

RESEARCH ARTICLE

DESDE UNA PERSPECTIVA ACTUAL: METANFETAMINAS Y CANNABIS, UNA COMPARATIVA EN LA SALUD MENTAL SOBRE EL INICIO DE LA ESQUIZOFRENIA COMO COMPLICACIÓN DEL CONSUMO CRÓNICO. REVISIÓN NARRATIVA

FROM A CURRENT PERSPECTIVE: METHAMPHETAMINES AND CANNABIS, A COMPARISON IN MENTAL HEALTH ON THE ONSET OF SCHIZOPHRENIA AS A COMPLICATION OF CHRONIC USE. NARRATIVE REVIEW

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RESUMEN

Objetivo: Realizar una revisión sobre el inicio de la esquizofrenia como complicación psiquiátrica al consumo crónico de metanfetaminas y cannabis. **Material y métodos:** Se realizaron búsquedas en las bases de datos PubMed, EBSCO y GOOGLE ACADEMICA, utilizando los términos: sobre el consumo de cannabis y metanfetaminas, su relación con el inicio de la esquizofrenia, identificando sus efectos en el entorno social, familiar, psicológico y personal, como complicación. Se consideraron artículos realizados en los últimos 20 años publicados en inglés y español. Se encontraron 4383 artículos y tras depurarlos quedaron 16. El instrumento más usado fueron los criterios del DSM V. **Resultados:** El consumo crónico de cannabis se presenta en edades tempranas entre los adolescentes, el cual acelera la enfermedad psiquiátrica y por lo tanto la esquizofrenia, si bien también ya es conocido este hecho con el consumo prolongado de metanfetaminas, este se presenta en consumidores de mayor edad. En conjunto son varios los factores que predisponen la manifestación clínica de la esquizofrenia como la vulnerabilidad del consumidor, defectos genéticos, sociales y riesgos asociados que se hacen presentes al momento de conjugarlos.

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Conclusiones: El consumo crónico de cannabis a diferencia de las metanfetaminas a edades tempranas en una línea de tiempo se manifestará en algunas veces clínicamente como precursor de muchas enfermedades, trastornos mentales y la misma esquizofrenia. Por lo tanto, el consumo del cannabis en consumidores adolescentes va a perpetuar también como un desencadenante de la esquizofrenia sobre todo en las personas con vulnerabilidad genética, desarrollando una modificación muy marcada entre los individuos que padecen esquizofrenia que han sido consumidores de cannabis, y los individuos con esta enfermedad, pero sin antecedentes de uso y abuso de esta droga.

PALABRAS CLAVE. Esquizofrenia. Cannabis. Metanfetaminas. Consumo crónico.

ABSTRACT

Objective: To review the onset of schizophrenia as a psychiatric complication of chronic methamphetamine and cannabis use. **Material and methods:** PubMed, EBSCO and GOOGLE ACADEMICA databases were searched using the terms: cannabis and methamphetamine use, its relationship with the onset of schizophrenia, identifying its effects on the social, family, psychological and personal environment, as a complication. Articles published in the last 20 years in English and Spanish were considered. A total of 4383 articles were found and after filtering, 16 remained. The most commonly used instrument was the DSM V criteria. **Results:** Chronic cannabis use occurs at an early age among adolescents, which accelerates psychiatric disease and therefore schizophrenia, although this fact is also known with prolonged methamphetamine use, this occurs in older users. As a whole, there are several factors that predispose the clinical manifestation of schizophrenia such as the vulnerability of the consumer, genetic defects, social and associated risks that are present at the time of combining them. **Conclusions:** Chronic cannabis use unlike methamphetamines at early ages on a timeline will sometimes manifest clinically as a precursor to many illnesses, mental disorders and schizophrenia itself. Therefore, cannabis use in adolescent users will also perpetuate as a trigger for schizophrenia especially in people with genetic vulnerability, developing a very marked modification between individuals with schizophrenia who have been cannabis users, and individuals with this disease, but without a history of use and abuse of this drug.

