

# Reasons for increased substance use in psychosis

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## Abstract

Around half of all patients with schizophrenia are thought to abuse drugs or alcohol and there is good evidence to suggest that they have poorer outcomes than their non substance using counterparts. However, despite more than twenty years of research there is still no consensus on the aetiology of increased rates of substance use in people with psychosis. There is a clear need to understand the reasons for such high rates of substance use if treatments designed to help patients abstain from substance use are to be successful. This paper provides an update of the literature examining the reasons for substance use by people with psychosis, and includes a comprehensive review of the self report literature. The main theories as to why people with psychosis use substances are presented. There is evidence to suggest that cannabis may have a causal role in the development of psychopathology but not for other substances. The self report literature provides support for an ‘alleviation of dysphoria’ model of substance use but there is little empirical support for the self medication hypothesis, or for common factor models and bidirectional models of comorbidity. It is likely that there are multiple risk factors involved in substance use in psychosis and more work to develop and test multiple risk factor models is required.

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## 1. Introduction

A substantial number of patients with schizophrenia are known to abuse drugs and alcohol (Mueser, Yarnold, & Bellack, 1992; Regier et al., 1990) and rates of substance use are significantly higher in this group than in the general population (Regier et al., 1990). Comorbidity has profound implications for the course and treatment of schizophrenia: there is good evidence to suggest that people with schizophrenia who abuse drugs and alcohol have poorer outcomes than both their non substance using counterparts and substance users in the general population (e.g. Drake & Wallach, 1989; Margolese, Malchy, Negrete, Tempier, & Gill, 2004). Whilst substance use disorders have such negative consequences for this patient group, motivation for reduction of substance use in clients with psychosis is usually low (Baker et al., 2002; Barrowclough et al., 2001) Hence there is a clear need to understand the reasons for such high rates use of substance if treatments designed to help patients reduce their substance use are to be successful. The aim of this

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paper is to review the literature which examines the causes of and reasons for substance use by people with schizophrenia, and to critically review research on the self reported reasons for substance use by this client group.

Research examining the relationship between psychosis and substance use has continued apace since the publication of previous reviews (e.g. Batel, 2000; Blanchard, Brown, Horan, & Sherwood, 2000; Mueser, Drake, & Wallach, 1998) and a number of good quality studies have been conducted with the aim of investigating reasons for increased comorbidity in this client group. Six prospective cohort studies examining the link between cannabis use and psychosis have been reported in the past five years and eight investigations of the self reported reasons for substance use have been conducted. Prior to 2000 there was little in the way of longitudinal research and the studies examining the self reported effects or reasons for substance use had significant methodological limitations (Green, Kavanagh, & Young, 2004). Hence an updated review of the literature in this area is timely. To provide a context to the review, the prevalence of substance use by patients with schizophrenia will first be discussed and the correlates and the consequences of substance use by this patient group will be described.

## 2. Substance use prevalence

Estimates of lifetime prevalence for individuals with schizophrenia are around 50% (Mueser, Bennet, & Kushner, 1995; Regier et al., 1990) and rates for current substance use have been reported to be as high as 65% in some samples (Mueser et al., 1992). Estimates vary significantly across studies, primarily because of methodological differences such as the way that such “dual diagnosis” is defined. Such differences include the diagnostic criteria for both psychosis and substance use; the validity of the measures employed to assess both disorders; the population that is being investigated (for example, whether inpatient or outpatient) and the location of that population (the country the research is taking place in and whether the population is rural or urban) but nevertheless, the overwhelming majority of studies have reported that substance use disorders are more prevalent in patients with psychosis than in the general population.

In the largest prevalence study conducted in the US (the Epidemiologic Catchment Area Study, ECA, Regier et al., 1990) more than twenty thousand structured interviews were conducted. More than a quarter (27%) of those with schizophrenia had experienced a drug abuse disorder in comparison to 6.1% of the general population and one third (33.7%) had an alcohol disorder compared to 13.5% in the general population. For individuals with schizophrenia, the odds of having an alcohol disorder were three times higher than in the general population and the odds of having another substance use disorder were six times higher. Overall, 47% of people with schizophrenia had experienced some substance abuse or dependence. The US National Comorbidity study (Kessler, Crum et al., 1997) reported comparable lifetime comorbidity rates to the ECA.

The UK studies have tended to reveal lower lifetime prevalence rates than those in the US. The UK national psychiatric morbidity survey (Farrell et al., 1998) reported a lifetime substance use prevalence rate of 7% for those with schizophrenia, delusional disorders or schizoaffective disorders and Duke, Pantelis, McPhillips, and Barnes (2001) reported a lifetime prevalence rate of 16% for substance use by patients with schizophrenia in a London based survey. More recently, the West London First-Episode Schizophrenia study (Barnes, Mutsatsa, Hutton, Watt, & Joyce, 2006) reported lifetime rates of 27% for problems with alcohol use and 68% for lifetime substance use.

Current and one-year substance use prevalence rates range between 19.5% (Cantwell et al., 1999) and 44% (Weaver et al., 2003) in the UK and are broadly comparable to those reported in the US (Kessler, Crum et al., 1997; Swartz et al., 2006).

Research in Germany (Soyka et al., 1993); Finland (Korkeila et al., 2005) Italy (Mauri et al., 2006); Canada (Margolese et al., 2004; Van Mastrigt, Addington, & Addington, 2004); Australia (Jablensky et al., 2000) and Brazil (Rossi Menezes & Ratto, 2004) has demonstrated considerable variability in both prevalence rates and in patterns of use across countries. Undoubtedly some of this variability will be due to methodological differences between the studies but it may also indicate that substance use comorbidity depends on environmental and cultural differences, including drug availability.

The types of substance used by patients with schizophrenia vary widely (Schneier & Siris, 1987). Alcohol and cannabis are the substances used most commonly in both US and UK samples (e.g. Kessler, Crum et al., 1997; Weaver et al., 2003) but patterns of stimulant and opiate use vary across studies. For many ‘dually diagnosed’ patients multiple drug and alcohol use is common, with a significant number of patients with schizophrenia abusing more than one substance (Baigent, Holme, & Hafner, 1995; Drake, Osher, & Wallach, 1989). In the study by Weaver et al. (2003) 40.2% of those reporting problem drug use also reported harmful alcohol use. This polysubstance use means that it is often difficult to disentangle the correlates of and the effects of different substances or classes of substances.

