Psychological Medicine

cambridge.org/psm

Discussing the concept of substance-induced psychosis (SIP)

Jørgen G. Bramness[1,2,3,4], Carsten Hjorthøj[5,6], Solja Niemelä[7,8], Heidi Taipale[9,10,11] and Eline Borger Rognli[4]

[1]Institute for Clinical Medicine, UiT The Arctic University of Norway, Tromsø, Norway; [2]Norwegian Institute of Public Health, Oslo, Norway; [3]National Advisory Unit on Concurrent Substance Abuse and Mental Health Disorders, Innlandet Hospital Trust, Brumunddal, Norway; [4]Section for Clinical Addiction Research, Oslo University Hospital, Oslo, Norway; [5]Copenhagen Research Center for Mental Health – CORE, Mental Health Centre Copenhagen, Denmark; [6]University of Copenhagen, Department of Public Health, Section of Epidemiology, Copenhagen, Denmark; [7]Department of Psychiatry, University of Turku, Turku, Finland; [8]Department of Psychiatry, Addiction Psychiatry Unit, Turku University Hospital; [9]Niuvanniemi Hospital, Kuopio, Finland; [10]Department of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden and [11]Center for Psychiatry Research, Stockholm City Council, Stockholm, Sweden

Review Article

Cite this article: Bramness JG, Hjorthøj C, Niemelä S, Taipale H, Rognli EB (2024). Discussing the concept of substance-induced psychosis (SIP). *Psychological Medicine* 1–5. https://doi.org/10.1017/S0033291724001442

Received: 7 February 2024
Revised: 2 May 2024
Accepted: 17 May 2024

Keywords:
substance induced psychosis; first episode psychosis; diagnostic criteria; DSM-5; ICD-10

Corresponding author:
Jørgen G. Bramness;
Email: jobr@fhi.no

Abstract

Substance-induced psychosis (SIP) is characterized by both substance use and a psychotic state, and it is assumed that the first causes the latter. In ICD-10 the diagnosis is categorized as and grouped together with substance use disorders, and to a large extent also treated as such in the health care system. Though criticism of the diagnostic construct of SIP dates back several decades, numerous large and high-quality studies have been published during the past 5–10 years that substantiate and amplify this critique. The way we understand SIP and even how we name it is of major importance for treatment and it has judicial consequences. It has been demonstrated that substance use alone is not sufficient to cause psychosis, and that other risk factors besides substance use are at play. These are risk factors that are also known to be associated with schizophrenia spectrum disorders. Furthermore, register-based studies from several different countries find that a large proportion, around one in four, of those who are initially diagnosed with an SIP over time are subsequently diagnosed with a schizophrenia spectrum disorder. This scoping review discusses the construct validity of SIP considering recent evidence. We challenge the immanent causal assumption in SIP, and advocate that the condition shares many features with the schizophrenia spectrum disorders. In conclusion, we argue that SIP just as well could be considered a first-episode psychotic disorder in patients with substance use.

The diagnosis of substance-induced psychosis

Substance-induced psychosis (SIP) is a psychotic condition appearing in close temporal relationship to drug intake in people without primary psychotic disorders. In ICD-10, SIP is coded as a decimal specification of a substance use disorder, F1x.5, where '.5' indicates a psychotic state and the x is replaced by numbers from one to nine according to which substance is believed to have caused the psychosis. In ICD-11, the system is kept, but evolved to differentiate between even more substances. The North American system of DSM 5 indicates a diagnosis of 292.1 or 292.2 for alcohol or substance/medication-induced psychosis, respectively.

There is scarce knowledge regarding how common SIP is. We recently found a relatively stable annual incidence rate of SIP in treatment in Scandinavia over the past twenty years of between 9.3 and 14.1 per 100.000 person-years, though cannabis-induced psychosis significantly increased in the second half of the period (Rognli et al., 2022). We are not aware of similar studies outside of Scandinavia. Further, SIP represents between 6.5% (Thompson et al., 2016) and 10.3% (O'Connell, Sunwoo, McGorry, & O'Donoghue, 2019) of all first-episode psychoses (FEP) entering early intervention services.

