

# Biological Psychiatry

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## TRANSLATING BIOLOGY TO TREATMENT IN SCHIZOPHRENIA

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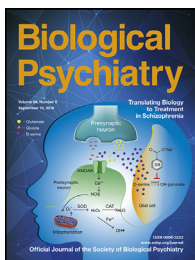
- 422 Sodium Benzoate, a D-Amino Acid Oxidase Inhibitor, Added to Clozapine for the Treatment of Schizophrenia: A Randomized, Double-Blind, Placebo-Controlled Trial**  
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- e49 Conference Report: Psychiatric Genomics Consortium Meeting: Pathways to Drugs, London, March 2017**  
*Hélène A. Gaspar, David A. Collier, Daniel H. Geschwind, Cathryn M. Lewis, Qingqin Li, Bryan L. Roth, Patrick F. Sullivan, and Gerome Breen*



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As depicted on the cover, sodium benzoate inhibits the activity of D-amino acid oxidase (DAAO) and thereby raises levels of D-amino acids such as D-serine, which are the neurotransmitters for the glycine site of the N-methyl-D-aspartate receptor (NMDAR). Lin *et al.* (in this issue, pages 422–432) found that adjuvant therapy of sodium benzoate improved symptomatology of patients with clozapine-resistant schizophrenia. Further, sodium benzoate not only inhibited DAAO but also increased catalase (a vital antioxidant), therefore reciprocally stabilizing the NMDAR-redox state. Overall, sodium benzoate may rescue both NMDAR hypofunction and oxidative stress in schizophrenia pathogenesis. SOD, superoxide dismutase.

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