### 3. Correlates of substance use

The demographic correlates of substance use are well documented (e.g. Kavanagh et al., 2004; Sevy et al., 2001). Demographic profiles vary according to the type of substance used (Mueser et al., 1992), for example alcohol users tend to be older than users of non-alcoholic substances (Salyers & Mueser, 2001), but there is some consistency in the other main correlates identified. People with schizophrenia who also have substance use disorders are more likely to be male than their non substance using counterparts, they also tend to be younger (with the exception of alcohol users), less well educated and are more likely to have a family history of substance use problems (e.g. Barnes et al., 2006; Cantwell, 2003; Kavanagh et al., 2004; Menezes et al., 1996; Mueser et al., 1995). Problem substance use has also been associated with an earlier onset of schizophrenia (Kovaszny, Fleischer, & Tanenberg-Karant, 1997; Mauri et al., 2006). Other, less reliable correlates include higher IQ (Sevy et al., 2001) and racial origin (Mueser et al., 1992). Relatively few studies have investigated the relationship between substance use and psychiatric history but those that have reported that substance use is associated with better premorbid functioning (e.g. Carey, Carey, & Simons, 2003; Dixon, Haas, Weiden, & Frances, 1991; Sevy et al., 2001). People with schizophrenia who are more socially active are reported to have increased exposure to substances through their social networks (Salyers & Mueser, 2001). The only reliable clinical correlate to be identified is antisocial personality disorder (e.g. Kavanagh et al., 2004). Studies have shown that patients with schizophrenia and antisocial personality disorder (ASPD) are more likely to have comorbid substance use disorder than patients without ASPD (Caton, Shrout, Eagle, Opler, & Felix, 1994; Mueser et al., 2000). Furthermore, Mueser, Drake et al. (1997), Mueser, Valentiner, and Agresta (1997) found that for patients with schizophrenia and substance use disorder ASPD is associated with a more severe course of substance use disorder including earlier age of onset and larger quantities of substance use.

### 4. Consequences of substance use

In addition to the negative impacts on a person's internal state caused by substance use (for example depressed mood, increased perceptual and cognitive anomalies, increased arousal, unpleasant withdrawal symptoms) and the physical consequences of drug or alcohol use (for example liver damage) there are a number of long term social and clinical consequences associated with drug and alcohol use. In common with substance users in the general population, substance users with schizophrenia are likely to experience financial problems associated with that use. They are also at increased risk of illness and injury (Dickey, Azeni, Weiss, & Sederer, 2000) including problems associated with risky behaviours such as unprotected sex and needle sharing, for example HIV (Carey et al., 2004). It has been argued that people with psychosis are particularly sensitive to the negative effects of certain substances (Chambers, Krystal, & Self, 2001; Verdoux, Gindre, Sorbara, Tournier, & Swendsen, 2003) and that negative consequences result from lower levels of use than in the general population (Drake et al., 1989).

Clinically, substance users with schizophrenia are at increased risk of poorer symptomatic and functional outcomes than their non substance using counterparts. Substance use is associated with more positive symptoms (Pencer & Addington, 2003) and with more relapses and hospitalizations (Linszen, Dingemans, & Lenior, 1994; Swofford, Kasckow, Scheller-Gilkey, & Inderbitzin, 1996). The study by Menezes et al. (1996) reported that inpatient admission rates among dually diagnosed patients were almost double those of patients with psychosis alone. Patients with schizophrenia who abuse drugs and/or alcohol have increased rates of suicidal ideation (Bartels, Drake, & McHugo, 1992; Hawton, Sutton, Haw, Sinclair, & Deeks, 2005; Kamali et al., 2000) increased aggression and violence (Cuffel, Shumway, Choulgian, & MacDonald, 1994; Fulwiler, Grossman, Forbes, & Ruthazer, 1997) and higher rates of treatment noncompliance (Coldham, Addington, & Addington, 2002; Drake & Wallach, 1989; Janssen et al., 2006; Owen, Fischer, Booth, & Cuffel, 1996) including medication non-adherence and failure to attend appointments. As a result, comorbid patients are more likely to be offered typical antipsychotic medication via depot injection. Substance users also tend to report greater extrapyramidal symptoms than abstinent patients (Potvin et al., 2006) and are at greater risk of tardive dyskinesia (Dixon, Weiden, Haas, Sweeny, & Frances, 1992). Other consequences of substance use in this patient group include interpersonal conflict and stress (Barrowclough, Ward, Wearden, & Gregg, 2005; Kashner et al., 1991) for example, conflict with relatives, partners and service providers who disapprove of substance use and blame clients for worsening their situation. People with schizophrenia who use substances are also at increased risk of social exclusion (Todd et al., 2004) and ultimately, homelessness and housing instability (Drake, Osher, & Wallach, 1991).

## 5. Explanations of comorbidity

Four broad explanations of substance use in schizophrenia have been suggested (Kushner & Mueser, 1993; Mueser et al., 1998): (1) substance use causes schizophrenia; (2) substance use is a consequence of schizophrenia; (3) schizophrenia and substance use share a common origin; and (4) schizophrenia and substance use interact and maintain each other. The bulk of the existing research literature has focused on the first two explanations of aetiology.

