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# THE INFLUENCE OF CANNABIS ON THE DEVELOPMENT OF SCHIZOPHRENIA - A REVIEW

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**ABSTRACT**

**Background:** Schizophrenia is a complex mental disorder influenced by both genetic and environmental factors. Cannabis use, particularly products with high tetrahydrocannabinol (THC) content, has been identified as a potential environmental risk factor for psychosis.
**Aim:** This review aims to summarize current knowledge on the epidemiology, etiopathogenesis, and risk factors of schizophrenia, with a particular focus on the impact of marijuana use on the development of psychotic symptoms.
**Methods:** A narrative synthesis of epidemiological, genetic, neurobiological, and clinical studies was conducted, including analyses of cannabis use, THC potency, and age of first exposure.
**Results:** Evidence indicates strong familial aggregation of schizophrenia and sex-related differences in the age of onset. Genetic studies highlight polygenic susceptibility, dopaminergic and glutamatergic dysregulation, and the impact of prenatal and early-life exposures. High-THC cannabis use, especially during adolescence, is associated with increased risk of psychotic symptoms, earlier onset, and more severe disease progression. Rising THC concentrations and increasing cannabis consumption pose significant public health challenges.
**Conclusions:** Understanding the dose-dependent effects of cannabis on schizophrenia is critical for prevention strategies and public health policy, particularly in the context of increasing legalization and product potency.

**KEYWORDS**

Schizophrenia, Cannabis, THC, Psychosis, Genetic Predisposition, Environmental Risk Factors, Adolescence, Dose-Dependent Effect

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## 1. Introduction

Schizophrenia is a heterogeneous disease characterized by 3 main concepts: 1) the Kraepelinian emphasis on avolition, chronicity, and poor outcomes; 2) the Bleulerian view, in which dissociative pathology is fundamental and primary, and emphasizes negative symptoms; and 3) the Schneiderian view of reality distortion or positive symptoms [1]. Currently, the diagnosis of schizophrenia is based on a specific combination of symptoms, duration, and functional criteria. The disease is a brain disorder in which positive symptoms (delusions, hallucinations), negative symptoms (amotivation, flat affect), and/or disorganization (behavioral, cognitive) are interconnected [2]. Clinical criteria for schizophrenia are included in 2 approaches to the schizophrenia classification systems in DSM-5 and ICD-11, which differ in the minimum duration of symptoms (1 month in ICD-11 and 6 months suggested in DSM-5) [3]. The basic criterion must be met, which is persistent hallucinations of any type, persistent delusion of any type, distortion of self-awareness and thought disorder. Schizophrenia is defined by 6 symptoms: positive, negative, manic, depressive, cognitive and psychomotor deficits [4].

The aim of this study will be to review the current knowledge on the epidemiology, etiopathogenesis and risk factors of schizophrenia, with particular emphasis on the effect of marijuana on its development.

## 2. Characteristics of Schizophrenia

### 2.1. Epidemiology

Schizophrenia is the most common mental illness in the United States, occurring in about 1% of the population and largely diagnosed among black people [5]. Schizophrenia is inherited in families, and the risk of illness in first-degree relatives is 5.6% in parents with schizophrenia, 12.8% in children of one parent, and 46.8% in children of both parents with schizophrenia. In dizygotic twins and siblings, the risk is about 15%, and in monozygotic twins over 50%, regardless of the way they were raised, together or apart [6]. Almost 37% of women with schizophrenia develop symptoms by the age of 45, the so-called "second peak" in connection with the onset of menopause [7]. About 4% of diagnosed schizophrenia occurs before the age of 15, and about 1% before the age of 10. About two-thirds of all patients with schizophrenia are between 20 and 40 years old [8].

Symptoms in men usually appear in their late teens and early 20s, while in women they do not appear until their second or third decade of life [9].

### 2.2. Etiology

A combination of genetic predisposition and environmental factors is most likely responsible for the development of schizophrenia [5].

