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Abstract

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The effects of regular long-term cannabis use on auditory mismatch negativity (MMN)

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BACKGROUND: Attenuated auditory mismatch negativity (MMN) is a robust finding in schizophrenia and has been proposed as an endophenotype for the disorder. Glutamatergic dysfunction at the genetic and protein level has been observed in schizophrenia and may underlie reduced MMN. Exogenous cannabinoids such as delta-9-tetrahydrocannabinol (THC) modulate CB1 regulation of NMDA receptors and disrupt normal glutamatergic functioning. Acute administration of cannabinoids and NMDA receptor antagonists has been shown to modulate MMN. Cannabis is a known risk factor for the development of schizophrenia and psychosis in vulnerable individuals. Chronic exposure has been shown to lead to similar structural and cognitive deficits observed in schizophrenia, suggesting a common underlying pathology. This study aimed to investigate the effects of regular long-term cannabis use on MMN as a vulnerability marker of schizophrenia and psychosis.

METHODS: Long-term heavy cannabis users (n=47) and healthy age and gender matched non-user controls (n=47) were compared on MMN response to duration, frequency and intensity deviants presented in a multi-feature paradigm. Tones were presented binaurally using headphones at a constant 500ms stimulus onset asynchrony with a 5ms rise/fall time. MMN waveforms for each condition were extracted from difference waveforms between the standard (50ms, 80dB, 1000Hz, 82%) and deviant (duration, 100ms, 6%; frequency, 1200Hz, 6%; intensity, 90dB, 6%) event related potentials. EEG was recorded with an initial nose reference and extracted waveforms were re-referenced to the average of the mastoids. Cannabis users had a mean frequency use of 24.37 days/month and a mean quantity use of 406.38 cones/month. Cannabis users remained abstinent for a minimum of 12 hours prior to testing. Cannabis use parameters and symptomatic measures were examined in relation to MMN peak amplitude and latency at the Fz electrode.

RESULTS: Group differences showed reduced MMN peak amplitude in the cannabis group relative to controls for duration ($F(1,78) = 35.55, p < .001$) and frequency ($F(1,80) = 9.38, p = .003$) conditions. Peak amplitude for intensity MMN did not differ between groups. No group differences were observed for MMN peak latency (all F values < 1). Exploratory analysis of symptomatic measures showed that cannabis users scored significantly higher than controls on Community Assessment of Psychiatric Symptoms (CAPE) total frequency scores ($p = 0.01$) and CAPE negative dimension frequency scores ($p = 0.034$). Cannabis users also scored higher on Schizotypal Personality Questionnaire (SPQ) total scores ($p = 0.009$) and on several subscale scores. None of the symptomatic measures nor any cannabis use parameters correlated with MMN peak amplitude in the duration or frequency condition.

DISCUSSION: The current findings provide support for chronic cannabis use as an

increased risk factor in the development of depressive and negative symptoms. The current data also provide some evidence of a glutamatergic dysfunction in regular long-term cannabis users, indicated by reduced MMN amplitude to frequency and duration deviants. Attenuated MMN to changes in duration and frequency of auditory stimuli is a robust finding in schizophrenia. The current findings of reduced MMN amplitude to duration and frequency deviants in cannabis users suggest that chronic cannabis use may affect early sensory information processing in a similar way to that observed in schizophrenia. This could potentially be explained by glutamatergic dysfunction as a result of prolonged exposure to cannabis and inform mechanisms by which cannabis could trigger psychosis.