

A Journal of Psychiatric Neuroscience and Therapeutics

Biological Psychiatry is the official journal of the Society of Biological Psychiatry. The Journal rapidly publishes reports of novel results on a broad range of topics related to the pathophysiology and treatment of major neuropsychiatric disorders. Both basic and clinical neuroscience contributions are encouraged, particularly those addressing genetic and environmental risk factors, neural circuitry and neurochemistry, and important new therapeutic approaches. Except where explicitly stated otherwise, Biological Psychiatry conforms to the guidelines set forth by the International Committee of Medical Journal Editors (ICMJE) (see Recommendations for the Conduct, Reporting, Editing, and Publication of Scholarly Work in Medical Journals [August 2013]: Available from http://www.ICMJE.org).

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- Krystal JH, Carter CS, Geschwind D, Manji HK, March JS, Nestler EJ, et al. (2008): It is time to take a stand for medical research and against terrorism targeting medical scientists. Biol Psychiatry 63:725–727.
- American Psychiatric Association (1994): Diagnostic and Statistical Manual of Mental Disorders, 4th ed. Washington, DC: American Psychiatric Press.
- Martin JH (1985): Properties of cortical neurons, the EEG, and the mechanisms of epilepsy. In: Kandel ER, Schwartz JH, editors. *Principles of Neural Science*, 2nd ed. New York: Elsevier, 461-471.

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always include estimates of power.

We realize that independent replication of an initial finding in the same manuscript may not be feasible in every case, but studies providing such replication of findings in an independent sample will be given highest priority. Confirmation of the functional consequences of a common disease-associated variant is useful information, but does not substitute for a rigorous demonstration of a statistically significant association. Analysis of pathways or candidate regional analysis is encouraged over single gene studies. Candidate gene studies must have strong positional or biological rationale or precedents in the literature that motivate gene choice.

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For studies of anonymous variants, there should generally be sufficiently dense marker coverage to allow a relatively comprehensive analysis of common variants within a gene or genes. Analysis of the extent of marker coverage using standard methods to assess linkage disequilibrium should be presented. If rare variants are being tested, the same method of assessment (sequencing, copy number assessment, etc.) should be used in both case and control groups.

We will consider both negative and positive association studies, as well as large replication studies. Negative studies should be based on an attempt to replicate previous studies. Power calculations considering reasonable effect sizes must be provided to show that the study had sufficient power to be informative.

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