In the acute phase, SIP and schizophrenia are hard to distinguish (Dawe, Geppert, Occhipinti, & Kingswell, 2011; Medhus, Mordal, Holm, Mørland, & Bramness, 2013b). The difference is related to time, as a SIP should (Kendler, Ohlsson, Sundquist, & Sundquist, 2019) resolve relatively rapidly with abstinence, reflected in the diagnostic criteria. According to ICD-10 (and similarly in ICD-11), symptoms should be significantly attenuated within one month and completely within six months (World Health Organization, 2004). According to the DSM-5, a SIP diagnosis should not be given if the psychosis lasts 'a considerable period of time, for example over one month' (American Psychiatric Association, 2013).

The neurobiological mechanisms underlying different SIPs may vary from one drug to another. Specifically, cannabis, amphetamines, and cocaine, as well as hallucinogens exhibit

distinct neurobiological effects that *could* contribute to SIP, as described well in two recent reviews (Fiorentini et al., 2021; Garson, Castle, & George, 2023). We fully acknowledge the capacity of some types of substances to, at times and in some, precipitate psychotic symptoms. These associations and possible mechanisms are well described by others, such as the mentioned reviews, and will not be covered here.

In this current narrative review, we argue that even though there are properties with many drugs of abuse that could increase the risk of SIP, these characteristics do not sufficiently explain SIP. The construct validity of SIP has been questioned (Mathias, Lubman, & Hides, 2008; Thirthalli, Benegal, & Gangadhar, 2010), and recent evidence has further substantiated this critique. Our aim is to present an updated understanding of SIP and to discuss implications of this understanding. Ultimately, we challenge the concept of SIP altogether.

An assumption of cause

In earlier times, diagnoses often included information about the assumed cause of the mental disorder, such as reactive and endogenous depression and psychosis. Modern diagnostics place more emphasis on describing signs and symptoms and avoid pointing to possible underlying causes. In the introduction to DSM III in 1980, Robert L. Spitzer wrote: '*For most* […] *disorders, however, the etiology is unknown. A variety of theories have been advanced, buttressed by evidence – not always convincing – to explain how these disorders come about. The approach taken in DSM-III is atheoretical with regard to etiology or pathophysiological process except for those disorders for which this is well established and therefore included in the definition of the disorder*' (Spitzer, JB, & Skodol, 1980). With the introduction of DSM III, it was thus recognized that for most mental disorders the cause is largely unknown and at least very complex and varies from individual to individual. Diagnoses hence need to be descriptive. A diagnose should be based on what we can observe and describe, not be causal or explanatory, because we rarely know or fully understand the cause of the disorder. Some diagnoses deviate from this principle. In Post-Traumatic Stress Disorder (PTSD), the assumption of cause may be seen as more immanent, and treatment focuses on both the cause (reprocessing the traumatic event) and its consequences (dampening anxiety and avoidance) (Kirkpatrick & Heller, 2014). However, even in PTSD vulnerability will play a role as the majority of those exposed to potential traumatic events do not develop PTSD. The term 'substance-induced psychosis' is also an explanatory diagnosis. But SIP differs from PTSD in terms of how the causality assumptions impact treatment. In SIP, removing the assumed cause (drug use) is the predominant intervention, while treatment of the consequence (psychosis) to receives less attention. As we shall see, substance use is not a sufficient cause of SIP, and several other risk factors are largely overlooked, partly due to this nomenclature. We argue that in SIP, the assumption of cause disproportionately guides our attention to substance use, possibly resulting in suboptimal treatment.

Substance use is not the cause of SIP

The fact that an SIP diagnosis follows the use of substances is not an argument for causality, but rather a tautology, driven by the diagnostic criteria themselves. It does not bring us very far in understanding the nature of SIP. Even if substance use forgoes the psychosis and some ways precipitates it, substance use only becomes a *necessary* risk factor because we choose to have a distinct diagnostic category for it. Contrary to this we believe it should be viewed as only one of many risk factors for SIP. It is necessary, also given the diagnostic criteria, but it is not sufficient. The majority of those who use any drug do not become psychotic. This applies even to those with extensive drug use. For instance, a prospective cohort study on methamphetamine users showed a strong dose–response association between days of use and probability of having psychotic symptoms the same month, but even among the most frequent users, some had no psychotic symptoms (McKetin, Lubman, Baker, Dawe, & Ali, 2013). In experimental studies where subjects were administered increasing amounts of amphetamines or cannabis to induce psychosis, not everyone became psychotic, even at high doses (Angrist & Gershon, 1970; van der Steur, Batalla, & Bossong, 2020). We have previously published a study comparing blood concentrations of amphetamines in people admitted to an acute psychiatric ward with blood concentration in apprehended drivers suspected for driving under the influence (Medhus, Holm, Mørland, & Bramness, 2013a). Interestingly, among those positive for amphetamines, blood concentrations were in fact *lower* among the psychiatric patients than among the apprehended drivers, indicating that other factors than amphetamine blood concentration are essential for the development of psychosis.