An understanding of the temporal relationship between the onset of schizophrenia and substance may help to elucidate whether either of the two disorders is primary (if substance use is generally found to occur prior to schizophrenia the second hypothesis would be less plausible). However, temporal order is extremely difficult to establish. Both schizophrenia and substance use disorder tend to develop gradually after beginning in adolescence or early adulthood, and the marked functional decline that accompanies them both makes it difficult to determine the most relevant factor. The studies that have attempted to establish temporal order have so far been contradictory. Silver and Abboud (1994), for example, reported that 60% of patients with schizophrenia who used drugs had done so before their first admission and Linszen et al. (1994) reported that cannabis use preceded onset of schizophrenia in 23 out of the 24 patients in their study. Cantwell et al.'s (1999) study of 168 patients with first episode schizophrenia found that over a third (37%) reported substance use before presentation to services. In contrast, the US national comorbidity survey (Kessler, Crum et al., 1997) noted that in patients with co-occurring mental health and substance related disorders the mental disorder developed first in the vast majority of cases. Hambrecht and Hafner (1996) conducted a retrospective study with 232 patients with schizophrenia and found that one third of patients had a drug problem for more than one year before the schizophrenia began, for another third the onset of schizophrenia occurred at a similar time to the onset of substance use and for the final third the began more than a year before the substance use. Hambrecht and Hafner interpreted these findings in terms of a vulnerability-stress-coping model stating that the first group might suffer have their vulnerability threshold reduced or their coping resources diminished as a result of their substance use. The second group might contain people who are already vulnerable to schizophrenia for whom substance misuse is a stress factor precipitating the onset of psychosis whilst the third group uses substances for self medicating against or 'coping with' the symptoms of schizophrenia.

The results of these studies are difficult to interpret and compare because of the choice of marker used to date illness onset. For Silver and Abboud (1994) the date of the first psychiatric admission was taken to be the onset of schizophrenia but for some patients, the first symptoms of schizophrenia appear months or even years before admission and may still, therefore, predate substance use. In other studies, onset of illness is based on patient reports but the use of retrospective patients' reports is also problematic: patients' memories of past states are likely to be unreliable, especially when complicated by intoxication.

### 5.1. Does substance use cause psychosis?

The studies that are best positioned to test whether substance use causes psychosis are prospective cohort studies (preferably from birth) but few of these exist and those that do have focused exclusively on the link between cannabis use and psychosis.

#### 5.1.1. Cannabis and psychosis

There is now a huge literature on the relationship between cannabis and psychosis: 180+ articles have been indexed on PubMed since 2000 with over 100 of these appearing in the last two years. Estimates of cannabis use by people with schizophrenia are high. Green, Young, and Kavanagh (2005) analysed prevalence data from 53 English language treatment studies and reported a prevalence rate for current use of 23% and 11.3% for current misuse. Lifetime prevalence rates for use and misuse were 42.1% and 22.5% respectively. A large number of studies have reported a significant association between cannabis use and psychosis and there is abundant evidence of the link between the two in epidemiological studies involving the general population (e.g. Cuffel, Heithoff, & Lawson, 1993; Degenhardt & Hall, 2001).

Recent experimental work investigating the effects of delta-9-tetrahydrocannabinol ( $\Delta$ -9-THC, the major psychoactive component of cannabis) provides evidence of an association between cannabis and psychosis:  $\Delta$ -9-THC produces schizophrenia-like positive and negative symptoms in healthy individuals (D'Souza et al., 2004) and transiently increases positive, negative and general schizophrenia symptoms in patients with schizophrenia (D'Souza et al., 2005). Furthermore,

patients with schizophrenia are more vulnerable to the effects of  $\Delta$ -9-THC than those without. Whilst confirming that cannabis can cause transient psychosis or transiently exacerbate existing psychosis these studies do not show whether cannabis can actually cause schizophrenia or other functional psychotic illness in the long term.

Evidence for a more long term causal association comes from the cohort studies which have examined the link in prospective longitudinal studies. The first of these studies was conducted by [Andreasson, Allebeck, Engstrom, and Rydberg \(1987\)](#). More than forty-five thousand soldiers who had been conscripted into the Swedish army were followed up for fifteen years. Data on substance use at the time of conscription were available and psychiatric diagnoses for the fifteen years after conscription were obtained. A strong relationship between history of cannabis use at baseline and presence of schizophrenia at follow up was reported. The 'heavy' cannabis users (defined as at least 50 occasions of use) were six times more likely to have a diagnosis of schizophrenia at follow up than less frequent users or those who had never used cannabis. Significantly, no such associations were found for any of the other drugs used. The follow up to this study ([Zammit, Allebeck, Andreasson, Lundberg, & Lewis, 2002](#)) reported that those who were 'heavy cannabis users' by the age of 18 were 6.7 times more likely to be diagnosed with schizophrenia. This result held even when the polysubstance users were excluded from the analysis and was reduced (but was still significant) when other confounds (low IQ, cigarette smoking, growing up in a city) were controlled for. The authors reported these results to be "consistent with a causal relationship between cannabis use and schizophrenia".

The results of the Swedish study have since been replicated in six more prospective cohort studies: two conducted in the Netherlands ([Ferdinand et al., 2005](#); [van Os et al., 2002](#)), one conducted in Germany ([Henquet et al., 2005](#)), two in New Zealand ([Arseneault et al., 2002](#); [Fergusson, Horwood, & Ridder, 2005](#)) and one in Israel ([Weiser, Knobler, Noy, & Kaplan, 2002](#)). The first Netherlands study ([van Os et al., 2002](#)) used data from the Netherlands Mental Health Survey and Incidence Study and followed 4045 people between 1996 and 1999. Participants with no psychotic disorder at baseline who were also cannabis users were at increased risk of clinically significant psychotic symptoms at the end of follow up (OR = 3.5–3.7). This association was independent of any comorbid non-psychotic psychiatric disorder or use of other substances at baseline. The second Netherlands study ([Ferdinand et al., 2005](#)) was a 14 year follow up of 1580 young adults in the 'Zuid Holland' study. In this sample, cannabis use significantly predicted psychotic symptoms in participants who did not have psychotic symptoms before they began using cannabis (OR = 2.8). However, psychotic symptoms in those who had not used cannabis before the onset of psychotic symptoms also predicted future cannabis use (OR = 1.7). The German study ([Henquet et al., 2005](#)) followed 2437 young people aged 14 to 24 for four years between 1996 and 1999. Any cannabis use at baseline was reported to increase the risk of psychotic symptoms at the four year follow up in a dose–response fashion, regardless of confounders. The association was much stronger for those who had been identified as being prone to psychosis at baseline. Again, the relationship was significant even when the analysis was corrected for baseline use of other substances. Significantly, [Henquet et al. \(2005\)](#) report an increased risk of cannabis use in those who had displayed psychotic experiences 3 to 4 years earlier and who had not used cannabis before (OR = 1.4). The first New Zealand study ([Arseneault et al., 2002](#)) was a birth cohort based on 1037 people born in Dunedin, New Zealand between 1972 and 1973. Cannabis use at age 15 and 18 increased the risk of presenting with psychotic symptoms or schizophreniform disorder at age 26 (OR = 11.4 for those who had used cannabis before the age of 15). Like the other cohort studies, this relationship was independent of the use of other substances. Significantly, this study also assessed the presence of psychotic symptoms at age 11 and was therefore able to demonstrate that the observed association between cannabis use and increased risk of psychosis was independent from pre-existing psychotic symptoms. The second New Zealand study ([Fergusson et al., 2005](#)) followed 1011 individuals taking part in the Christchurch health and development study from birth. Assessments were conducted annually until age 16 and then again at 18, 21 and 25. Those who were daily users of cannabis had rates of psychotic symptoms that were between 2.3 and 3.3 times higher than the rates for those who did not use cannabis. The study conducted in Israel ([Weiser et al., 2002](#)) was a population-based cohort of 50413 adolescent males aged 16 to 17. Those who were later hospitalized for schizophrenia were more likely to have smoked cannabis at baseline than those who were not hospitalized (adjusted OR = 2.0).

