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Translating Biology to Treatment in Schizophrenia

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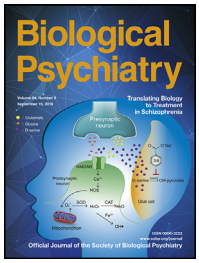

As depicted on the cover, sodium benzoate inhibits the activity of D-amino acid oxidase (DAAO) and thereby raises levels of D-amino acids such as D-serine, which are the neurotransmitters for the glycine site of the *N*-methyl-D-aspartate receptor (NMDAR). Lin *et al.* (in this issue, pages 422–432) 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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