KEY WORDS. Schizophrenia. Cannabis. Methamphetamines. Chronic use.

INTRODUCCIÓN

Schizophrenia is a severe mental disorder affecting approximately 1 in

300 individuals worldwide, equivalent to an estimated 24 million people. It is characterized by significant disturbances in the perception of

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reality, alongside alterations in thought processes, emotions, language, perception, and behavior (World Health Organization [WHO], 2022).

In alignment with this analysis, stimulant use disorders involving methamphetamine and cannabis are defined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5, 2020) by a pattern of consumption of amphetamines, cannabis, cocaine, or other stimulants that leads to significant distress or clinically meaningful impairment. This disorder is characterized by the presence of at least two of the following criteria within a 12-month period: consumption of the stimulant in larger amounts or over a longer duration than intended; persistent desire or unsuccessful efforts to reduce or control stimulant use; considerable time spent obtaining, using, or recovering from the effects of the stimulant; cravings or a strong urge to use the stimulant; recurrent use resulting in failure to fulfill major role obligations at work, school, or home; continued use despite persistent or

recurrent social or interpersonal problems caused or exacerbated by stimulant use; important social, occupational, or recreational activities are given up or reduced due to stimulant use; recurrent use in physically hazardous situations; continued use despite awareness of persistent or recurrent physical or psychological problems likely caused or worsened by the stimulant; and increased tolerance to the stimulant's effects.

Within the context of these two substances use disorders, chronic methamphetamine and cannabis consumption has been identified as a risk factor for the development of psychosis. This relationship is described by Kaplan (1996), who referenced five models proposed by Dixon (1995), outlined as follows:

1. Etiological model, which associates psychotic disorders with the excessive use and abuse of substances.
2. Dopaminergic dysfunction has been previously implicated as a common

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biological vulnerability underlying both substance use and abuse disorders.

3. A promotive association that facilitates socialization of the user with individuals experiencing psychotic disorders.

4. self-medication.

5. Independent disorders model, which posits that the two conditions vary independently due to their high prevalence in the general population (Lorenzo P. et al., 2003).

THEORETICAL FRAMEWORK

The DSM-5 (2020) defines substance-induced psychosis as psychotic symptoms that precede the onset of substance use or persist for less than one month following cessation of use or acute intoxication. A diagnosis of a primary psychotic disorder (e.g., schizophrenia) is made when the symptoms are sufficiently severe to meet established diagnostic criteria.

According to the World Health Organization (WHO, 2022), the global incidence of schizophrenia has remained relatively stable over recent years, with a higher prevalence observed in men compared to women, affecting approximately 12 million and 9 million individuals, respectively.

Regarding the age of onset of clinical manifestations of this disorder, approximately 75% of diagnosed individuals develop symptoms between the ages of 16 and 25 (Khokhar et al., 2018).

Epidemiological data indicate that over 25% of individuals with schizophrenia meet criteria for cannabis dependence, while approximately 10–35% are dependent on methamphetamine (Courtney et al., 2014).

Substance use represents a significant public health concern across all levels, as it may contribute to an increased incidence of psychotic disorders among users of methamphetamine and cannabis, particularly in individuals diagnosed with

schizophrenia. Although the association between substance use and psychosis is well-documented, it remains unclear which of the two substances methamphetamine or cannabis is more strongly associated with the initial onset of the disorder. This uncertainty is further compounded by the tendency to downplay the severity of cannabis-related effects, especially among adolescents, and by the lack of precise epidemiological data on patterns of drug use (World Health Organization [WHO], 2003).

On the other hand, in patients diagnosed with schizophrenia, symptom severity may be exacerbated by substance addiction. This has led to increased interest in research exploring the relationship between chronic methamphetamine and cannabis use and the variability in the onset and progression of schizophrenia symptoms (Arciniegas, 2015).