A Greek study ([Stefanis et al., 2004](#)) conducted a cross sectional analysis of data from an existing cohort involving 3500 representative 19-year olds. Cannabis use was associated with both positive and negative dimensions of psychosis. First use of cannabis below 16 years of age was associated with a much stronger effect than first use after age 16 years, independent of lifetime frequency of use.

Research on non-clinical samples has shown a relationship between cannabis use and psychosis proneness or schizotypy (e.g. [Barkus, Stirling, Hopkins, & Lewis, 2006](#); [Dumas et al., 2002](#); [Verdoux et al., 2003](#); [Williams,](#)

Wellman, & Rawlins, 1996). Recently, Verdoux et al. (2003) used the experience sampling method (ESM) to assess the temporal relationship between cannabis use and psychotic experiences over a one week period. ESM is a diary technique which uses a signalling device, usually a watch, to alert participants to fill out self reports when an alarm sounds and provides a representative sample of moments in a person's daily life (De Vries, 1992). Participants with high psychosis vulnerability were more likely to report abnormal perceptions and thought influence when they used cannabis. Barkus et al. (2006) found that cannabis use per se was not related to schizotypy in their sample of healthy volunteers but that high scoring schizotypes were more likely to report psychosis-like experiences and unpleasant after-effects associated with cannabis. Kwapil et al. (1996) conducted a 10 year follow up of a high risk sample and reported psychosis proneness to be predictive of substance use. Not all studies involving psychosis prone people have reported an association between cannabis use and psychosis, however. A 12 month prospective study of high risk individuals (Phillips et al., 2002) did not find cannabis use at baseline to be associated with the onset of psychosis but there were low levels of substance use in their sample.

Although the evidence from the prospective cohort studies seems to suggest a causal link between cannabis use and psychosis we know that most people who smoke cannabis do not go on to develop schizophrenia and in countries where there has been a documented increase in rates of cannabis use in the general population (e.g. Australia) there has not been a corresponding increase in rates of schizophrenia (Degenhardt, Hall, & Lynskey, 2003). If the relationship between cannabis and psychosis is indeed causal but not all cannabis users go on to develop psychosis then we must consider the possibility that some individuals are more vulnerable to the effects of cannabis than others. Van Os et al. (2005) suggest a gene-environment interaction, with some individuals being genetically vulnerable to the effects of cannabis. Caspi et al. (2005) tested this hypothesis in a longitudinal birth cohort study and found that a functional polymorphism of the catechol-*O*-methyltransferase (COMT) gene moderated the influence of adolescent cannabis use on adult psychosis: carriers of the COMT valine<sup>158</sup> allele were more likely to experience psychotic symptoms after cannabis consumption than carriers of the COMT methionine allele. Recent experimental work also supports this link. Henquet et al. (2006) exposed patients with a psychotic disorder and their relatives to delta-9-tetrahydrocannabinol in a double-blind placebo controlled study and found that carriers of the valine allele were most sensitive to  $\Delta$ -9-THC induced psychotic experiences. Significantly, this finding was conditional on pre-existing psychosis liability.

### 5.1.2. Alcohol and psychosis

Studies have shown alcohol dependence to be predictive of psychotic experiences in the general population (e.g. Johns et al., 2004; Tien & Anthony, 1990) but not alcohol use per se. There is some evidence that patients with psychotic symptoms are more likely to abuse alcohol than those who do not have psychotic symptoms (e.g. Olfson et al., 2002) but it is generally accepted that although alcohol abuse may worsen the symptoms of those with schizophrenia and precipitate relapse it does not actually cause schizophrenia (Bernadt & Murray, 1986; Hambrecht & Hafner, 1996). Given that alcohol is reported to be the most commonly used substance by people with schizophrenia (Regier et al., 1990) this may limit the significance of models of drug-induced schizophrenia (Mueser et al., 1998).

### 5.1.3. Amphetamines and psychosis

The phenomenon of brief amphetamine-induced psychosis is well documented but the extent to which amphetamine use contributes to schizophrenia is not known. Baker et al. (2004) found a high rate of mental health problems among regular amphetamine users. More than a quarter (26.7%) of those with mental health problems were diagnosed with psychosis and the majority of these (71.4%) reported that they had received this diagnosis after commencing regular amphetamine use. Dawe, Saunders, Kavanagh, and Young (2005) reported that 20% of injecting methamphetamine users had had a psychiatric admission but that for 43% of these the admission was *prior* to the onset of regular amphetamine use. Chen et al. (2003) investigated 445 amphetamine users and found that amphetamine users with psychosis were younger when amphetamine use was first initiated and used larger amounts of amphetamines than those without psychosis. They conclude that pre-morbid schizotypal personality predisposes amphetamine users to psychosis. Curran, Byrappa, and McBride (2004) systematically reviewed 54 studies investigating stimulants and psychosis and found that a single dose of a stimulant drug could produce a brief increase in psychosis ratings in 50–70% of participants with schizophrenia and pre-existing acute psychotic symptoms. Those with schizophrenia who do not have acute psychotic symptoms respond, but less frequently (30%). Thus, individuals who are already experiencing psychosis are more likely to have a psychotic reaction to stimulants. However, there is little evidence to suggest that stimulant use results in chronic psychosis or schizophrenia.

