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**Abstract:** • Memory impairments and psychiatric effects induced by repeated ketamine exposure. • Repeated ketamine exposure attenuated synaptic transmission and plasticity. • Dimethylglycine prevented ketamine-induced behavioral and synaptic deficits. Ketamine, an N-methyl- d -aspartate receptor (NMDAR) blocker, is gaining ground as a treatment option for depression. The occurrence of persistent psychosis and cognitive impairment after repeated use of ketamine remains a concern. N, N-dimethylglycine (DMG) is a nutrient supplement and acts as an NMDAR glycine site partial agonist. The objective of this study was to assess whether DMG could potentially prevent the behavioral and synaptic deficits in mice after repeated ketamine exposure. Male ICR mice received ketamine (20 mg/kg) from postnatal day (PN) 33–46, twice daily, for 14 days. The locomotor activity, novel location recognition test (NLRT), novel object recognition test (NORT), social interaction test, head twitch response induced by serotonergic hallucinogen, and the basal synaptic transmission and long-term potentiation (LTP) in the hippocampal slices were monitored after repeated ketamine treatment. Furthermore, the protective effects of repeated combined administration of DMG (30 and 100 mg/kg) with ketamine on behavioral abnormalities and synaptic dysfunction were assessed. The results showed that mice exhibited memory impairments, social withdrawal, increased head twitch response, reduced excitatory synaptic transmission, and lower LTP after repeated ketamine exposure. The ketamine-induced behavioral and synaptic deficits were prevented by co-treatment with DMG. In conclusion, these findings may pave a new path forward to developing a combination formula with ketamine and DMG for the treatment of depression and other mood disorders.

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**Subjects:** KETAMINE abuse; KETAMINE; LABORATORY mice; COGNITION disorders; NEURAL transmission; LONG-term potentiation; All Other Animal Production;

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