The present study aimed to conduct a narrative literature review covering the

past 20 years. This bibliographic review was based on scientific articles published in peer-reviewed, indexed journals in both Spanish and English, employing a descriptive research design. The objective of this monograph was to examine the long-term use of cannabis and methamphetamine and its relationship with the clinical onset of schizophrenia, while also identifying the effects on individuals' social, familial, psychological, and personal domains, as well as the contributing factors to the disorder's emergence. Databases and search engines such as PubMed, EBSCO, and Google Scholar were utilized. No conflicts of interest were declared.

A total of 4,383 records were initially identified across the aforementioned databases. After the removal of 535 duplicates and the exclusion of 3,834 records that were not directly related to the specific topic of schizophrenia in the context of methamphetamine and cannabis use, 16 articles remained. During the initial screening process, 27 additional articles were excluded due

to being published in languages other than Spanish or English. Ultimately, 16 articles were included for analysis: 8 focused specifically on cannabis use and its association with schizophrenia, and 8 addressed the long-term and pathological use of methamphetamine in relation to schizophrenia.

CANNABIS AND SCHIZOPHRENIA ASSOCIATION

Cannabis-related schizophrenia and its initial clinical manifestations often resemble an acute paranoid schizophrenic pattern, as described by Díaz et al. (2009). These manifestations are typically associated with periods of heavy cannabis use, or may emerge within a month following intoxication or during withdrawal. This presentation differs from primary psychotic disorders, as it can be distinguished through an analysis of the semiology, onset, progression, and contextual factors of the mental illness. Accordingly, when such symptomatology is observed in cannabis users, diagnoses such as cannabis-induced psychosis or

cannabis hallucinosis may be considered. These conditions are commonly characterized by delusional ideation, hallucinations, heightened anxiety, affective lability, depersonalization, derealization, and episodes of amnesia. In individuals with a predisposition, these symptoms can progress into a persistent psychotic reaction, although in most cases, the episode resolves rapidly, often within a day or several days (Tziraki et al., 2012). El uso y abuso de cannabis es muy común entre los pacientes con trastornos psicóticos. Más del 25% de las personas con esquizofrenia tienen una dependencia de cannabis concomitante. (Rentero et al. 2021)

Moreover, according to Rentero (2021), pathological cannabis use may be considered a significant risk factor for the development of schizophrenia, particularly among individuals who initiate intensive and disproportionate consumption at an early age.

The aforementioned risk is predicted to be higher for cannabis than for other

substances, as cannabis use has been associated with an earlier onset of clinical manifestations of psychosis, particularly among adolescents. Rentero (2021) highlights emerging evidence indicating that a significant proportion of cannabis users who experience psychotic episodes estimated at approximately 50% may go on to develop chronic psychotic disorders (Rentero et al., 2021; Pierre et al., 2016). Furthermore, the potential of cannabis to contribute to mental impairment has been substantiated by findings linking higher-potency cannabis measured by elevated levels of tetrahydrocannabinol (THC) to an increased risk of psychosis (Rentero et al., 2021; Starzer et al., 2018).

Prolonged cannabis use has been scientifically shown to induce a neurobiological process known as neuroadaptation, which is directly associated with the development of cannabis tolerance and dependence (Rentero et al., 2021; Starzer et al., 2018). Specifically, the overstimulation of cannabinoid receptors (CB

receptors) by Δ^9 -tetrahydrocannabinol (THC) leads to alterations in the cellular activity of the molecular systems in which these receptors operate. As a result, new patterns of receptor functioning are established, thereby modifying neural pathways involved in the development of substance dependence (Rentero et al., 2021; Starzer et al., 2018).

On the other hand, with respect to the timeline of clinical manifestations of schizophrenia associated with cannabis use and abuse, Gutiérrez B. et al. (2009), in their study "Variabilidad en el gen COMT y modificación del riesgo de esquizofrenia conferido por el consumo de cannabis", report that prolonged cannabis use may represent a significant risk factor for the development of psychosis, particularly schizophrenia. Moreover, this risk appears to be modulated by genetic predisposition, suggesting that individual genetic variability such as polymorphisms in the COMT gene may influence the extent to which

cannabis use contributes to the onset of this psychiatric disorder.