#### 5.1.4. Cocaine and psychosis

As with amphetamines, a number of studies have reported that cocaine can induce psychotic symptoms in some users but comparatively little research has been conducted to date. Brady, Lydiard, Malcolm, and Ballenger (1991) interviewed 55 individuals consecutively admitted for treatment of cocaine dependence. Fifty-three percent (29/55) reported that they had experienced transient cocaine-induced psychosis.

Floyd, Boutros, Struve, Wolf, and Olivia (2006) assessed 51 cocaine dependent subjects and found that 36 (71%) had experienced psychotic symptoms during cocaine use. However, all participants in the study were polysubstance users.

#### 5.1.5. Opiates and psychosis

Research into the relationship between opiate use and psychosis is limited.

Studies have generally shown a low comorbidity rate between opiate use and psychosis (Brooner, King, Kidorf, Schmidt, & Bigelow, 1997; Dalmau, Bergman, & Brismar, 1999; Margolese et al., 2004; Schneier & Siris, 1987) and there is evidence to suggest that heroin users may actually be at *lower* risk of psychosis than users of other substances (Farrell et al., 2002). Thus the available data do not appear to support the hypothesis that opiate use causes schizophrenia.

It is clear that large numbers of patients presenting to mental health services for the first time are already using substances. The evidence from prospective longitudinal studies suggests that for some patients at least, cannabis can have a causal role in the development of psychopathology. However, there is little evidence to suggest that other substances, including alcohol, are a causative factor in psychosis.

### 5.2. Does psychosis cause substance use?

The most well known model which states that substance use disorder is a consequence of psychiatric problems is the self-medication hypothesis (Khantzian, 1985, 1997) and it is this model that has received the most attention in the research literature, perhaps because of its intuitive appeal. The model proposes that substance abuse is an attempt to self-medicate psychiatric symptoms. It assumes that people with mental health problems use substances to reduce their symptoms and that problematic use develops as a result. The hypothesis suggests that substances are not chosen at random. Rather, there is selective matching of specific substances with specific symptoms. As Khantzian (1985) states: “*The drugs that addicts select are not chosen randomly. Their drug of choice is the result of an interaction between the psychopharmacologic action of the drug and the dominant painful feelings with which they struggle*” (p. 1259). The theory applies not just to the positive symptoms of severe mental illness (i.e. hallucinations and delusions) but also the negative symptoms. Variants of the self-medication model postulate that drugs and alcohol are also used to self-medicate extrapyramidal symptoms caused by neuroleptic medication (Schneier & Siris, 1987) or to alleviate dysphoria. Dixon et al. (1991) go as far as to suggest that dysphoria might actually be the common factor underpinning increased comorbidity. “*Perhaps only those patients whose symptoms (positive, negative or extrapyramidal) lead to distress or depression are the ones who abuse drugs*” (Dixon et al., 1991, p. 75).

According to Mueser et al. (1998) three types of evidence would provide support for the self medication hypothesis: (1) if epidemiological studies suggested that clients with particular psychiatric diagnoses were more prone to abusing specific types of substances, (2) if psychiatric clients with more severe symptoms were more likely than less symptomatic clients to abuse substances and (3) if clients with dual disorders described beneficial effects of substance use on symptoms.

Empirical data do not suggest a consistent relationship between substance use and specific diagnoses. The review by Schneier and Siris (1987), for example, reported that patients with schizophrenia prefer drugs which counteract negative symptoms (e.g. cocaine, amphetamines and cannabis) to those which have predominantly sedative effects (e.g. opiates and alcohol) and that people with schizophrenia were more likely to use stimulants than those with different diagnoses. Similarly, Dixon et al. (1989) found that patients with schizophrenia preferred activating drugs (e.g. cocaine, cannabis, stimulants and hallucinogens) whereas bipolar patients preferred sedative/hypnotics and alcohol. In contrast, Regier et al. (1990) and Mueser et al. (1992) found that the patterns of use observed by people with schizophrenia is similar to that found in patients with other diagnoses. Mueser et al. (1992) suggest that it is the availability of different types of substances rather than their subjective effects that determine which substances are abused.

A number of studies have attempted to assess the link between severity of symptoms and levels of substance use but again, the evidence to date has been contradictory. Brunette, Mueser, Xie, and Drake (1997) and Dervaux et al. (2001)

found no relationships between severity of symptoms and substance abuse whereas Pencer and Addington (2003) reported that substance use was associated with more severe positive symptoms. Recently, Talamo et al. (2006) tested the hypothesis that comorbid patients had more positive versus negative symptoms than non-comorbid patients by conducting a meta analysis of 8 previously published cross sectional studies ( $n=725$ ) and found that comorbid patients had significantly higher positive symptom scores and significantly lower negative symptoms scores (assessed using the Positive and negative symptom scale, PANSS, Kay, Fiszbein, & Opler, 1987). Chapman, Labhart, and Schroeder, (1996) found that alcohol users had a higher PANSS composite score: those who were currently abusing alcohol had an overall greater severity of positive relative to negative symptoms. Scheller-Gilkey, Moynes, Cooper, Kant, and Miller (2004) compared the PANSS scores of schizophrenia patients with a history of substance against those with no such history and found no differences in either the positive or negative symptom scales. However, patients with a history of substance abuse had significantly higher general psychopathology scale scores. Comorbid patients displayed more somatic concern, guilt feelings, depression and poorer impulse control.

### 5.2.1. *The self report literature*

We reviewed the literature to identify articles containing self reported reasons for substance use. Studies for review were identified following a search for combinations of the key words schizophrenia, psychosis, dual diagnosis, comorbidity, drug use, drug abuse, substance use, substance abuse, alcohol use, alcohol abuse in two main abstract databases: PsycINFO (the American Psychological Association's abstract database) and PubMed (published by the U.S. National Library of Medicine). In addition, the bibliographies of articles were examined in order to identify further citations. English language studies that asked patients with psychosis to report their *current* reasons for substance use were included. Thirteen studies were identified. Two of these summarized their findings without reporting the numbers of patients endorsing each reason for use and were therefore excluded.

