
Faculty of Addictions Psychiatry essay prize

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Introduction:

During the mid-twentieth century several clinicians alluded to the concept of 'self-medication' as an integral process in the development and maintenance of drug dependence. Contrary to Freud’s focus on the hedonic effects of drugs, Fenichel (1946) and Rado (1957) observed that addicts seemed to have underlying depression, of varying degree, which was deemed to be the motivation for drug use.

Khantzian collated these concepts and proposed that the key drive for the development of drug dependence is based on psychological suffering, where the specific psychological disturbances of an individual leave a need for a specific substance, to palliate this pain.

Perhaps the greatest strength of this hypothesis is that it offers an explanation for why only a minority of individuals who use drugs become dependent, whereby the need to master and convert the passive and confusing experience of psychological problems is the missing link. Such problems include alexithymia, the inability to put feelings into words; resulting in a compromised ability to process emotions and thus regulate behaviour that is driven by such emotions.

Khantzian et al. (2014) acknowledged that the two main arguments posed against the SMH are: (1) not all who suffer with pain and distress become drug-dependent and (2) there is as much, if not more, suffering as a consequence of drug use. He subsequently proposed that, in light of these questions and issues raised, it was necessary to adopt a ‘superordinate or more overarching paradigm of the addictions as a self-regulation disorder’.

(1) and (2) above contribute to concerns that the direction of causality is not clear-cut, and this shall be analysed through empirical assessment of the temporal acquisition of substance use and other psychological vulnerabilities. Further to this, a comprehensive review of literature will be conducted to analyse evidence surrounding concepts such as the ‘drug of choice’ phenomenon and the potential need for the incorporation of biological evidence to this framework to generate a truly biopsychosocial appraisal of substance dependence.
Edward Khantzian’s Self-Medication Hypothesis of substance-use disorders.

Khantzian only constructed Self-Medication (SM) into a coherent hypothesis in 1975. He initially proposed that inadequate ego-defence mechanisms cause an individual to find an external means to control aggressive impulses and that heroin use may successfully do this. He further argued that because heroin is able to suppress these impulses, methadone treatment might actually be doing more than preventing physical withdrawal, as through its psychotropic similarities to heroin, it may be able to prevent the dependence-causing dysphoria (Khantzian, 1975)

This hypothesis of heroin addiction was eventually developed to include cocaine addiction (Khantzian, 1985). Similarly, Khantzian believed that cocaine had its appeal because its use allowed an escape from disturbances one may experience from hypomania, hyperactivity or depression. Alcoholism was subsequently incorporated into the hypothesis with the idea that alcohol use facilitates access to feelings and relationships to those who feel cut-off from them (Khantzian, 1990). This eventually progressed into a fully developed, more comprehensive theory (Khantzian 1997) that included all drugs of addiction.

This hypothesis proposes that due to arrested emotional or psychosexual development, one may be subject to overwhelming emotions, or alexithymia which form a drive to use a drug which can provide one with a desired or ‘normal’ affect state, or at least give one control over the affect one is experiencing. One may argue that many individuals experience discomfort of this nature but do not abuse drugs, which leads to a further proposal that this affect dysregulation ‘malignantly combines’ with an impaired capacity for self-care (survivability).

These self-care problems are believed to derive from developmental deficiencies that interfere with the ability to predict and protect against harm. Consequently, this aforementioned combination makes ‘experimentation with, dependence on and relapse to substances more likely and compelling’ (Khantzian, 2007).

It is believed that it is more than just the ego strengthening effects of drug use that leads to dependence. Other contributing factors include the progressive effect of promoting stable functioning along with the regressive effect of perpetuating the existing vulnerability, which enforce continued use.

The ‘drug of choice’ concept, coined by Weider and Kaplan is important in Khantzian’s hypothesis, as he believes the substance one becomes dependent on is by no means random. ‘Preferential drug use’ (Milkman and Frosch, 1973) is proposed by the SMH to be the result of an interaction between the individual’s primary affect state, the psychopharmacological profile of the drug,
an individual’s personality and the availability of the substance (Khantzian and Albenese, 2008).

The SMH now considers three drug groups to have a distinct psychopharmacological profile. These are opioids, Central Nervous System (CNS) depressants (alcohol, benzodiazepines, and barbiturates) and CNS stimulants (cocaine and amphetamine). The proposed profiles of these drugs and their corresponding abuser are summarised below:

**Opioids:** Opioids are thought to have ‘calming and normalizing effects’ (Khantzian, 1997). The SMH proposes that they function as a means to temporarily mute or attenuate rage that is believed to be associated with a traumatic background of abuse, loss or painful disappointment (Khantzian, 1985; Khantzian and Albenese, 2008).

