SUBSTANCE USE AND SEXUAL DYSFUNCTION
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ABSTRACT: Substance use disorders form a major part of global disease burden. With increasing trend of use of psychoactive substance, the deleterious effects associated with it also increases. These effects may be biological, social or legal. Among the biological consequences of substance use, little is known of its effect on sexual functioning. In common parlance it is said that many substances increase the sexual desire and hence act as an aphrodisiac. To what extent this is true remains a question of debate. The purpose of this article is to review and summarize the available literature on the impact of psychoactive substances like alcohol, tobacco, cannabis and others on sexual functioning. Almost all of them are associated with one or other form of sexual dysfunction. The mechanism by which they exert such deleterious effect also varies. Further, the sexual dysfunction resulting from substance use can itself have bearing on treatment aspects of substance use. The relationship between sexual dysfunction and substance is attributed not only to pharmacological effects, but also to psychological and social factors stemming from substance use. This information of sexual consequence of substance will be of interest and may serve as a powerful tool to healthcare providers.

KEYWORDS: Substance, Drugs, Sexual dysfunction, erectile dysfunction,

INTRODUCTION: Substances which have an ability to bring about any change in an individual’s consciousness, mood or thinking are termed as psychoactive substances (drugs). These substances can either be licit like caffeine, tobacco or alcohol; and illicit like heroin, amphetamine, 3,4-methylenedioxymethamphetamine (MDMA), cannabis, cocaine etc.

Substance use causes a significant burden on the society worldwide. Substance use is responsible for 8.9% of the total disease burden worldwide, tobacco accounting for 4.1%, alcohol 4% and illicit drugs 0.8%.\(^1\) The harmful effects of these substances can be divided into:

- Chronic health effects, e.g. liver cirrhosis in alcoholism, lung cancer and emphysema due to cigarette smoking.
- Acute biological health effects, e.g. accidents and other causalities associated with drug use.
- Acute social problem like a break up in relationship or an arrest
- Chronic social problems like defaults in working life or in family roles.\(^2\)

Sexual dysfunction affects many men and women in their lifetime. Sexual dysfunction covers a range of problems including erectile dysfunction (ED), premature or delayed ejaculation in men, pain associated with intercourse, low libido and poor response to sexual contact. Psychoactive substances are known to affect a person’s sexual behaviour and ability to function. These substances are often taken as a means to camouflage psychological or emotional problems or to ignore physical
difficulties which are contributing to sexual dysfunction. Many substance abusers feel that their sexual performance improves after substance use. But, their partners often report the opposite.

**Alcohol:** Alcohol is commonly believed to be a powerful sexual facilitator and aphrodisiac due to its disinhibiting properties. Alcohol abuse is the leading cause of impotence and other disturbances in sexual dysfunction. Alcohol is disinhibiting, and even in small doses it may lead to increased sexual desire. But it diminishes performance and delays orgasm and ejaculation. Acute intoxication can result in erectile failure. Episodic erectile dysfunction is significantly higher in men consuming more than 3 standard units of alcohol (12g of ethanol). Chronic and persistent alcohol use is known to induce sexual dysfunction which leads to marked distress and interpersonal difficulty. This in turn worsens the alcohol abuse and hence a vicious cycle sets in. Sexual dysfunction in persons abusing alcohol may be due to its depressant effect; alcohol related disease or due to a multitude of psychological factors related to alcohol use.

Virtually all aspects of the human sexual response are affected by alcohol especially sexual desire and erection. The most common condition reported by Arackal and Benegal (2007), in India, was premature ejaculation followed closely by low sexual desire and erectile dysfunction. Van Thiel and Lester (1979) reported that 61% of patients dependent on alcohol reported sexual dysfunction, the most common being erectile dysfunction followed by reduced sexual desire. Erectile dysfunction and reduced sexual desire are frequently seen to be coexisting. Alcohol-induced sexual dysfunction may not be reversible with abstinence. Long-term alcohol abuse interferes with the HPA axis, resulting in reduced testosterone and feminization in men, and thus reducing sex drive and performance.

Ethanol has biphasic behavioral effects. At low doses, the first effects that are observed are heightened activity and disinhibition. At higher doses, cognitive, perceptual and motor functions become impaired. Ethanol increases the inhibitory activity mediated by gamma-aminobutyric acid-A (GABA-A) receptors and decreases the excitatory activity mediated by glutamate receptors. GABA-A receptor activation mediates many of the behavioral effects of ethanol including motor incoordination, anxiolysis and sedation. Alcohol also inhibits the hypothalamic-pituitary-adrenal axis and reduces the release of gonadotropin from the pituitary. The chronic abuse of alcohol may cause testicular atrophy, inhibition of T-cell production and inhibition of spermatogenesis, apart from its direct oxidative toxicity.

**Tobacco:** Cigarette smoking is a major public health problem worldwide and moreover the warnings of cancer and cardiovascular disease have lost their ability to charm the population to quit smoking. Some studies have shown that smoking is not associated with ED. However, the Massachusetts Male Aging Study found that the incidence of ED doubled in a sub-group of men smokers free from vascular-disease. Little is known about the effect of smoking on the recovery from ED and that of ED on starting or stopping smoking. It has also been shown that past smoking is also associated with ED. Excess risk of ED in past smokers decreases substantially in the initial 2-3 years; thereafter the risk reduction slows down, so that up to 10 years is required for smokers to achieve the risk level of never smokers. However, a prospective study found no change in ED among smokers who stopped smoking for an eight-year period. There is little evidence regarding recovery from ED after stopping smoking. Stopping smoking may improve ED in a considerable
proportion of smokers. Higher rate of ED in former smokers may be related to smoking induced vascular diseases.