A relatively recent study from one of the co-authors showed that infections, which have been identified as possible risk factor for schizophrenia, are also associated with an increased risk of developing SIP (Hjorthøj, Starzer, Benros, & Nordentoft, 2020). Another study showed that family vulnerability to psychosis is significantly more common in those who developed SIP compared to the general population (Kendler et al., 2019; McKetin et al., 2023). More studies are needed to identify other risk factors for SIP besides substance use, but these examples demonstrate that *vulnerability* is also a key factor for SIP, similarly as for the development of primary psychosis (Løberg et al., 2014). In other words, just like for other mental disorders, the etiology of SIP is multifaceted, and the idea of substance use as *the* cause stands corrected in the view of scientific evidence.

The crucial question is whether substance use can cause psychosis in individuals who, *if they had not used* drugs, would have remained healthy. Large, randomized experiments might bring us closer to an answer, but would of course be unethical and impossible to conduct. At the individual level, it will be impossible to know whether the person would have developed psychosis even without drug intake.

One in four with SIP later receive a schizophrenia diagnosis

The knowledge that around one in four with SIP after some years are diagnosed with a schizophrenia-spectrum disorder, is relatively new. This was first described in 2007, when a 1-year follow-up study of 319 cases of SIP found a transition rate of 25% (Caton et al., 2007). Some years later, in 2013, a large register-based study including more than 18.000 SIP cases found highest transition rate to schizophrenia-spectrum disorder for cannabis-induced psychosis (46%) and lowest for alcohol (5%), but without reporting a pooled transition rate for all SIPs together. Additional large, register-based studies from different countries followed from 2017 and onwards (Alderson et al., 2017; Kendler et al., 2019; Rognli, Heiberg, Jacobsen, Høye, & Bramness, 2023; Starzer, Nordentoft, & Hjorthoj, 2018), and a

https://doi.org/10.1017/S0033291724001442 Published online by Cambridge University Press

meta-analysis in 2020 described a pooled transition rate of 25% for all SIPs together, and the highest for cannabis-induced psychosis (34%) (Murrie, Lappin, Large, & Sara, 2020). No other risk factors for schizophrenia have an effect size of comparable magnitude. People with SIP, particularly those associated with cannabis use, have comparable transition rates to schizophrenia as what we see for those with brief psychotic episode (ICD-10 F23) and atypical psychoses (ICD-10 F29) (Murrie et al., 2020). The understanding of the possible implications of these findings have shifted over time. While the authors of the first paper stated that *'the distinction between a SIP and a primary psychotic disorder is important because these two disorders require fundamentally different approaches to treatment'* (Caton et al., 2007), an editorial following the most recent transition paper (Rognli et al., 2023) emphasized the need for early intervention efforts for individuals experiencing SIP (Vassos, 2023).

Repeated episodes of SIP are associated with an increased risk of a diagnostic transition to schizophrenia (Crebbin, Mitford, Paxton, & Turkington, 2009; Kendler et al., 2019; Rognli et al., 2023). The risk of transition is also increased with longer first admission for SIP, which probably is an indicator of the severity of the episode (Alderson et al., 2017).