The genetic basis is underscored by the fact that the inheritance of schizophrenia for monozygotic oscillates at ~45% [10]. Although no single gene or variant responsible for the development of schizophrenia has been discovered to date, it is believed that the genetic aspect of schizophrenia depends most likely on polygenic conditioning [10, 11].

There is also evidence highlighting the importance of neurochemical abnormalities related to dopamine function and glutamatergic N-methyl-D-aspartate receptor function [11]. This is supported by the fact that in one of the most recent genome-wide association studies 270 loci linked to the development of schizophrenia have been identified, among them the dopamine and glutamatergic transmitters and ion channel function already mentioned. The role of the immune response has also been marked [10].

As already mentioned, environmental factors are also responsible for the development of schizophrenia. Of these, the main role is attributed to the mother's prenatal and perinatal stress exposure, her exposure to infectious agents such as Toxoplasma gondii, cytomegalovirus, and others and the associated immune response which contains the role of inflammation markers- cytokines and C-reactive protein, the pregnant woman's contact with stimulants and toxic substances and nutritional deficiencies [10, 12].

Environmental risk factors also include the potential role of emergency cesarean section, perinatal complications such as diabetes, bleeding, eclampsia, or exposure to Toxoplasma gondii, Cytomegalovirus and others. In addition, it appears that winter births are associated with a higher risk of developing schizophrenia [10].

Despite the notable trend as to the influence of the environment on the development of schizophrenia in prenatal life and early childhood, it has been shown that there are also environmental exposures that influence the development of the disease occurring later in life. Such factors include cannabis use [10].

## 3. Cannabis

Cannabis is a genus of flowering plants that, in the form of dried flower buds, are called marijuana. Cannabinoids are bioactive substances contained in cannabis, the psychoactive effect of which depends on binding to CB1 and CB2 receptors coupled to G proteins throughout the body. Of the nearly 100 types of cannabinoids, the most well-known are delta-9-tetrahydrocannabinol (THC) and cannabidiol (CBD) [13]. In 2021, according to the European Monitoring Centre, marijuana was used by 15.4% of Europeans aged 15-34 at least once in the past year. The psychotropic effects of cannabis are mainly caused by THC by acting on the CB1 receptor, the largest amount of which is located in the central nervous system and is one of the components of the endocannabinoid system [14]. The effect of marijuana after inhalation occurs within a few minutes and reaches its peak after about 30 minutes. Long-term, intensive use of cannabis that begins in adolescence can lead to psychotic disorders due to its negative impact on brain development. This also includes the use of products containing high levels of THC [15].

Schizophrenia is a psychosis that is associated with a higher risk of illness in people who smoke marijuana compared to the general population. Abusing cannabis increases the risk of developing psychotic disorders, causes longer hospitalizations and more severe symptoms [16]. Young men are more likely to develop schizophrenia than women aged 16-20. This may be due to the fact that men of the same age are more susceptible to the psychogenic effects of marijuana compared to women [17].

### 4. Schizophrenia

Studies do not unequivocally determine that marijuana smoking directly contributes to the development of schizophrenia. However, they do indicate that it constitutes one of the potential factors capable of triggering psychotic symptoms.

Although it may appear that individuals who use marijuana intensively are responsible for the development of schizophrenia, research shows that only a small proportion of them actually develop the disorder. This is related to the fact that the low incidence of psychosis depends not on a single factor but on several, particularly the degree of exposure to cannabis, environmental risks, and genetic predisposition. Thus, most marijuana users do not suffer from schizophrenia, and not all patients with schizophrenia are cannabis users [18].

The frequency of marijuana use among psychotic patients is higher mainly among young adults who begin using high-THC cannabis during adolescence. Moreover, meta-analyses have not demonstrated an association between cannabis abuse or dependence and the transition to psychosis. Instead, they have shown that individuals under the age of 16 who use high-potency cannabis strains (>10% THC) are more likely to develop dependence [19].