Table 1 contains the remaining eleven studies. Reasons for use were grouped into five main categories: intoxication effects, social reasons, dysphoria relief, psychotic symptoms and medication side effects. As the table shows, there is considerable variability between studies. Between 35 and 95% endorsed the intoxicating effects of drugs and alcohol as a reason for their consumption ('to get high', 'for the buzz', 'to feel good'). Between 8 and 81% endorsed social reasons ('to get on with others better', 'to fit in with the crowd'). Between 2 and 86% reporting using drugs and/or alcohol to relieve dysphoria (feelings of depression, anxiety, depression and other negative emotional states). Between 0 and 42% used drugs and/or alcohol to either alleviate or cope with the symptoms of psychosis (hallucinations, feelings of suspicion and paranoia) and between 0 and 48% reported using substances to reduce or cope with medication side effects. A range of 'enhancement' reasons for use were also endorsed in five of the studies (Addington & Duchak, 1997; Dixon et al., 1991; Goswami, Mattoo, Basu, & Singh, 2004; Gregg, Haddock, & Barrowclough, submitted for publication; Warner et al., 1994). These studies reported that patients use drugs and or alcohol to 'increase pleasure' (62–95%); 'to feel more energetic' (24–56%); 'to increase emotions' (13–49%), 'to talk more' (18–61%) and to improve concentration (13–33%).

Some of the apparent variability in reported reasons for use will be attributable to differences in sampling and in methodology. Not all patients included in these studies actually met the criteria for a substance use disorder; some were merely substance 'users'. Some studies required patients to select their reasons for use from predetermined lists whilst others used free response or open ended questions. Some requested patients to list all of the reasons they used substances for whilst others requested only the 'main' reason. Significantly, none of the studies outlined employed self report methods with known validity and reliability for this patient group. Additionally, these studies have failed to examine reasons for use in the context of the known demographic risk factors such as age and gender. Do males report different reasons for use to females? Are younger patients with schizophrenia more likely to report certain reasons for substance use? What other factors (both illness-related and demographic) influence self reported reasons for use? Nevertheless, despite their shortcomings, the evidence from these self report studies provides some support for the self medication hypothesis, in particular the 'alleviation of dysphoria' version of the hypothesis. In the majority of studies, the most frequently endorsed reasons for use are from this category. Interestingly, some of the studies report that for some patients, their stated reasons for substance use and their outcome expectancies for the effects of that substance are incongruous with the actual achieved effect. For example, in Addington and Duchak's (1997) study, participants reported using drugs to increase pleasure, to get high and to reduce depression. However, subjective effects of increased depression and positive symptoms were also reported. Some patients may report using drugs and alcohol to make them feel better yet report feeling worse afterwards.



Table 1  
Self reported reasons for use by substance users with psychosis

Authors	Sample	Methodology	Reasons for use			
			<i>Intoxication effects (to get high, to feel good)</i>			
Test, Wallisch, Allness, and Ripp (1989)	27 drug and/or alcohol users with schizophrenia or schizoaffective disorder	Free response and inspection of a list	–			
Dixon et al. (1991)	53 drug and/or alcohol users with schizophrenia, schizoaffective or schizophreniform disorder	Questionnaire ('Stated reasons scale, SRS' developed for the study)	72%			
Warner et al. (1994)	55 drug and/or alcohol users with schizophrenia, schizoaffective or bipolar disorder	Interview (adapted from Test et al., 1989)	–			
Addington and Duchak (1997)	41 drug and/or alcohol users with schizophrenia	Questionnaire (SRS, Dixon et al., 1991)	<i>Alcohol</i> (74%) <i>Cannabis</i> (95%)			
Fowler, Carr, Carter, and Lewin (1998)	194 drug and/or alcohol users with schizophrenia	Interview.	<i>Alcohol</i> – <i>Cannabis</i> (41%) <i>Amphetamine</i> (79%)			
Gearon, Bellack, Rachbeisel, and Dixon (2001)	25 drug and alcohol users with schizophrenia or schizoaffective disorder	Questionnaire (Inventory of Drug Taking Situations (Annis, Turner, & Sklar, 1997); 'Self medication questionnaire' developed for the study)	–			
Baker et al. (2002)	160 drug and/or alcohol using psychiatric inpatients	Interview.	<i>Alcohol</i> (35.2%) <i>Cannabis</i> (56.9%) <i>Amphetamine</i> (44%)			
Goswami et al. (2004)	22 male drug and alcohol users with schizophrenia	Questionnaire modified SRS (Dixon et al., 1991)	81%			
Green et al. (2004)	45 male cannabis users with psychosis	Telephone interview	–			
Schofield et al. (2006)	49 cannabis users with psychosis	Questionnaire (SRS, Dixon et al., 1991)	–			
Gregg et al. (submitted for publication)	45 drug and alcohol users with schizophrenia or schizoaffective disorder	Q methodology	51.1%			
Authors	Reasons for use					
	<i>Social reasons (to facilitate social interaction/to fit in)</i>	<i>Dysphoria relief (to relieve anxiety/depression/boredom; to relax)</i>	<i>Psychotic symptoms (to alleviate/cope with hallucinations/feelings of suspicion/paranoia)</i>	<i>Medication side effects</i>		
Test et al. (1989)	44.4%	44.4–63%	7.4%	18.5%		
Dixon et al. (1991)	55%	64–72%	4–11%	15%		
Warner et al. (1994)	72.7%	47.3–61.8%	10.9%	72.7%		
Addington and Duchak (1997)	<i>Alcohol</i> 56% <i>Cannabis</i> 71%	<i>Alcohol</i> 71–82% <i>Cannabis</i> 81%	<i>Alcohol</i> 24–29% <i>Cannabis</i> 19–40%	<i>Alcohol</i> 24% <i>Cannabis</i> 38%		
Fowler et al. (1998)	<i>Alcohol</i> 58% <i>Cannabis</i> 58% <i>Amphetamine</i> –	<i>Alcohol</i> 58% <i>Cannabis</i> 62% <i>Amphetamine</i> 47%	0–9%*			
Gearon et al. (2001)	48–68%	28–56%	36%	48%		
Baker et al. (2002)	<i>Alcohol</i> 14.3% <i>Cannabis</i> 8.8% <i>Amphetamine</i> 8%	<i>Alcohol</i> 47.3% <i>Cannabis</i> 19.6% <i>Amphetamine</i> 36%	0–2.2%*			
Goswami et al. (2004)	23%	35–54%	42%	12%		
Green et al. (2004)	37.8%	2.2–26.7%	0%	0%		
Schofield et al. (2006)	81%	49–86%	8–11%	15%		
Gregg et al. (submitted for publication)	71.1%	60–77.8%	26.7–31.1%	4.5%		

\* These studies combined illness related and medication related reasons for substance use.

