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The Relationship between *Cannabis* and Schizophrenia

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Abstract

Cannabis is one of the most abused substances in the world, and it is getting legalized around the world. Consequently, it is crucial to understand its effect on mental health. Its impact on the schizophrenia spectrum needs special attention. Even though *cannabis* has been around for a long time, its exact effects on schizophrenia. Schizophrenia is a chronic illness affecting approximately 20 million people worldwide. In this review the relationship between cannabis and schizophrenia will be reviewed.

Keywords: Cannabis; Schizophrenia

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Introduction

In a Danish study, an increase in the proportion of cases of schizophrenia associated with *cannabis* use disorder increased from a 2% level in 1990 to a level of 8% in 2010 [1]. Supporting this finding there has been a doubling of schizophrenia and other psychotic disorders in the United States where marijuana has been legalized in many states [2].

The biochemical mechanism by which cannabis exerts its effects on physiology and behavior are associated with Delta-9-tetrahydrocannabinol (THC). THC is the main psychoactive constituent of *cannabis* [3]. The isolation of THC indicated the presence of an endogenous receptor in the brain to which cannabinoids exert their effects. *Cannabis* exposure may be a component which interacts with other factors that contributes to schizophrenia spectrum disorders but is neither necessary nor sufficient to do so alone [4]. In the absence of the known causes of schizophrenia, the role of component causes remains important.

The endocannabinoid system is comprised of the CB1 and CB2 (and possibly other CB1 receptors) which are expressed in both the central nervous system and periphery. THC induces long-term memory deficits and is mediated by CB1 expressed on gamma-amino butyric acid GABA neurons [5]. A growing body of literature has demonstrated that this system plays a highly specialized and functionally distinct role during development that extends beyond the regulation of neurotransmitter release.

The main possibility is the effect on the neurodevelopmental process, such as synaptic plasticity, which is likely impaired in schizophrenia. Despite considerable variation in how cannabis exposure and psychosis are elicited, there is a notable consistency in the findings of different studies [6]. These studies suggest that cannabis is a risk factor which increases the chances of developing schizophrenia spectrum disorders by approximately three-fold [7].

It is reported that approximately 4% of women in the United States abuse substances, with marijuana being by far the most common substance used during pregnancy (75%) [8]. Pre-natal *cannabis* exposure has an impact on the maturation of neurotransmitter systems, which play key roles in mood, motivation, and reward [9]. The endocannabinoid system also plays a crucial role in the ontogeny of the central nervous system and its activation during brain development which can induce subtle and long-lasting neuro functional alterations in offspring, despite being previously considered as relatively harmless [10]. Epidemiological and longitudinal studies have shown that newborns and infants born to cannabis users have neurological signs associated with increased tremors, exaggerated startle responses, and poor habituation to novel stimuli [11].

Adolescence is a critical phase for brain development, characterized by neuronal maturation and rearrangement processes, such as myelination, synaptic pruning, and dendritic plasticity. Several studies have investigated whether exposure to cannabis during adolescence is a risk factor for schizophrenia. Harkany et al., reported that those subjects who used *cannabis* on a daily basis between the ages of 15 to 18 had significantly more schizophrenic symptoms than control subjects (never used cannabis or had used cannabis 'once or twice') at age 26 [12]. Subjects in the earlier age group had a greater risk of schizophrenia at a later age [13].

Cumulative exposure to environmental stressors increases the risk for schizophrenia in an additive fashion. The main stress factors studied are childhood sexual abuse and urbanicity [14]. Consistent results are also seen for an increased risk for schizophrenia with urban birth and/or upbringing, especially among males. The mechanism of association is unclear but may relate to biological or social/environmental factors or both, acting considerably before psychotic symptoms manifest.

Disclosure

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