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ACUTE KETAMINE CHALLENGE EFFECTS ON VISUAL INFORMATION PROCESSING: IMPLICATIONS FOR PSYCHOSIS

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Introduction: Ketamine, a NMDA antagonist, replicates both cognitive and psychotic features of schizophrenia when administered to healthy volunteers. In this study, we aimed to test whether the administration of IV ketamine would replicate with cognitive and electrophysiological patterns that was observed in schizophrenia patients and schizotypal individuals.

Methtods: 44 healthy volunteers were randomised to receive IV infusion of ketamine or placebo. A 64 channel EEG kit was used to obtain event-related potentials in response to a working memory (WM) task. The two groups were compared in respect to their performance task as well as the amplitude of the P1 and P300 ERPs.
Results: The psychiatric scales scores (BPRS, CADSS) were significantly increased in the ketamine group when compared to saline. While there was no difference in terms of reaction times to the task, accuracy in the ketamine group worsened significantly with increase in working memory load than in controls. Ketamine significantly increased the P1 but lead to a decrease in P300.

Conclusion: In this study acute NMDA antagonism induced a WM deficit that was associated with visual processing and memory abnormalities. Specifically, ketamine increased the amplitude of the P1 potential and reduced the P300 amplitude. In addition P1 but not P300 predicted performance on the WM task. These effects could be mediated ketamine-induced acute glutamate release in the visual cortex, enhancing neuronal responses to visual stimuli and increasing the signal-to-noise ratio which in turn disrupted higher order cognitive function.

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