### 5.2.2. *Are some people with schizophrenia 'supersensitive' to the effects of substances?*

An alternative secondary substance use model is the supersensitivity model (Mueser et al., 1998) which hypothesizes that certain individuals with schizophrenia have biological and psychological vulnerabilities that are a result of genetic and early environmental effects in their lives: these vulnerabilities can interact with stressful life events to cause psychiatric disorder. Substance use is believed to increase this vulnerability so that people with schizophrenia are more likely to experience negative consequences as a result of substance use than people in the general population. Furthermore, these negative consequences result from lower levels of use than those needed by the general population (Drake et al., 1989; Drake & Wallach, 1993). In short, these individuals are “supersensitive” to the effects of certain substances. According to the hypothesis, dually diagnosed individuals should be more likely to be diagnosed with a substance abuse as opposed to a substance dependence diagnosis and experience greater negative consequences associated with lower levels of use when compared to substance users without schizophrenia. The first empirical test of the hypothesis has recently been conducted. Gonzalez, Bradizza, Vincent, Stasiewicz, and Paas (in press) compared 42 individuals with substance use disorder (SUD) to 53 dually diagnosed individuals (DD). Although the DD group had significantly greater levels of psychological symptoms they did not experience greater negative consequences. Rates of substance use were comparable between the two groups and the DD group had higher proportions of individuals meeting substance use dependence criteria.

### 5.3. *Do substance use and psychosis share a common origin?*

Common factor models propose that substance use and schizophrenia share a common origin. These common factors could be biological, individual or social. There is a good deal of evidence to suggest that genetic factors independently contribute to schizophrenia (e.g. Gottesman & Shields, 1976) and to substance use disorder (e.g. Rhee et al., 2003; Tsuang, Bar, Harley, & Lyons, 2001) although it is not clear which genes are involved and how genetic predisposition is transmitted. The extent to which the two disorders share a common genetic vulnerability, however, is unknown. The main method of assessing the role of genetic factors in the co-development of schizophrenia and substance use disorder has been to examine family history but the studies that have been conducted to date have been conflicting. For such a link to be supported studies would be expected to find that patients with schizophrenia have more relatives with substance use disorders than people in the general population or that people with substance use disorder would be more likely to have family members with schizophrenia. Whilst some studies have reported that dually diagnosed patients are more likely to have family members with substance use disorders than patients with schizophrenia alone (e.g. Noordsy, Drake, Biesanz, & McHugo, 1994) other studies have not found this to be the case (Gershon et al., 1988). It is possible that genetic vulnerability may contribute to the development of comorbid substance use in some patients but the available evidence does not appear to support the idea that increased comorbidity is a result of a common genetic basis for both disorders.

Another common factor that could potentially influence both substance use and schizophrenia is neuropathology i.e. that the neuropathology of schizophrenia impacts on the neural circuitry mediating drug reward and reinforcement resulting in an increased vulnerability to addictive behaviour. Put simply, patients with schizophrenia might be biologically vulnerable to the rewarding effects of drug abuse. The dopamine opioid neurotransmission systems have been implicated. In these models schizophrenia and substance use are thought to be independent manifestations of the same disease. (see Chambers et al., 2001 for a review).

These results may actually imply a common underlying vulnerability for both disorders in which the pathology of the cannabinoid system in schizophrenia patients is associated with both increased rates of cannabis use and increased risk for schizophrenia (Weiser & Noy, 2005). Further research is needed to determine the relevant underlying neuropathological processes before firm conclusions can be drawn.

Social and environmental factors that could potentially underpin both disorders have also been hypothesized, for example family dysfunction (Fergusson, Horwood, & Lynskey, 1994) and economic and social disadvantage. Another possible mechanism is traumatic early childhood experience. We know that members of the general population who report physical or sexual abuse in childhood are more likely to abuse substances in adulthood (Kessler, Davis, & Kendler, 1997) and that for some, childhood abuse can also contribute to psychosis (Briere, Woo, McRae, Foltz, & Sitzman, 1997). Scheller-Gilkey et al. (2004) compared 70 patients with schizophrenia and a history of substance abuse with 52 patients without a history of substance abuse and found that the former had significantly higher scores on a measure of childhood traumatic events and on a PTSD scale. The available evidence suggests that the relationship may

be bidirectional: PTSD precedes the onset of substance use in some people with schizophrenia but may also put people with schizophrenia at increased risk of subsequent retraumatisation. Mueser et al. (1998) present evidence suggesting that ASPD and its childhood correlate conduct disorder might be a common factor. Studies have shown that patients with schizophrenia and antisocial personality disorder (ASPD) are more likely to have comorbid substance use disorder than patients without ASPD (Caton et al., 1994; Mueser et al., 2000) and for patients with schizophrenia and substance use disorder ASPD is associated with a more severe course of substance use disorder including earlier age of onset and larger quantities of substance use (Mueser, Drake et al., 1997).

Impairments in cognitive functioning have also been hypothesized to have an impact (Tracy, Josiassen, & Bellack, 1995), as have poorer coping skills, lower educational attainment, lower socioeconomic status, poor interpersonal and social problem solving skills. It must be noted that it is unlikely that any of these cognitive and social risk factors operate independently to increase rates of comorbidity but their cumulative effects might. Few multiple risk factor models have been proposed but the cross sectional literature does seem to suggest that some of these factors may play a part.

#### 5.4. Do psychosis and substance use interact and maintain each other?

Bidirectional models propose that psychosis and substance use problems may both trigger and maintain each other. For example, substance use may serve as a stressor precipitating onset of schizophrenia in vulnerable individuals and mental health problems are then subsequently maintained by continued substance use due to socially learned cognitive factors such as beliefs, expectancies and motives for substance use (Mueser et al., 1998). Thus bidirectional models tend to involve multiple risk factors but although a variety of different models have been proposed there have been no empirical investigations.