Figure 1 illustrates that the risk factors for psychosis in the landscape of drug use v. SIP v. transition to schizophrenia may influence *either* the precipitation of SIP (step 1) *or* the transition to schizophrenia (step 2). If these risk factors are more important in the first step, this indicates that SIP resembles more a schizophrenia spectrum disorder, while a greater influence on the transition step would indicate SIP not being so closely related to schizophrenia spectrum disorders. Only a few studies have investigated this. The study by Kendler and coworkers (Kendler et al., 2019) and the study by Hjorthøj and coworkers (Hjorthøj et al., 2020) investigated if known risk factors for schizophrenia also increase the risk for SIP (step 1) and the transition from SIP to schizophrenia (step 2). Hjorthøj found that serious infections are a risk factor SIP, but not for the transition from SIP to schizophrenia, providing evidence for step 1 (Hjorthøj et al., 2020). This speaks for SIP belonging more to the schizophrenia spectrum. Kendler on the other hand found that familial vulnerability predicts both SIP, and the transition to schizophrenia (Kendler et al., 2019), giving support to both steps. The family-based vulnerability was a greater risk factor for the transition, than for SIP, at a level we would find for people receiving a diagnosis of schizophrenia, without first having a SIP diagnosis.

How does this affect our understanding of SIP? For those who convert, the initial diagnostic assessment of SIP could of course have been wrong, and the correct understanding of the repeated episodes was that they were signs of an underlying chronic disorder (schizophrenia), with periodical (drug-induced) symptom exacerbations. Alternatively, something fundamental changed over time and with repeated admissions, causing a different disorder to occur. However, when asking if the SIP was erroneously diagnosed in the first place (if the 'true' diagnosis in fact would have been schizophrenia) or if the diagnosis changed from SIP to schizophrenia, it can be useful to remind ourselves that diagnostic cut-offs are constructed. The psychosis continuum perspective advocated by among others Jim van Os et al. describes how psychosis proneness over time may make psychotic symptoms become abnormally persistent and subsequently clinically relevant, depending among other things on the degree of environmental risk the person is additionally exposed to (van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009). This is in our view a third and more viable understanding, and one which is compatible with placing SIP in the psychosis continuum.

Clinical implications

There may be good reasons for being restrictive in diagnosing people with schizophrenia-spectrum disorder (Mitchell, 2007). Clinicians may not want to give the patient too poor a prognosis and 'label' people with something that could be a chronic illness. However, for fear of falling into one ditch, there is a risk of falling into the opposite, namely that by holding back, sick people do not get the help they need. This applies to people with repeated SIPs without healthcare personnel being able to properly assess and treat the psychotic symptoms.

There are comprehensive clinical guidelines for the treatment of first-episode psychosis (FEP), and they recommend rapid and high-quality interventions in many areas, and long-term follow-up (Early Psychosis Guidelines Writing Group, 2016; International Early Psychosis Association Writing Group, 2005; NICE, 2013). The brief interventions aim to reduce the duration of untreated psychosis, as this is considered one of the few potentially modifiable predictors of the outcome of schizophrenia (Malla, 2022; Penttilä, Jääskeläinen, Hirvonen, Isohanni, & Miettunen, 2014). However, SIP is not considered a FEP, and efforts to reduce duration of untreated psychosis seemingly do not apply to these patients. Most clinical guidelines on comorbid psychosis and substance use disorder prerequisite that the patient has an F2x-category diagnosis (NICE, 2011, 2013, 2016, 2020). To the extent that guidelines for SIP exist, the recommendations are mostly limited to stopping drug use. The message is also that differential diagnosis in relation to schizophrenia-spectrum psychosis is difficult, but that it is important to arrive at the correct diagnostic conclusion since the implications for further follow-up are great. This indicates that SIP is to be treated differently. In this way, a diagnosis of F1x.5 v. F2 seems to have great consequences. Our diagnostic practice may thus contribute to a poorer prognosis. This is also reflected in the recent review that states the striking paucity of information on outcomes, treatments, and best practices of SIP (Fiorentini et al., 2021).

Dual diagnosis was first described in the 1980s, referring to individuals with coexisting severe mental illness and substance use disorders (Buckley, 2006; Drake, Mercer-McFadden, Mueser, McHugo, & Bond, 1998). Is SIP in fact automatically a dual diagnosis as F1x.5 by necessity will include harmful use of a substance (F1x.1 refers to use that gives negative health consequences), and as psychosis is the negative health consequence? Or does one need something from *both* the F1 and F2 chapters to be considered having a dual diagnosis? And in that sense, does F1x.5 represent a move to avoid dual diagnosis? Some have called for better definition of dual disorders (Hryb, Kirkhart, & Talbert, 2007).