A longitudinal cohort study conducted in Dunedin, New Zealand, followed individuals from birth to 26 years of age and identified that those homozygous for the Val allele (Val158) of the catechol-O-methyltransferase (COMT) gene polymorphism exhibited a significantly increased risk associated with adolescent cannabis use. Specifically, these individuals demonstrated at least a fivefold higher likelihood of developing psychotic symptoms and schizoaffective disorders compared to genotype-matched peers who had not used cannabis during adolescence (Gutiérrez et al., 2009).

Similarly, it has been demonstrated that cannabis use and chronic abuse before the age of 18 are associated with a significantly higher risk of developing schizophrenia compared to individuals who begin use later in life or who do not use cannabis. Moreover, daily cannabis consumption has been shown to increase the risk of

developing psychosis by a factor of nine relative to non-users or non-abusive users (López L. et al., 2021).

Several studies have also documented that daily cannabis use and abuse, particularly when initiated during adolescence, are associated with an earlier age of onset of psychosis. Specifically, patients who consume cannabis tend to experience the onset of psychotic symptoms at a younger age compared to non-users.

According to López L. et al. (2021), up to 51% of schizophrenia cases could potentially be prevented by reducing or eliminating exposure to daily cannabis use, assuming a causal relationship.

Finally, further research is needed to elucidate the association and variability of clinical manifestations of schizophrenia related to cannabis use (Montes S. et al., 2022). Notably, the average frequency of cannabis consumption has been significantly correlated with central nervous system effects, whereas the majority of consumption patterns were observed exclusively in the subgroup of

individuals diagnosed with schizophrenia without comorbid substance use disorder (Rojas et al., 2022). Furthermore, Horcajadas F. et al. (2002) describe a strong relationship between cortical processes and the mediation of prolonged cannabis use in the development of schizophrenia. This evidence underscores cannabis as a potent factor contributing to psychosis development by altering neural circuits in genetically vulnerable adolescents and young adults (Horcajadas, 2023) (see Table 1).

Table 1. Cannabis, Results

AUTHOR	CHARACTERISTICS OF THE STUDY AND TOTAL POPULATION EVALUATED.	RESULTS (% OF USERS WITH SCHIZOPHRENIA)
<i>Rentero D.et al. (2021)</i>	The study utilized the Psychiatric Research Interview for Substance and Mental Disorders (PRISM-IV) scale to differentiate substance-induced psychotic episodes. The total sample comprised 331 hospitalized patients, stratified into three groups, including a cohort diagnosed with cannabis-induced psychosis (CIP).	The primary finding of this study was the demonstrated precipitating role of cannabis in the onset of psychotic disorders, evidenced by a lower age of hospital admission among cannabis users. Clinical differences between patients diagnosed with schizophrenia, irrespective of cannabis use, were minimal. Notably, both groups exhibited a similarly reduced age at first hospital admission for psychosis. Furthermore, no distinct clinical pattern characteristic of cannabis-induced psychosis was identified.
<i>Gutiérrez B. et al. (2009)</i>	The study was conducted to investigate the gene-environment interaction between the COMT polymorphism and cannabis use, and its effect on the risk of developing schizophrenia. The total sample comprised 283 individuals, including 91 participants (66 males and 25 females) in one group, and 192 participants (96 males and 96 females) in the other.	The most notable finding of this study was that the Val158Met polymorphism of the COMT gene showed no significant association with schizophrenia overall. However, individuals homozygous for the Val allele (Val/Val) who consumed cannabis exhibited a significantly higher predisposition within the patient group compared to the control group. Additionally, a non-significant increase was observed among female cannabis users carrying the Val allele in the patient group relative to controls. Ultimately, the results suggest that this genetic variation may be relevant predominantly in the female subgroup.
<i>López L. al. (2021)</i>	The study was conducted as a retrospective observational cohort investigation with case-control design at the Consultation Service of the Hospital of Costa Rica, comprising a total sample of 80 cases and 160 controls.	The results indicate that male and adolescent cannabis users exhibit a stronger association with schizophrenia, presenting a ninefold increased risk compared to non-users.