##### 5.4.1. Multiple risk factor models

Blanchard et al. (2000) proposed an affect regulation model of substance which in common with the self medication literature suggests that patients with schizophrenia use drugs and alcohol to cope with negative emotions and problems. The proposed model emphasizes the role of enduring personality characteristics stating that stable personality traits, stress and coping are the factors underlying long term risk for substance use. Aspects of this model find some support in the literature: the self report literature shows that people with schizophrenia report using substances to regulate negative affects such as dysphoria and anxiety and a handful of empirical studies have shown that substance use is related to personality components in patients with schizophrenia (e.g. Blanchard et al., 1999; Dervaux et al., 2001; Kwapil, 1996).

Barrowclough et al. (2007) propose a model of substance use maintenance in psychosis which incorporates the key features of Marlatt and Gordon's social-cognitive model of addiction (Marlatt & Gordon, 1985). The model proposes that certain situations and cues trigger drug or alcohol related thoughts which in the absence of alternative coping strategies and in the context of low self efficacy for resisting use and positive expectancies from use make the person vulnerable and more likely to use substances. This interaction between situations and cognitive/emotional reactions becomes the basis of a repeated cycle which maintains drug or alcohol use. As the self report literature outlined above shows, people with schizophrenia indicate that situations and cues triggering use may be related to psychotic symptoms and to the negative consequences of the disorder, particularly dysphoria and distress.

Studies have shown that people with schizophrenia often experience difficulty in coping with stresses (Corrigan & Toomey, 1995; Mueser, Drake et al., 1997; Mueser, Valentiner, & Agresta, 1997) and that they may possess a relatively limited repertoire of coping strategies (Rollins, Bond, & Lysaker, 1999). A number of studies have investigated coping in relation to psychotic symptoms (e.g. Falloon & Talbot, 1981; Kinney, 1999; Lobban, Barrowclough, & Jones, 2004), affective symptoms (e.g. Brier & Strauss, 1983) and negative symptoms (e.g. Mueser, Drake et al., 1997; Rollins et al., 1999) in patients with schizophrenia. Such studies have highlighted that individuals with schizophrenia use a diverse range of strategies to influence symptoms. People with schizophrenia use mainly avoidant coping strategies and tend to have a greater array of coping strategies for positive symptoms than for negative symptoms (Rollins et al., 1999). From the perspective of social learning theory (e.g. Bandura, 1977) both drug and alcohol use are seen as habitual maladaptive coping responses employed by people who hold positive beliefs about the effects of that substance, coupled with inefficient coping resources (Abrams & Niaura 1987). According to this perspective, deficiencies in general coping skills and positive expectancies about the effects of drug and alcohol operate independently and jointly to contribute to the use of drugs or alcohol as a coping mechanism. Substance use could be viewed as a general coping mechanism invoked in situations where more appropriate coping responses are either unused or not available. Social

learning theory assumes that the coping functions of a substance are learned through initial exposure to that substance and in subsequent use in different situations, hence the salience of particular functions should show considerable variation across individuals (Wills & Hirky, 1996).

## 6. Summary and conclusions

We have presented a review of the main models that have been proposed to explain the etiological relationship between substance use and psychosis. Although these four models have no doubt served to clarify our understanding of the reasons for substance use by people with schizophrenia, it is clear that no single model is able to adequately explain all comorbidity. The hypothesis that substance use causes schizophrenia is not supported sufficiently or consistently. Evidence from recent prospective cohort studies suggests that cannabis can have a causal role in the development of psychopathology and studies involving psychosis prone individuals indicate that cannabis use might precipitate psychosis among vulnerable individuals but there is little evidence to suggest that other substances, including alcohol, are a causative factor in psychosis. The literature provides little support for the self-medication hypothesis in its original formulation i.e. that specific substances are chosen for their specific pharmacological properties. The self report studies do show that some people with schizophrenia report using substances in an attempt to alleviate specific psychopathological symptoms or medication side effects but there has been little research to show whether substances are selected differentially. There is greater support for an ‘alleviation of dysphoria’ model of comorbidity than Khantzian’s (1985) original self medication formulation: patients report using substances to alleviate or to cope with unpleasant affective states (e.g. boredom, depression, anxiety and loneliness). Common factor models have implicated both genetics and neuropathology but no common gene has yet been identified and the neurobiological evidence is not consistent. Additional research is required. It may even be the case that some other as yet unresearched variable or variables may account for the relationship between substance use and schizophrenia.

It is likely that there are multiple risk factors involved in substance use in psychosis. We know that a number of demographic factors (such as age, gender and socioeconomic status) and contextual factors (such as family history of substance use) predict substance use in schizophrenia. Social networks, quality of living environment, poverty and stressful life events influence substance use as do individual differences in personality, coping, interpersonal skills and social functioning but the extent to which these explain increased comorbidity is not known: there are few well developed multiple risk factors models. More work to develop and test multiple risk factor models is required.

Bidirectional models integrating aspects of the different causative models outlined in this review suggest that separate factors may be responsible for the initiation and maintenance of substance use by people with schizophrenia. It is possible, for example, that substance users whose drug use precipitated or caused their schizophrenia (perhaps because of biological vulnerability) may continue using cannabis in order to alleviate or cope with the symptoms of schizophrenia better. As yet, however, there have been no empirical investigations of bidirectional models. Longitudinal prospective cohort studies would be ideally placed to identify the factors related to the development and maintenance of substance use and psychosis and the factors which mediate and moderate the paths between the two. Such studies would also allow for the causal hypotheses to be tested. The existing longitudinal research is constrained by a failure to assess the predictive impact of substances other than cannabis on psychosis and has largely ignored the relationships between current symptomatology, substance use and other potential risk factors.

The dually diagnosed population is a heterogeneous group and as Mueser et al. (1998) suggest, it is likely that different models may account for comorbidity in different groups of people and multiple models may apply for some individuals. The challenge now it is to identify which models apply to which people if we are to be able to develop more effective treatments.

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