SIP is in most cases treated in the acute phase in mental health care. There are many indications that it would also be sensible to follow these patients in mental health care, as is the case for other types of psychotic disorders. In the event of an FEP, the patient is usually offered long-term follow-up, including pharmacotherapy, various forms of psychotherapy, family follow-up, as well as a focus on physical health, social life, housing, work, and collaboration with the municipality (Norwegian Directorate of Health, 2013). For SIP, our impression is that the typical treatment is a brief hospitalization at an acute psychiatric ward, and discharge with a request to refrain from substance use, perhaps

https://doi.org/10.1017/S0033291724001442 Published online by Cambridge University Press

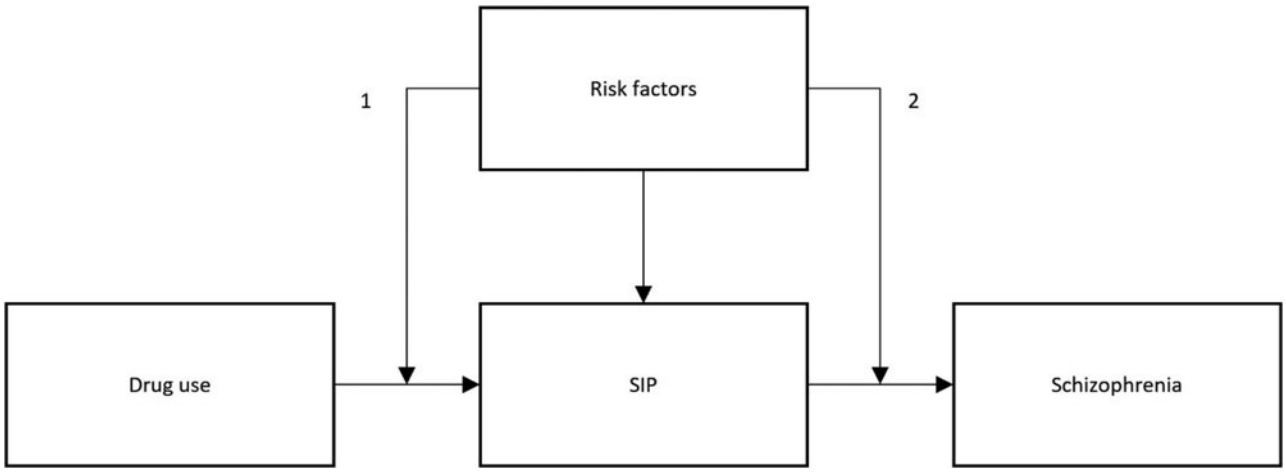

Figure 1. The influence of vulnerability on the relationship between drug use, SIP, and schizophrenia. If the vulnerability influences the precipitation of SIP (step 1) rather than the transition to schizophrenia (step 2) this speaks for moving SIP closer to the primary psychosis realm.

accompanied by a referral to an outpatient clinic for substance use disorder treatment. Here we believe that the diagnosis may hamper proper follow-up. Research from the last decade could encourage us to, rather than viewing SIP as a specification of a substance use disorder, consider it as a type of psychotic disorder. Or, in other words, a SIP should be considered a FEP in an individual who uses substances. We believe that the evidence base indicates that SIP, at least those with recurring episodes, should be offered comparable treatment as other FEPs. It is noteworthy that in a recently published paper SIP is viewed as a sub-category of FEP, also because the disorders are undisguisable in a clinical setting (Inchausti, Gorostiza, Gonzalez Torres, & Oraa, 2023).

Ideally, a sound clinical assessment should guide treatment, based on the patient's condition and needs, regardless of diagnostic codes. However, in a fragmented and specialized healthcare system, with scarce resources and a push towards rapid discharge, we know that diagnostic codes matter regarding what department is responsible for which patients. We are aware that in clinical practice some use a diagnosis of unspecified psychosis (ICD-10 F29) together with a diagnosis of harmful use of the substance (F1x.1) instead of a diagnosis of SIP (F1x.5), because this ensures better follow-up. This illustrates how diagnostic categories may serve as gatekeepers for treatment.