**CNS Depressants:** The hypothesis proposes that alcohol abusers cut themselves off from awareness of emotions that may be potentially distressing by employing rigid defences that can result in disaffected states. Among the key psychological features of alcohol are its relaxing and sedating effects (Dodgen and Shea, 2000). These effects are thought to relieve emotional tension as they allow defensive structures to be softened (Khantzian, 1997). Benzodiazepines, now much more commonly used than barbiturates are thought to follow a similar course to alcohol but in pill form (Winger et al, 2004).

**CNS Stimulants:** The psychological effect of cocaine in the short-term is elevated self-esteem, confidence, mood and energy state (Dodgen and Shea, 2000). This is due to the increased dopaminergic and noradrenergic activity secondary to decreased vesicular reuptake and transport of these monoamines (Winger et al, 2004). The SMH proposes that those drawn to use cocaine will be either ‘high energy’ or ‘low energy’ individuals. The former will have an increased need for elated sensations (Khantzian, 1985; Khantzian et al. 1990) whilst the latter, who likely mirror a depressive state, will use cocaine to escape anhedonia (Khantzian and Albenese, 2008).
Do psychological disturbances drive one to self-medicate or are they caused by substance abuse?

Whilst the Epidemiological Catchment Area (Arbor et al., 1994) and clinical studies have managed to indicate a significant comorbidity between anxiety (and affective disorders) and substance-use disorders, it has been argued as unclear whether the affective disorder is a causal agent or a by-product of the substance abuse.

In cases of comorbidity, it is vital to consider Berkson’s paradox (Westreich, 2012), a phenomenon relevant to retrospective studies that examines the risk factors of a particular disease. It proposes that disorders which appear to be related may indeed just be coexisting due to an ascertainment bias in a study design, whereby the sample is on average more severely ill than normal and thus happen to have multiple disorders.

Frances (1997) also highlighted this problem with the self-medication hypothesis and its supporting research by warning it is not clear cut which came first between the substance abuse and the psychiatric symptoms. Given that the nature of much of the evidence requires patient self-report, there is potential for an overly reductionist interpretation and consequently confusion around causality.

In light of this, Frances suggests that it can be clinically useful to determine the temporal onset of each disorder, how the two disorders interact and the mechanisms through which each may perpetuate the other. They conclude that it is ‘the wrath of grapes that leads to the greater part of suffering in substance use disorders’ (Frances, 1997).

Conversely, one may argue that alcohol abuse is of greater significance in the development of psychopathology (e.g. anxiety) than psychopathology is in the development of alcoholism. This is further corroborated by Schuckit and colleagues (1990) who emphasised that anxiety and depressive symptoms are more likely to be caused by alcohol abuse than be its antecedent. However, it must be noted that these participants were a sample of primary alcoholics, which confounds their assertion.

Dackis and Gold (1986) propose an explanation for why this may be the case. They believe that the depressive symptoms associated with drug abuse are the result of abstinence symptoms as the depletion of dopamine stores results in ‘craving’ for dopamine and further cocaine use. This would then become a vicious cycle and lead to pathological dopamine depletion and, in keeping with the monoamine hypothesis, the presentation of anhedonia - a key symptom of depression.

Work by Rounsaville et al. (1982) may help to settle this disagreement as they argue that even when substance use seems to cause depression, the possibility
remains that prior to the dependence the patient experienced sub-threshold levels of depressive symptoms. This concept fits the self-medication hypothesis quite well, as in his later work Khantzian asserts that it is not psychiatric disorders that one medicates but a wider range of affect dysfunction.

This is further supported by the work of Kellam et al. (1991) who studied young males and documented that childhood aggression and maladaptive social behaviours, were the main antecedents of later drug use. In this example, the antisocial behaviour and aggression represent a probable ‘sub-threshold’ disaffection that the participants may be medicating. This substance use may lead to a subsequent psychopathology, similar to that observed by Schuckit.

Silver and Abboud (1994) conducted an investigation of the relationship between the onset of drug abuse and the onset of psychiatric illness (in this case, schizophrenia). The forty-two participants were selected from Flugaman psychiatric hospital when patients who identified as drug abusers experienced their first hospitalisation due to overwhelming schizophrenic symptoms. The patients underwent a semi-structured interview consisting of a 140-part questionnaire, where particular attention was paid to the onset of drug use and the use of specific substances. The researchers noted that drug abuse was a very socially sensitive issue and thus incorporated redundant and cross-referenced questions in the questionnaire, in an attempt to remove any potential bias.