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<i>Montes S. et al. (2022)</i>	The study was conducted as a descriptive investigation examining the role of cannabis in schizophrenia by assessing schizotypal traits, patterns of substance abuse, and their influence on experiential avoidance. The sample consisted of 270 Spanish participants.	Weak correlations were observed between problematic substance use and substance dependence with schizotypal personality traits, alongside moderate associations between the presence of schizotypal traits and experiential avoidance. In conclusion, these findings underscore the need for further research to clarify these relationships and inform the development of appropriate interventions.
<i>Rojas B. et al. (2022)</i>	The study employed a cross-sectional quantitative design to assess differences in parameters related to duration (NDMM) and frequency (NFMM) of use. The sample comprised a total of 89 individuals, including patients diagnosed with schizophrenia who use cannabis, as well as healthy control participants.	The results indicate a risk association between cannabis use and the variability in progression (ripening condition) as well as the frequency of cannabis consumption. Specifically, individuals with schizophrenia who do not use substances exhibited lower levels of these parameters compared to cannabis-using individuals with schizophrenia. However, no significant differences were observed within the subgroup of schizophrenia patients who use cannabis.
<i>Rojas B. et al. (2022)</i>	The study employed Mismatch Negativity (MMN) assessment as its primary methodology. The total sample comprised 77 participants, including 23 active cannabis users diagnosed with schizophrenia (ESQ+CN), 23 schizophrenia patients without cannabis use (ESQ), and 21 cannabis users without a schizophrenia diagnosis (CN). MMN potentials were recorded utilizing a 32-channel electroencephalogram (EEG).	The results indicate that the amplitude of the MMN duration (MMNd) was reduced in both the ESQ+CN and ESQ groups compared to controls. Regarding MMN frequency (MMNf), the ESQ group exhibited lower amplitudes than both the ESQ+CN and CN groups. No significant differences were observed in latency across groups. These findings suggest that MMN frequency deficits are associated with schizophrenia, whereas MMN duration may serve as a potential biomarker for monitoring cannabis use.
<i>Horcajadas F. et al. (2023)</i>	An evaluation was conducted using the Addiction Severity Index (ASI) and the Positive and Negative Syndrome Scale (PANSS), with a six-month follow-up period. The sample comprised 82 outpatients diagnosed with schizophrenia.	The results indicate that cannabis and tobacco use may alleviate the negative symptoms of schizophrenia or mitigate the side effects of neuroleptic medications; alternatively, patients exhibiting fewer negative symptoms may be more inclined to use these substances. In contrast, alcohol consumption appears to exacerbate negative symptoms.
<i>Horcajadas, F. et al. (2002)</i>	The Positive and Negative Syndrome Scale (PANSS), developed by Kay, Fiszbein, and Opler (1987), was utilized in this study. The sample comprised a total of 379 individuals genotyped for polymorphisms related to the cannabinoid receptor system, including patients diagnosed with schizophrenia without substance use disorder (n = 379), schizophrenia with cannabis use disorder (n = 124), cannabis dependence without associated psychosis (n = 71), and a control group (n = 316). Participants were recruited from various hospitals and healthcare centers across Spain.	The results indicate a significant association between polymorphisms in the CNR2 gene alleles rs35761398 and rs12744386 and schizophrenia comorbid with cannabis use and abuse disorder. Additionally, a loss of heterozygosity in the rs324420 polymorphism of the FAAH gene was linked to cannabis dependence within the Spanish population. These findings suggest an elevated risk of cannabis use and abuse among younger individuals, although specific age ranges were not delineated.

Source: Own elaboration

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METHAMPHETAMINE AND SCHIZOPHRENIA ASSOCIATION

Methamphetamine is a highly prevalent substance of abuse, ranking as the second most commonly preferred illicit drug globally (Pierre et al., 2016). Specifically, in the northern region of Mexico, crystal methamphetamine represents a significant public health challenge, accounting for the primary drug of concern with a prevalence of 49.2%, compared to alcohol, which constitutes only 15.4% of substance use in the population (De La Rosa Garza et al., 2022).

