P-1260 - NEUROCHEMICAL MODULATION OF AUDITORY PROCESSING IN HEALTHY CONTROLS STRATIFIED FOR LOW AND HIGH AUDITORY HALLUCINATORY EXPERIENCES

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Introduction: Contributing to poor global functioning, auditory hallucinations (AH) also interfere with elementary cognitive processes, including auditory discrimination. This is evidenced in schizophrenic (SZ) hallucinators (vs. non-hallucinators) by a greater reduction of the MMN, an auditory event-related brain potential (ERP) generated in part by NMDA receptor activity and normalized with nicotinic (nACh) agonist treatment.
Objectives: To increase our understanding of NMDA-nACh interactions with auditory processing, using healthy young adults varying in their degree of experience with AH, thereby reducing the confounding influence of illness chronicity and medication associated with the study of SZ patients.
Aims: To investigate MMN differences between low and high AH subjects during separate and combined administration of ketamine, an NMDA antagonist, and nicotine, an nACh agonist.
Methods: In 40 healthy controls, all rated for AH with the Bell Object Relations and Reality Testing Inventory, MMN to frequency deviants was assessed in a randomized, placebo-controlled crossover design involving the separate and combined administration of a intravenous sub-psychotomimetic dose of ketamine (0.04 mg/kg) and a dose of nicotine gum (4 mg).
Results: In high AH subjects, ketamine reduced MMN, with the resulting amplitude being smaller than that of low AH subjects. This ketamine-induced MMN reduction was evident only with placebo gum; furthermore combined nicotine-ketamine treatment acted to increase MMN in high scorers.
Conclusions: AH in otherwise healthy individuals is associated with heightened sensitivity to NMDA receptor blockade, the effects of which are moderated by nicotinic neurotransmission. Both neurotransmitters may interact to moderate auditory processing and AH in SZ.

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