Their study found that over 60% of participants used drugs before their hospitalisation and they argue that this supports the SMH. Given that a ‘schizophrenic-break’ severe enough to warrant hospitalisation would be preceded by a progressive development of symptoms, it stands to reason that at the time when substance dependence began, the participants would have been experiencing alexithymia. Whilst this study supports the SMH, more clear-cut evidence is required and with a larger sample than used here because one so small (n=42) is not generalisable.

Abraham and Fava (1999) improved on the above weaknesses and sought to elucidate an accurate sequence of onset of pathology in depressed drug users. In order to do this they conducted a retrospective, blinded case-controlled assessment of the drug and depressive history of depressed patients. 375 patients were chosen and their diagnosis with major depressive disorder was confirmed using the Structural Clinical Interview for DSM-III-R (SCID), a scale used for comorbid drug dependence. They found that the patients with alcohol dependence experienced their substance use problems ~4.7 years subsequent to the first experience of depression. Furthermore, they showed that in polydrug dependent patients, alcohol dependence occurred around 4.5 years after their first episode of major depression, and cocaine dependence was evident 6.8 years after this first episode.

This evidence indicates that disaffect predates drug dependence. The comorbidity of depression and cocaine dependence is also in keeping with the
aforementioned notion that cocaine-dependent individuals are medicating a state of depression.

Robinson et al. (2009) found further evidence supporting the SMH through assessing comorbidity of substance dependence and anxiety disorders. They used the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) to sample patients and had an 81% response rate of substance dependent individuals. They then used the Alcohol Use Disorders and Associated Disabilities Interview Schedule IV (AUDADIS-IV) to make Axis I and II diagnoses such as affect, anxiety or personality disorders. Once diagnoses were confirmed, they asked questions such as “did you ever drink alcohol to keep from having panic attacks?”. The results they collected provided evidence of self-medication across all types of anxiety disorders and that the anxiety preceded the substance use, in line with the SMH.

Harris and Edlund (2005) provide further corroboration in an investigation into the association between past 30-day alcohol and drug use and past year unmet need for use of mental health care. Their sample came from the Substance Abuse and Mental Health Services Administration (SAMHSA), which conducts an annual survey to determine the prevalence of illicit drug, alcohol and tobacco use in the United States. A quarter the respondents between age eighteen and sixty-five reported at least one mental health symptom. Of this subset, 29.9% of the symptomatic respondents reported psychological distress severe enough to be considered seriously mentally ill. Furthermore, 17.7% of this subset reported unmet need for mental health care. Even when the substance dependent subgroup was excluded, it was found that those with mental health problems were more likely to use illicit drugs, the rate of which increased with the severity of mental illness. Overall, the study found that substance use in the preceding 30 days increased if the participant had a significant unmet need for healthcare in the previous year. This indicates that an increase in substance use is followed by the unmet need for healthcare and in turn an increase in mental distress.

It is of note that Katerndahl and Realini (1999) found evidence that previous drug use (e.g. cocaine) may cause panic disorders, which contradicts Khantzian’s idea that it is developmental problems that cause the affective disorders that one medicates. However, the findings of Thorberg and Lyvers (2005) suggest that substance users were still deficient in regulating their emotions after an extended period of abstinence, indicating that self-regulation problems and negative affect states precede drug dependence.

Whilst the evidence above does indicate that psychopathology precedes substance use, it is worth noting that it is likely not the only factor. For example, socioeconomic status and availability of drugs will logically have a significant impact on substance abuse and as such it is difficult to determine to what extent disaffected states are contributing to development and maintenance of drug dependence.
Empirical investigations of the motive for substance use and the concept of a ‘drug of choice’.

In the analysis of Khantzian’s Self-Medication Hypothesis (SMH), it is imperative to determine whether drug use can indeed medicate psychiatric symptoms as suggested and if this occurs in a manner in keeping with the ‘drug of choice’ phenomenon.