De la Rosa Garza et al. (2022) described the neurological effects of methamphetamine use as a cascade of neurochemical events involving alpha-2 adrenergic receptor agonism and activation of sigma receptors—showing a higher affinity for σ_1 than σ_2 —as well as inhibition of monoamine oxidase A (MAO-A) and monoamine oxidase B (MAO-B). The effective activation of sigma receptors by methamphetamine facilitates its

potent stimulant effects on the central nervous system and contributes to its neurotoxic impact on neural and cerebral structures. In summary, methamphetamine use leads to increased synaptic concentrations of dopamine, noradrenaline, serotonin, and beta-endorphins (Table 2).

As a result, Soto C. et al. (2023) identified a relationship between long-term methamphetamine use and schizophrenia, highlighting specific risk factors. The study determined that being male, approximately 27 years old, and lacking a stable work life significantly increased the risk of developing schizophrenia. Furthermore, the authors established that factors such as age, sex, occupation, and treatment adherence were associated with methamphetamine use in 34% of the young patients included in their study, “Factors Associated with the Consumption of Psychoactive Substances in a Group of Patients Diagnosed with Schizophrenia at a Mental Health Institution in Medellín,

Antioquia.” The majority of participants were men diagnosed with schizophrenia linked to methamphetamine use, who exhibited a higher risk of relapse and lower adherence to pharmacological treatment.

In relation to this research, Soto C. et al. (2023) described the timeline of methamphetamine-related schizophrenia, reporting that the age of onset of methamphetamine use was negatively correlated with the total psychosis score and the Activation subscale score. Additionally, the duration of methamphetamine use showed a positive correlation with the duration of psychotic symptoms, consistent with findings reported by Hartel-Petri R. et al. (2005).

In comparison with the clinical manifestations associated with methamphetamine use, several studies have reported that the presence of non-persecutory delusions and multimodal hallucinations serves as an indicator of persistent methamphetamine-

associated psychosis (MAP) or primary psychotic disorders among methamphetamine users (McKetin et al., 2017).

Finally, multiple studies, as summarized in the attached table, indicate a significant association between early-onset methamphetamine use and increased severity of psychotic symptoms, potentially suggesting long-term effects on brain development. The observed correlation between the duration of methamphetamine use and the persistence of psychosis may point to a cumulative neurotoxic effect. Additionally, the presence of paranoia and comparatively milder negative symptoms among methamphetamine users may reflect relatively better-preserved social functioning in this population. These findings highlight the need for further mechanistic research to better understand the underlying pathways involved (Yang et al., 2020).

Lastly, key clinical features have been outlined along a temporal axis for the

diagnosis of methamphetamine-related schizophrenia. Notably, early onset of methamphetamine use has been associated with increased severity of psychotic symptoms, while

the duration of methamphetamine uses correlates with the overall duration of psychosis (Yang et al., 2020).