There are also important judicial aspects concerning the placing of SIP in the ICD-10 F1 or F2 chapters. In many jurisdictions people who are intoxicated are viewed as criminally sane, meaning that even if they are psychotic following drug use, they stand trial and serve time, different from people with schizophrenia spectrum disorder, who often are referred to treatment, not incarcerated. This has been problematized also by other research groups (Dominique, 2013; Liu et al., 2017).

Conclusion

The main problem with the diagnostic term 'substance-induced psychosis' is that it exaggerates the importance of substance use and that this impacts treatment. The importance of vulnerability even in the acute phase, the similarity in symptoms with schizophrenia, and the major transition rate to primary psychosis suggest that the condition belongs just as much in the F2 chapter as in the F1 chapter in the ICD-10. The fact that these the psychotic episode was preceded by substance use indicates that these patients should avoid using drugs, but such a recommendation could also be directed to other patients experiencing psychosis. Guidelines should be developed that recommend treatment and follow-up closer to what is done with FEP. This would be in line with updated evidence. We need to give these patients quicker access to specialized psychosis treatment, which could shorten the duration of untreated psychosis, improve prognosis, and perhaps even prevent transition to schizophrenia.

Data availability statement. Not applicable.

Funding statement. Funded by internal sources only.

Competing interests. None.

Ethics approval and consent to participate. Not applicable.

Consent for publication. Not applicable.

References

Alderson, H. L., Semple, D. M., Blayney, C., Queirazza, F., Chekuri, V., & Lawrie, S. M. (2017a). Risk of transition to schizophrenia following first admission with substance-induced psychotic disorder: A population-based longitudinal cohort study. *Psychological Medicine*, *47*(14), 2548–2555. doi:10.1017/S0033291717001118

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Association.

Angrist, B. M., & Gershon, S. (1970). The phenomenology of experimentally induced amphetamine psychosis--preliminary observations. *Biological Psychiatry*, *2*(2), 95–107.

Buckley, P. F. (2006). Prevalence and consequences of the dual diagnosis of substance abuse and severe mental illness. *Journal of Clinical Psychiatry*, *67*(Suppl 7), 5–9.

Caton, C. L. M., Hasin, D., Shrout, P. E., Drake, R. E., Dominguez, B., First, M. B., … Schanzer, W. B. (2007). Stability of early-phase primary psychotic disorders with concurrent substance use and substance-induced psychosis. *British Journal of Psychiatry*, *190*, 105–111. doi:10.1192/bjp.bp.105.015784

Crebbin, K., Mitford, E., Paxton, R., & Turkington, D. (2009). First-episode drug-induced psychosis: A medium term follow up study reveals a high-risk group. *Social Psychiatry & Psychiatric Epidemiology*, *44*(9), 710–715. doi:10.1007/s00127-008-0490-2

https://doi.org/10.1017/S0033291724001442 Published online by Cambridge University Press

Dawe, S., Geppert, L., Occhipinti, S., & Kingswell, W. (2011). A comparison of the symptoms and short-term clinical course in inpatients with substance-induced psychosis and primary psychosis. *Journal of Substance Abuse & Treatment*, *40*(1), 95–101. doi:10.1016/j.jsat.2010.08.002

Dominique, B. (2013). Forensic considerations of substance-induced psychosis. *Journal of the American Academy of Psychiatry and the Law Online*, *41*(2), 168. Retrieved from http://jaapl.org/content/41/2/168.abstract

Drake, R. E., Mercer-McFadden, C., Mueser, K. T., McHugo, G. J., & Bond, G. R. (1998). Review of integrated mental health and substance abuse treatment for patients with dual disorders. *Schizophrenia Bulletin*, *24*(4), 589–608. doi:10.1093/oxfordjournals.schbul.a033351

Early Psychosis Guidelines Writing Group. (2016). *Australian Clinical Guidelines for Early Psychosis*. Retrieved from Melbourne.

Fiorentini, A., Cantù, F., Crisanti, C., Cereda, G., Oldani, L., & Brambilla, P. (2021). Substance-induced psychoses: An updated literature review. *Frontiers in Psychiatry*, *12*, 694863. doi:10.3389/fpsyt.2021.694863

Garson, E., Castle, D. J., & George, T. P. (2023). Substance-induced psychosis: A narrative review. *Current Addiction Reports*, *10*(2), 335–340. doi:10.1007/s40429-023-00475-6

Hjorthøj, C., Starzer, M. S. K., Benros, M. E., & Nordentoft, M. (2020). Infections as a risk factor for and prognostic factor after substance-induced psychoses. *American Journal of Psychiatry*, *177*(4), 335–341.