A study by Richard Castaneda (1994) contested the notion of drug of choice. He used a sample of 83 male subjects who, when hospitalised for unmanageable psychiatric problems had a then-confirmed diagnosis of both an Axis I and Axis II disorder, which constitute acute disorders in need of treatment (e.g. depression) and personality/intellectual disabilities respectively. The patients’ psychiatric symptoms were assessed using the Hopkins Symptom Checklist-Revised (HSCL-90-R), a self-report symptom inventory with nine symptom dimensions. They were asked to disclose how their ‘drug of choice’ had affected their symptoms in the two weeks preceding admission to hospital. They found that heroin addicts reported improvement on four of the nine dimensions, while; conversely, alcoholics and cocaine addicts reported an alcohol- and cocaine-induced aggravation of all nine symptom-dimensions.

These findings suggest that only heroin follows the prediction of the SMH and that symptom improvement depends on one’s drug of choice and not their symptom profile, constituting evidence against the SMH. However, none of the symptom dimensions were more prominent among heroin addicts than the other groups, indicating that even heroin users had not specifically selected heroin to relieve their symptoms.

In agreement with Castaneda (1994), a study by Arendt et al. (2007) also suggests that substance use is not used as a means of self-medication. Using a sample of 119 subjects, all aged thirty or below, from 19 different treatment centres in Denmark, they investigated the reason for cannabis dependence. All subjects had to meet criteria for cannabis dependence based on the International Classification of Diseases-10, which was confirmed using the Composite International Diagnostic Interview. Furthermore, their comorbidity was ascertained using the Schedules for Clinical Assessment in Neuropsychiatry (1992) and corroborated by the Beck Depression Inventory II, validating the presence of affective symptoms. The investigation showed no evidence of specific purpose to their cannabis abuse and therefore related to SM, especially when there was some evidence that it actually increased adverse symptoms.

Further to the question of drug specificity raised by Castaneda (1994), Schinka et al. (1994) used the Personality Assessment Inventory to examine group differences in symptomatology. 238 patients took part in four groups based on their addiction: alcohol dependence only, cocaine dependence only, alcohol and cocaine dependence or polysubstrate dependence. Their results suggested that there were group differences in symptomatology; but not those that
Khantzian had predicted. The group discriminations were made based upon psychological disturbances believed to be secondary to addiction.

Further criticism of the SMH was highlighted by Greene et al. (1993) who used the MMPI (a personality and psychopathology inventory) to assess differences in symptoms between cocaine and marijuana abusers. No significant difference was found and this was still true when the results were compared to those of alcoholics and non-addicted psychiatric inpatients around the same age in the same facility.

Khantzian and Mack (1983) reported on a potential treatment for intravenous cocaine dependence (IV CD), through use of methylphenidate - a substitute stimulant. In his SMH, he argued that cocaine acts as an augmenter for hypomanic, high-energy individuals as well as persons with atypical bipolar disorder. Furthermore, he noted that it might act paradoxically to calm ADHD. Khantzian proposed that the gnawing, depressive effects such as anhedonia, caused by craving, could be resolved by a prescription stimulant.

Mariani and Khantzian (2014) offered the case of Bobby, the ‘Miracle Cure’ as an example of the efficacy of this treatment concept and the validity of the SMH. Bobby was a forty-nine year old male treated by Khantzian in the summer of 2011, with sustained release methylphenidate. After treatment with methylphenidate he reported that he finally felt that he had a choice in using cocaine and had been abstinent from cocaine use for 8 months at the time of publishing. This fits the model of the SMH because it may indicate that the methylphenidate helped alleviate emotional difficulties that the cocaine was being used to treat.

However, this support for the SMH is questioned by the use of case study evidence, which is subject to researcher bias and cannot be extrapolated to a larger population where variables such as race, age, and gender could confound the evidence.

With developments in neural imaging and our knowledge of the neural networks of reward, the SMH came under additional criticism. For example, Kosten (1998) argues against the significance of psychopharmacological specificity because of how often the dopaminergic connections involving the nucleus accumbens and the ventral tegmentum have been identified as the ‘final common pathway’ with many abused substances.

A more recent study by Colman et al. (2015) proposes extensive experimental flaws in earlier research disputing psychopharmacological specificity. Specifically, they assert that the measures of the personality/psychic factors that contribute to dependence have been weak and inconsistent. Furthermore, the sample size has been much smaller than required, with some studies having as little as 20 and 29 participants in each drug group (Greene et al, 1993). In
addition, the self-report nature of earlier studies has been said to massively underestimate drug use (Magura and Kang, 1996).

In light of this, Colman et al. (2015) used two independent raters to classify individuals as being addicted to a depressant, stimulant or opiate. This combined with the use of urinalysis to corroborate report increases validity and reliability and adds more credence to their findings. Their study divided groups of participants according to the aforementioned diagnostic method into users of ‘depressants’, ‘opiates’ or ‘stimulants’. They performed MANOVAs on each group to assess if the drug group matched personality and psychic profiles of dependents, proposed in the SMH.