Tabla 2. Metanfetaminas, Resultados

AUTHOR	CHARACTERISTICS OF THE STUDY AND TOTAL OF THE POPULATION EVALUATED.	RESULTS
<i>De la Rosa G. et al. (2022)</i>	The study's methodology involved the administration of the Positive and Negative Syndrome Scale (PANSS) and the Brief Psychiatric Rating Scale (BPRS) to assess the clinical profiles of participants. Additionally, a structured questionnaire was employed to collect sociodemographic data. The total sample consisted of 61 patients diagnosed with schizophrenia or psychosis.	The results revealed significant differences between the groups diagnosed with psychotic disorders and schizophrenia. This distinction allowed the study to differentiate between patients who used and abused cannabis and those with no history of substance use. The findings highlight the increased risk associated with cannabis consumption among individuals with schizophrenia. Ultimately, the study emphasizes the need for further research to strengthen the evidence base and deepen understanding of this association.
<i>Soto C. et al. (2023)</i>	An observational, retrospective study with an analytical focus was conducted. The study identified sociodemographic factors, psychoactive substance (PAS) use, and clinical variables such as type of substances consumed, frequency of hospital readmissions, and adherence to pharmacological treatment. The total sample included 112 patients diagnosed with schizophrenia.	The findings indicate that male sex, a median age of 27 years, and unstable employment status are significantly associated with an increased risk of developing schizophrenia related to cannabis use.
<i>Härtel-Petri R. et al. (2005)</i>	An analysis of clinical records of hospitalized patients presenting with paranoid psychosis was conducted to examine its association with increased incidence of drug-induced psychosis. During the observation period from 1998 to 2000, diagnostic distributions and psychosocial data of all patients admitted to BKH Bayreuth were systematically reviewed.	The results indicate an increase in both the number of patients treated for chronic methamphetamine dependence and the number of acute cases presenting with drug-induced psychosis. Distinct differences were observed between patients hospitalized with induced psychosis and those diagnosed with schizophrenia. As stimulant abuse, particularly methamphetamine, continues to rise, the prevalence of drug-induced psychosis is expected to increase correspondingly. This trend underscores the necessity for specialized treatment settings that simultaneously address both addiction and psychotic symptoms.
<i>McKetin R. et al. (2017)</i>	The Brief Psychiatric Rating Scale was employed to assess suspicion, hallucinations, or unusual thought content within the past month. Lifetime psychotic diagnoses and symptomatology were evaluated using the International Composite Diagnostic Interview. The study included a total of 110	Transient methamphetamine-associated psychosis (MAP) was characterized by persecutory delusions and tactile hallucinations, compared to the asymptomatic group. Persistent MAP was additionally associated with referential delusions, thought interference, and complex auditory, visual, olfactory, and tactile hallucinations. In contrast, primary psychosis was further linked to symptoms such as

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	participants, who were categorized into four groups: (a) individuals with no current psychotic symptoms (n = 110); (b) individuals exhibiting psychotic symptoms exclusively during methamphetamine use (transient methamphetamine-associated psychosis [MAP], n = 85); (c) individuals presenting psychotic symptoms both during methamphetamine use and periods of abstinence (persistent MAP, n = 37); and (d) individuals meeting DSM-IV criteria for schizophrenia or lifelong mania (primary psychosis, n = 52).	erotomania and passivity phenomena. The presence of non-persecutory delusions and hallucinations across multiple sensory modalities serves as a distinguishing marker for persistent MAP or primary psychosis among methamphetamine users.
Yang M. et al. (2020)	The study utilized the Brief Psychiatric Rating Scale (BPRS) to assess symptom severity and examined the duration of psychosis in patients with methamphetamine-associated psychosis. Comparisons were made between groups based on BPRS scores. The sample consisted of 140 hospitalized patients, including 70 diagnosed with methamphetamine-associated psychosis and 70 with primary schizophrenic psychosis, matched according to sex, age, and duration of psychosis.	The results reveal an association between early onset of methamphetamine use and the severity of psychosis, which may suggest a lasting effect on brain development. The correlation between drug use and the duration of psychosis may suggest a cumulative effect of methamphetamine exposure. Paranoia and less severe negative symptoms in the methamphetamine user group could imply better social functioning in these patients. Further mechanistic studies are warranted. Key points: Early onset of methamphetamine use is associated with the severity of psychosis. Duration of methamphetamine use is associated with the duration of psychosis. Methamphetamine-associated psychosis and primary schizophrenic psychosis were similar in terms of symptoms. Patients with methamphetamine-induced psychosis exhibited less severe paranoia and negative symptoms.
Scheffler F. et al. (2022)	A longitudinal cohort study was conducted to examine the association between cannabis and methamphetamine use and cognitive performance in individuals with schizophrenia spectrum disorders during their first episode, over the initial two years of treatment. The study included a total of 81 patients who received treatment with flupentixol decanoate following a standardized protocol for 24 months.	Compared to the control group, patients exhibited greater cognitive impairment at baseline, which improved with treatment but remained significantly impaired throughout the treatment period. The number of positive methamphetamine tests, but not cannabis use, predicted reduced cognitive improvement in patients. These findings suggest a negative association between methamphetamine use and cognitive function, whereas no such association was observed for cannabis use.
Babina A. et al. (2023)	This study aims to examine the psychological characteristics of amphetamine-induced psychosis in drug-dependent patients, considering the duration of drug exposure, and to compare these characteristics with those of patients diagnosed with paranoid schizophrenia. The research was conducted in 2019 at the 1st Psychiatric Clinic in Kiev, Ukraine, with a total sample of 107 patients. Among the participants, 50 were assigned to Group 1 (methamphetamine-induced psychosis) and 57 to Group 2 (paranoid schizophrenia).	The results indicate that, in Group 1, the onset of paranoid symptoms is significantly correlated with the duration of amphetamine exposure (Spearman's correlation coefficient = 0.89). The efficacy and progression of pharmacological treatment in Group 2 were comparable to those observed in Group 1; however, symptom reduction in Group 2 was achieved within a shorter period of four months. Delusions, emotional disturbances, and hallucinations occurred 2.3 times more frequently in Group 1 compared to Group 2 ($p < 0.05$). Patients in Group 1 exhibited a higher prevalence of affective and behavioral disturbances. All exacerbations reported in Group 1 were temporally associated with amphetamine use. Furthermore, Group 1 patients demonstrated poorer verbal learning performance than those in Group