Hryb, K., Kirkhart, R., & Talbert, R. (2007). A call for standardized definition of dual diagnosis. *Psychiatry* (*Edgmont* (*PA. : Township*)), *4*(9), 15–16. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2880934/

Inchausti, L., Gorostiza, I., Gonzalez Torres, M. A., & Oraa, R. (2023). The transition to Schizophrenia spectrum disorder from a first psychotic episode that did or did not appear to be induced by substance use. *Psychiatry Research*, *328*, 115475. doi:10.1016/j.psychres.2023.115475

International Early Psychosis Association Writing Group. (2005). International clinical practice guidelines for early psychosis. *British Journal of Psychiatry Supplement*, *48*, s120–s124. doi:10.1192/bjp.187.48.s120

Kendler, K. S., Ohlsson, H., Sundquist, J., & Sundquist, K. (2019). Prediction of onset of substance-induced psychotic disorder and its progression to schizophrenia in a Swedish National sample. *American Journal of Psychiatry*, *176*(9), 711–719. doi:10.1176/appi.ajp.2019.18101217

Kirkpatrick, H. A., & Heller, G. M. (2014). Post-traumatic stress disorder: Theory and treatment update. *International Journal of Psychiatry & Medicine*, *47*(4), 337–346. doi:10.2190/PM.47.4.h

Liu, Y., Hao, B., Shi, Y., Xue, L., Wang, X., Chen, Y., & Zhao, H. (2017). Violent offences of methamphetamine users and dilemmas of forensic psychiatric assessment. *Forensic Science Research*, *2*(1), 11–17. doi:10.1080/20961790.2017.1287155

Løberg, E. M., Helle, S., Nygård, M., Berle, J., Kroken, R. A., & Johnsen, E. (2014). The cannabis pathway to non-affective psychosis may reflect less neurobiological vulnerability. *Frontiers in Psychiatry*, *5*, 159. doi:10.3389/fpsyt.2014.00159

Malla, A. (2022). Reducing duration of untreated psychosis: The neglected dimension of early intervention services. *American Journal of Psychiatry*, *179*(4), 259–261. doi:10.1176/appi.ajp.20220154

Mathias, S., Lubman, D. I., & Hides, L. (2008). Substance-induced psychosis: A diagnostic conundrum. *Journal of Clinical Psychiatry*, *69*(3), 358–367. doi:10.4088/jcp.v69n0304

McKetin, R., Clare, P. J., Castle, D., Turner, A., Kelly, P. J., Lubman, D. I., … Berk, M. (2023). How does a family history of psychosis influence the risk of methamphetamine-related psychotic symptoms: Evidence from longitudinal panel data. *Addiction*, *118*(10), 1975–1983. doi:10.1111/add.16230

McKetin, R., Lubman, D. I., Baker, A. L., Dawe, S., & Ali, R. L. (2013). Dose-related psychotic symptoms in chronic methamphetamine users: Evidence from a prospective longitudinal study. *JAMA Psychiatry*, *70*(3), 319–324. doi:10.1001/jamapsychiatry.2013.283

Medhus, S., Holm, B., Mørland, J., & Bramness, J. G. (2013a). Association between methamphetamine versus amphetamine and acute psychiatric symptoms. *Journal of Dual Diagnosis*, *9*(4), 292–300. doi:10.1080/15504263.2013.835161

Medhus, S., Mordal, J., Holm, B., Mørland, J., & Bramness, J. G. (2013b). A comparison of symptoms and drug use between patients with methamphetamine associated psychoses and patients diagnosed with schizophrenia in two acute psychiatric wards. *Psychiatry Research*, *206*(1), 17–21. doi:10.1016/j.psychres.2012.09.023

Mitchell, A. J. (2007). Reluctance to disclose difficult diagnoses: A narrative review comparing communication by psychiatrists and oncologists. *Supportive Care in Cancer: Official Journal of the Multinational Association of Supportive Care in Cancer*, *15*(7), 819–828. doi:10.1007/s00520-007-0226-y

Murrie, B., Lappin, J., Large, M., & Sara, G. (2020). Transition of substance-induced, brief, and atypical psychoses to schizophrenia: A systematic review and meta-analysis. *Schizophrenia Bulletin*, *46*(3), 505–516. doi:10.1093/schbul/sbz102

NICE. (2011). *Coexisting severe mental illness* (*psychosis*) *and substance misuse: Assessment and management in healthcare settings*. London: NICE

NICE. (2013). *Psychosis and schizophrenia in children and young people: Recognition and management*. London: NICE.