In the first group, they found that MMPI-II scores were lower in depressant-dependent individuals, in agreement with the SMH. They proposed that this reflects a dismissal of affect or denial. This in combination with evidence from Suh et al (2008) indicates that Khantzian’s prediction of a denial-based defence system among depressant users was correct. The second group (opiates) revealed further evidence congruent with the SMH as their analysis revealed significantly reduced ego strength compared to other groups. This again is in keeping with Khantzian’s proposal of early trauma driving the use of opiates. The stimulant group didn’t produce statistically significant data in favour of SMH. However, Colman et al. believe that this may be due to the physiological heterogeneity of stimulant SUDs, a unique feature not true of either other class of drug.

The use of scales such as MMPI-II, considered to be homogenous in nature, may provide evidence that carries greater weight. Suh and colleagues (2008) also used this homogenous scale and found further support for the SMH. Unlike Colman et al. they found that those identified as cocaine-dependent showed a higher drive for elation and restlessness, as proposed by the SMH.

Whilst some evidence does support Khantzian’s model of ‘drug of choice’, the flaws and variability in experimental design draw into question the validity of the evidence. Furthermore, whilst Colman et al. and Suh et al. provide the most convincing evidence in favour, their studies found agreement for specificity within separate drug classes.
Evaluating the self-medication hypothesis though the lens of the biological paradigm.

It has been argued that the SMH is overly reductionist as it ignores many crucial concepts in addiction, such as the wealth of biological evidence indicating both the importance of genetic vulnerabilities and the neuroplastic changes caused by repeated drug use.

For example, Cocores et al. (1987), propose that ADHD psychopathology involves dopamine dysregulation and not dysthymia, as predicted by Khantzian. They suggested that cocaine use in an already dopamine deficient individual could cause a reversible and temporary ADHD presentation. This backward causal directionality could indicate both that psychopathology follows drug use and that ignoring biological evidence is a critical conceptual weakness of the SMH.

Goldsmith (1993) believed that this reductionism could be resolved by using a new integrated psychology for addictions that incorporate biological vulnerabilities, reward mechanisms, quantity of substance and chronicity of use.

An explanation of this self-psychology highlights these weaknesses and offers a basis for potentially superior treatment options. The self can be considered as the primary psychological experience of the individual, whose motivations are the attainment of psychological wholeness and maintenance of such cohesion. This maintenance is achieved through a complex collection of important relationships named self-self object (SSO) relationships, which if disrupted cause personal turmoil.

In agreement with the SMH, personal turmoil may lead to substance use as one seeks reassurance from the effects of a drug. Unlike the SMH, the self psychology account includes the fact that repeated drug use stimulates important reward areas in the brain such as the nucleus accumbens and the ventral tegmentum and that to some degree repeated drug use is indeed drug motivated because of changing dopamine levels. Furthermore, when biological considerations are included, the importance of chronicity becomes apparent as one can appreciate that dopamine depletion becomes more severe over time leading to more intense desire for cocaine.

Whilst the SMH proposes that treatment be based on removing the supposedly causal dysthymia, self-psychology based treatment is focused on several very different factors. Abstinence is one such factor, which whilst not emphasised to the same degree by the SMH, is viewed with greater importance in Goldsmith’s model because of the aforementioned neurological changes.

In addition, the effect of the amount of substance that one uses is considered by self-psychology. Using alcoholism as an example, when one exceeds a few
drinks the hedonic effects disappear to be replaced by what Tamerin and Mendelson (1969) called a 'morose or suicidal state'. This state leads to an intrapsychic and interpersonal turmoil that further heightens the importance of abstinence when one considers that little beneficial effects are seen from prescribed antidepressant drugs.

Furthermore, this approach accounts for how eventually, repeated drug use will lead to a self-experience of loss of control, which will in turn cause an additional SSO relationship disruption, like those described above, inducing trauma and fear for the self, which further perpetuates drug use. This model’s appreciation for the importance of biological factors allows one to admit to having lost control. This is because once one can appreciate that biological changes in their brain have contributed to their addiction, they can disavow, as is seen in AA where there is a great focus on the reduction of guilt.

Whilst the aforementioned implies that the SMH may be incomplete, more recent contributions from a neuropsychodynamic perspective seem to answer this criticism.