		2, along with a higher frequency of errors and greater difficulty in shifting cognitive focus.
<i>Khalili N. et al. (2022)</i>	This cross-sectional study involved administering the Clock Drawing Test (CDT), the Rey–Osterrieth Complex Figure Copy Test (ROCF), and the Intertwined Fingers Test (IFT) to three groups, alongside the collection of demographic and clinical data. One-way analysis of variance (ANOVA) was employed to assess differences between groups. Additionally, multivariate covariance analysis was conducted to control for potential confounding variables. Post hoc pairwise comparisons were performed following adjustment for multiple testing. The sample comprised 30 patients diagnosed with schizophrenia, 30 patients with methamphetamine-induced psychosis (MIP), and 32 healthy control participants.	The results indicate that the schizophrenia group exhibited significantly greater impairment compared to the methamphetamine-induced psychosis (MIP) group on the Intertwined Fingers Test (IFT) and the Rey–Osterrieth Complex Figure Copy Test (ROCF). However, no significant differences were observed between the MIP group and healthy controls on these two assessments. Regarding the Clock Drawing Test (CDT), the only significant difference was between the schizophrenia group and the control group. If these findings are replicated in further studies, certain neurocognitive tests assessing parietal lobe function may prove useful in the differential diagnosis of schizophrenia and MIP.

Source: Own elaboration

CONCLUSIONS

Comparing the effects of chronic methamphetamine and cannabis use on the onset and presentation of schizophrenia remains a complex task due to the absence of well-defined parameters. Attempting to establish a timeline of clinical manifestations associated with the onset of this mental disorder in relation to the consumption of both substances is inherently influenced by multiple factors, including individual vulnerability, genetic predispositions, social determinants, and concomitant

risk factors present at the time of exposure, as well as patterns of substance use.

Therefore, within well-established contexts, early-age cannabis consumption may be directly linked to the development of schizotypal personality disorder, with schizophrenia being the mental illness most commonly associated as a subsequent outcome.

In comparison, chronic methamphetamine use predominantly affects young adults and directly impairs the dopaminergic,

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serotonergic, and GABAergic systems. Within the pathophysiological framework, dysregulation of these neurotransmitter pathways—particularly the dopaminergic system—represents the most widely accepted theory, as it accounts for the emergence of both positive and negative symptoms observed in schizophrenia.

In conclusion, the early use and abuse of cannabis, unlike methamphetamine, may clinically manifest over time as a precursor to various mental disorders, including schizophrenia.

Adolescent cannabis use may act as an additional risk factor or predispose individuals to the development of adverse mental health outcomes such as schizophrenia, particularly among those with a genetic vulnerability. Notably, significant differences have been observed between individuals with schizophrenia who have a history of cannabis use and those with the

disorder who have never used the drug.

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