Other studies, however, have shown that in people who use marijuana heavily and regularly, the likelihood of developing psychosis is four times higher than in the general population. They also confirmed that psychotic symptoms tend to emerge at a younger age in cannabis users compared with non-users of the same age group. Importantly, in patients already diagnosed with schizophrenia, continued marijuana use as opposed to cessation and abstinence leads to intensification of both positive and negative symptoms, a deterioration in quality of life, more frequent hospitalizations, and higher relapse rates [20].

A proper understanding of schizophrenia development must also take into account genetic predisposition and the complex interplay of environmental factors among various cannabis consumers. The effects of cannabinoids depend on dopaminergic signaling pathways and may increase the long-term risk of psychosis in individuals with metabolic dysfunctions [21]. Additionally, studies analyzing polygenic risk for schizophrenia alongside environmental exposures have demonstrated an additive interaction between genetic risk, lifetime cannabis use, and adverse childhood experiences (such as emotional neglect or emotional abuse) [22]. It has been observed that marijuana use and psychotic experiences are more strongly associated among individuals with a high genetic risk for schizophrenia. There is substantial evidence supporting this, and the risk is greater in predisposed individuals, particularly those exposed during adolescence or the prenatal period both critical phases of neural development [23].

### 5. Dose

In pharmacotherapy, it has long been understood that the dose makes the poison. This fundamental principle of toxicology is well established and not a recent discovery. In this section, we examine how THC concentrations in marijuana have changed over the years and how increasing potency affects the mental health of the population.

As of 1 February 2025, a total of 39 U.S. states had legalized medical marijuana, and 24 states had approved its recreational use. This evolving regulatory environment has contributed to the development of legal cannabis cultivation featuring increasingly high THC levels in cannabis flowers. In the past, THC concentrations typically ranged from 2% to 4%, whereas today the average level is approximately 20%. As much as 58% of all analyzed associations evaluating the occurrence of psychosis or schizophrenia indicated that higher THC concentrations, compared with lower levels or none, were linked to unfavorable outcomes. These findings were also consistent across both healthy participants (84%) and individuals with preexisting psychosis (89%) [24].

Results from the meta-analysis conducted by Marconi and colleagues demonstrated a steady increase in the risk of psychosis with higher doses of marijuana. The risk of schizophrenia is significantly higher among users who consume the most potent THC concentrations compared with those who use cannabis at any level [25].

## 6. Summary

Schizophrenia is a disorder with a complex pathogenesis involving the interplay of genetic and environmental factors. Although no single gene has been identified as responsible for its development, numerous studies indicate polygenic conditioning and the involvement of dopaminergic and glutamatergic system dysfunctions. Prenatal and perinatal factors such as infections, stress, malnutrition, or birth complications also play a significant role.

Cannabis use, particularly of high-THC products, represents an additional environmental risk factor that may increase the likelihood of psychosis, especially among genetically predisposed individuals. Research confirms that intensive, regular cannabis use contributes to earlier onset of psychotic symptoms, more severe disease progression, and more frequent relapses in already diagnosed patients. The increase in cannabis potency over the years from 2-4% THC to an average of approximately 20% is associated with a clear rise in the risk of adverse mental health outcomes. Meta-analyses further show a dose-dependent relationship: the higher the exposure to THC, the greater the probability of developing psychosis or schizophrenia.

Given the ongoing legalization of cannabis and the growing availability of high-potency products, this issue represents a significant concern for public health.

**Disclosure**

The authors declare no conflict of interest.
Conceptualization: [JM], [AH], [WM], [JN]
Methodology: [SM], [JS], [OS]
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Check: [JM], [WM]
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Resources: [AH], [ML]
Data curation: [JM], [MK]
Writing – original draft preparation: [JM], [WM]
Writing – review and editing: [AH], [ML], [SM]
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All authors have read and agreed with the published version of the manuscript.

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