NICE. (2016). *Coexisting severe mental illness and substance misuse: Community care and social care services*. London: NICE.

NICE. (2020). *Rehabilitation for adults with complex psychosis*. London: NICE.

Norwegian Directorate of Health. (2013). Assessment, treatment, and follow-up of persons with psychosis disorders. Retrieved from Oslo.

O'Connell, J., Sunwoo, M., McGorry, P., & O'Donoghue, B. (2019). Characteristics and outcomes of young people with substance induced psychotic disorder. *Schizophrenia Research*, *206*, 257–262. doi:10.1016/j.schres.2018.11.007

Penttilä, M., Jääskeläinen, E., Hirvonen, N., Isohanni, M., & Miettunen, J. (2014). Duration of untreated psychosis as predictor of long-term outcome in schizophrenia: Systematic review and meta-analysis. *British Journal of Psychiatry*, *205*(2), 88–94. doi:10.1192/bjp.bp.113.127753

Rognli, E. B., Heiberg, I. H., Jacobsen, B. K., Høye, A., & Bramness, J. G. (2023). Transition from substance-induced psychosis to schizophrenia spectrum disorder or bipolar disorder. *American Journal of Psychiatry*, *180*(6), 437–444.

Rognli, E. B., Taipale, H., Hjorthøj, C., Mittendorfer-Rutz, E., Bramness, J. G., Heiberg, I. H., … Niemelä, S. (2022). Annual incidence of substance-induced psychoses in Scandinavia from 2000 to 2016. *Psychological Medicine*, *53*(11), 5246–5255. doi:10.1017/S00332917229X

Spitzer, R., JB, W., & Skodol, A. (1980). DSM-III: The major achievements and an overview. *American Journal of Psychiatry*, *137*(2), 151–164. doi:10.1176/ajp.137.2.151

Starzer, M. S. K., Nordentoft, M., & Hjorthoj, C. (2018). Rates and predictors of conversion to schizophrenia or bipolar disorder following substance-induced psychosis. *American Journal of Psychiatry*, *175*(4), 343–350. doi:10.1176/appi.ajp.2017.17020223

Thirthalli, J., Benegal, V., & Gangadhar, B. N. (2010). Substance-induced psychosis: An overview. In M. S. Keshavan & P. S. Sachdev (Eds.), *Secondary schizophrenia* (pp. 112–126). Cambridge: Cambridge University Press.

Thompson, A., Marwaha, S., Winsper, C., Everard, L., Jones, P. B., Fowler, D., … Marshall, M. (2016). Short-term outcome of substance-induced psychotic disorder in a large UK first episode psychosis cohort. *Acta Psychiatrica Scandinavica*, *134*(4), 321–328. doi:10.1111/acps.12623

van der Steur, S. J., Batalla, A., & Bossong, M. G. (2020). Factors moderating the association between cannabis use and psychosis risk: A systematic review. *Brain Sciences*, *10*(2), 97. doi:10.3390/brainsci10020097

van Os, J., Linscott, R. J., Myin-Germeys, I., Delespaul, P., & Krabbendam, L. (2009). A systematic review and meta-analysis of the psychosis continuum: Evidence for a psychosis proneness-persistence-impairment model of psychotic disorder. *Psychological Medicine*, *39*(2), 179–195. doi:10.1017/s0033291708003814

Vassos, E. (2023). What is the link between substance-induced psychosis and primary psychotic disorders? *American Journal of Psychiatry*, *180*(6), 404–406. doi:10.1176/appi.ajp.20230298

World Health Organization. (2004). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines* (2nd ed.). Geneva: World Health Organization.

https://doi.org/10.1017/S0033291724001442 Published online by Cambridge University Press