That psychopathology and disaffect can often follow drug use is a significant criticism of the SMH. However, Khantzian (2001) proposes the concept of ‘disuse atrophy’ in response. This term is largely used when discussing orthopaedics or neurology, describing how without use, muscles and nerves atrophy. In this instance however, it is the already weak psychological defence mechanisms that ‘atrophy’ when replaced by an individual’s drug of choice. Khantzian proposes that this provides a neuropsychodynamic explanation of the role of chronicity in substance abuse, however this concept is not empirically testable which questions how valid it is in reality.

Self-psychology indicates that the SMH provides a valid platform upon which to build further diagnostic and treatment avenues for addiction, in a future with a wealth of biological knowledge.
**Case Study:**

The author had the opportunity to discuss substance misuse with a patient (anonymised as ‘L’) who presented to primary care for issues relating to unipolar depression.

L is a 27-year-old homosexual male who disclosed a 7-year history of recreational drug use. He reported that the use of substances began as a means to party ‘on the same level’ as those he was surrounded with. He reported a sense of inclusion when he first began taking MDMA, aged 20. This, he described, was because he felt that the use of recreational substances is an integral part of LGBTQ+ (and particularly gay male) culture and in partaking he felt a sense of belonging.

L subsequently began to experiment with cocaine and mephedrone whilst attending nightclubs and parties. After 2 years of drug use L describes beginning to take the aforementioned substances even when alone in an effort to ameliorate feelings of loneliness and isolation. This in turn led to the use of mobile applications in search of casual sexual partners (CSP). This new context of substance abuse led to sharing of drugs between partners and L’s introduction to GHB.

When asked if he felt he was substance dependent, L was adamant he was not. However, he did describe that he often feels deep regret after drug use and makes personal resolutions to abstain. Unfortunately, L finds himself seeking drugs and the concomitant sexual camaraderie without an apparent active decision to do so and feels this has contributed to his depression despite increasing doses of citalopram.

In the history provided by L, he displays clear psychological vulnerabilities that he could be endeavouring to attenuate with recreational drugs. Additionally, there seems to be a significant cultural pressure that might represent a high prevalence of dysthymia among the community.

In keeping with the findings of the literature review the ‘drug of choice’ concept doesn’t fit patient L. It seems that availability of substance or the sharing of substances with CSP is more important to him than the use of a specific substance.

Perhaps more important is that L represents a rising culture of substance abuse, ‘chemsex’. This can be defined as ‘intentional sex under the influence of psychoactive drugs, mostly among men who have sex with men’ (McCall et al., 2015).

Drugs commonly used are GHB (and its precursor GBL) as well as crystalline methamphetamine (Bourne et al., 2014). As a consequence, there is growing concern over the public health implications of ‘chemsex’ events, particularly
when ‘slamming’ (the injection of methamphetamine) is involved (Colfax et al., 2006).

One can speculate that institutional homophobia could be a cause of the maladaptive psychosexual development driving such behaviours. This has been corroborated in self-report studies where participants reported using ‘chemsex’ events to manage issues such as a lack of self-esteem, internalised homophobia and stigma regarding their HIV status (Bourne et al., 2014).

There is evidence of an increasing prevalence of ‘chemsex’ requiring the use of mental health services (Stuart, 2013). Despite this the National Institute for Health and Care Excellence (NICE) has no official recommendations regarding psychiatric sequelae of ‘chemsex’ (NICE, 2014) and as such the author proposes consideration of the self-medication hypothesis (SMH) when moving forward.
**Conclusion:**

The self-medication hypothesis proposes an explanation for the initiation and maintenance of drug dependence. It is proposed that arrested emotional development leaves an individual susceptible to alexithymia which can in turn induce a drive to use a drug that will give one control over the disaffect they suffer. Furthermore, integral to the SMH is the concept of drug of choice, whereby an individual displays preferential drug use that results from an interaction between the physiological action of the drug, an individual's affect state, and the availability of the substance.

Through review of the literature, the author discovered significant evidence in favour of the relationship between substance dependence and antecedent disaffect. Whilst knowledge of a drug of choice would be of huge help in treating those with substance dependence as it may reveal specific vulnerabilities motivating their use (Suh et al., 2008), the literature showed little evidence to support Khantzian’s proposed psychopharmacological specificity.

However, what is demonstrated by the case study is that the SMH may provide a useful paradigm to develop a much-needed model for understanding ‘chemsex’. If this were to be found applicable on a larger scale, perhaps measures could be taken to attenuate the public health impact of disinhibited high-risk sexual behaviour.
References:


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