology, physiology** and **protein chemistry** in which the primary emphasis involves mechanisms of nervous system changes with age or diseases associated with age. Reviews and primary research articles are included, occasionally accompanied by open peer commentary. Letters to the Editor and brief communications are also acceptable. Brief reports of highly time-sensitive material are usually treated as rapid communications in which case editorial review is completed within six weeks and publication scheduled for the next available issue. The accepted abbreviation for *Neurobiology of Aging* for bibliographic citation is *Neurobiol.Aging*

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Neuroscientists, Molecular Biologists, Gerontologists.

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AIMS AND SCOPE

Neurobiology of Aging publishes the results of studies in behavior, biochemistry, cell biology, endocrinology, molecular biology, morphology, neurology, neuropathology, pharmacology, physiology and protein chemistry in which the primary emphasis involves mechanisms of nervous system changes with age or diseases associated with age. Only original articles will be accepted. Reviews and primary research articles are included, occasionally accompanied by open peer commentary. Letters to the Editor and brief communications are also acceptable. Brief reports of highly time-sensitive material are usually treated as rapid communications in which case editorial review is completed within 6 weeks and publication scheduled for the next available issue. Negative Results will be published as one journal page (3 double-spaced typed manuscript pages), with supplementary material to be posted at the journal website.

Authors are referred to the following published editorial policy statements: Coleman, P.D. How old is old? *Neurobiol. Aging* 25:1;2004. Coleman, P.D.; Finch, C.E.; Joseph, J. The need for multiple time points in aging studies. *Neurobiol. Aging* 25:3-4;2004. Finch, C.E. Middle-age: An evolving frontier in gerontology. *Neurobiol. Aging* 12:1-2;1991. West, M.J. New stereological methods for counting neurons. *Neurobiol. Aging* 14:275-285;1993. West, M.J.; Coleman, P.D. How to count. *Neurobiol. Aging* 17:503;1996. Frisoni, G.B.; Coleman, P.D. Mild cognitive impairment: instructions for use at *Neurobiology of Aging*. *Neurobiol. Aging* 32:761-762;2011.

GUIDELINES

Genetic Analysis of Disease in the Era of Whole Genome Analysis and Public Databases. Over the past 5 years genetic analysis has changed almost beyond recognition. We now have the technology to assess association between any phenotype and alleles across the genome in a single analysis. Furthermore, these data are stored in publicly available databases such as dbGAP (www.ncbi.nlm.nih.gov/gap) and Alzgene (www.alzgene.org) where they are accessible and can be used in ongoing meta-analyses. In this environment, researchers should consider carefully the extent to which analyses they report substantively contribute to the literature.

In the future, we will expect authors of any manuscripts submitted to access these databases before submission. While there are circumstances when limited analyses are appropriate, in general, clearly whole genome analyses are the way forward and there is no doubt that findings which come out of such studies are more reliable than those which come from candidate gene analyses. Additionally, we caution against the overinterpretation of analyses of secondary phenotypes (such as age of onset, or rate of cognitive decline).

In studies where whole genome analyses are reported, we will always expect full summary statistics to be made available alongside the publication.

We note that for many major phenotypes, there remain no whole genome reports. Clear examples include Alzheimer's disease in populations outside of Europeans. We would welcome such studies.

Genetic Reports. It is our wish to provide rapid review of high quality-genetic studies for traits and conditions related to normal and diseased aging brain, whether these are positive or negative in outcome.

Genetic analysis and technologies have moved on and we want the studies we publish to be definitive. With this in mind, we suggest the following should be considered when you are submitting to *Neurobiology of Aging*:

(1) Does the study assess the whole gene? We would suggest that any analysis should include a haplotypic analysis of the whole gene of interest rather than single SNPs unless the SNP tested is believed to be the functional SNP.

(2) How is your study powered? This question should be addressed whether the study is positive or negative. In general, for dichotomous traits one should aim at reasonable numbers (cases and controls each of 500 is a good rule of thumb). These numbers can usually be achieved through collaboration.

(3) Is your study a hypothesis-generating or a hypothesis-testing study? Does it inform as to mechanism? In general, reviewers and editors are very wary of effects that purport to be present in only a subset of cases. Such contrasts should be pre-specified and designs appropriately powered to test for the effect of sex and other variables. A clear negative study has value. Digging around in data to generate positive findings does the field a disservice.

(4) Have your sample series been used in other studies? Clearly these should always be referenced so the audience can assess how much risk may have been reported to have been found in any sample series.

(5) Are there online data sources in which you can also assess your SNPs? There are now online resources of case control series for Alzheimer's disease, Parkinson's disease and brain gene expression. The number of these resources is increasing all the time: any association studies for which there is already data should reference and include these data, perhaps as secondary sample series.

These are not rules, but guidelines.

NB: The full text of Genetic Reports manuscripts submitted after November 30th 2010, if accepted, will be published as **e-pub only**. The full text of such manuscripts will appear online within 40-50 days of acceptance, with the abstract appearing in the next available print edition as well. The abstract in print will contain appropriate reference to the complete e-pub manuscript.

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- (2) The potential of the marker (based on evidence in the literature or in the manuscript) for directly revealing insight into disease mechanisms.
- (3) The clinical potential of the marker for differential diagnosis or prediction of disease progression (based on data in the manuscript).
- (4) The reliability of the supporting data based on size of the sample studied and statistical validation.

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