Even regular exposure to passive smoking at home and work increases the risk of ED among non-smokers. Regular exposure to passive smoking increases the risk of coronary heart disease and therefore, passive smoking may cause ED by causing vascular diseases. The dose effect of cigarette smoking on ED is controversial. Some studies have shown a strong association between the intensity of cigarette smoking and severity of ED whereas some studies have found that only heavy smokers are at higher risk of ED.

**Stimulants:** Psychostimulants tend to increase sexual desire in the short term, but long-term use may result in reduced sex drive. Amphetamine use is also associated with ejaculatory disturbance in the long term. Ecstasy alters libido and can increase sex drive at the expense of impaired sexual performance (delayed orgasm and erectile dysfunction), possibly due to increased prolactin secretion. Like ecstasy, methamphetamine is associated with a significant disinhibiting effect and has been implicated in an increase in sexual risk-taking behaviour. Unlike ecstasy, it appears not to be associated with negative effects on sexual function.

**Cannabis:** Derived from the cannabis sativa plant, del-9-tetra-hydrocannabinol, the primary active ingredient in cannabis. Cannabinoids are generally inhaled by smoking, but may also be ingested. Cannabis is the most widely cultivated and used illicit drug with an estimated 147 million people or 2.5% of the world population using it annually.

Cannabis use has been linked to earlier and more frequent sexual activity, having multiple sexual partners, having casual sexual partners while traveling, inconsistent contraceptive use, and being diagnosed with a sexually transmissible infection. Smith et al. (2010) studied the effects of cannabis on sexual dysfunction and showed that daily cannabis use compared with no use was associated with an increased likelihood of reporting two or more sexual partners in the previous year in both men and women. Further, daily cannabis use was associated with reporting a diagnosis of a sexually transmissible infection in women but not men. Frequency of cannabis use was unrelated to sexual problems in women but daily use vs. no use was associated with increased reporting among men of an inability to reach orgasm, reaching orgasm too quickly and too slowly. The sexual dysfunction is due to the effect of the active ingredient on central nervous and cardiovascular system.

**Opioids:** Heroin reduces sexual feelings and may decrease desire, and cause erectile and ejaculatory dysfunction. High-dose methadone is well known to be associated with sexual dysfunction. Buprenorphine is also associated with sexual dysfunction.

Opioids reduce testosterone level resulting in decreased libido and erectile dysfunction. Many men who are taking prescribed or illicit opioids suffer from several side effects including sexual dysfunction like erectile dysfunction and decreased libido. These unwanted side effects have been correlated to hypogonadism and likely hypogonadotrophic hypogonadism. Testosterone levels are typically lowered 1–4 hours after acute administration of opioids and return to normal levels within 24 hours of stopping the opioid. Chronic administration of opioids for
nonmalignant pain result in tonic decreases in both total (TT) and free (FT) testosterone levels in an apparent dose-dependent fashion. Studies examining potential adjuvant therapies treating potential hormonally mediated side effects are rare. Bliesener and colleagues (2005) studied the hormonal effects of opioid maintenance and found that individuals taking buprenorphine had significantly higher plasma testosterone levels and showed less sexual dysfunction compared to patients receiving methadone. Women also experience similar hormonally linked side effects of opioids including dysmenorrhea and sexual dysfunction. Several studies have demonstrated reduced estrogen levels in women on methadone maintenance. Reduced LH is also observed and appears to be more pronounced in postmenopausal women. Interestingly, testosterone levels also appear to be reduced in women taking prescribed opioids and may be related to body mass index and estrogen replacement therapy. The direct consequences of reduced LH and progesterone levels on dysmenorrhea are currently unclear.

**Cocaine**: Cocaine is found in the leaves of *Erythroxylon coca*. It is a powerful central and peripheral nervous system stimulant that can be taken intranasally, injected intravenously or smoked. In the brain, cocaine acts as a monoamine transporter blocker, with similar affinities for dopamine, serotonin, and norepinephrine transporters. Cocaine appears to have two opposite effects on sexual functioning according to its acute or chronic abuse. New or infrequent cocaine users may report that cocaine induces spontaneous erection and ejaculation. However, other research reported that ambiguous findings of cocaine’s impact on sexuality might be due to variation in the dosage, route of administration or other factors. MacDonald et al. (1988) found that of men who had used cocaine for 1 year or longer, 66% reported to have erection difficulties. With chronic abuse of cocaine, sexual dysfunction is attributed mainly to hyperprolactinemia and downregulation of the hypothalamic dopaminergic receptors. There are reports of cases of priapism associated with intracavernosal injection of cocaine.

**MANAGEMENT**: Before starting patients on any medications, it is necessary to obtain a sexual function history. Change in sexual function should be monitored on subsequent visits. Open discussion of the problem may help to reduce non-adherence later on. This will also help to distinguish substance-induced sexual dysfunction from independent sexual dysfunction. A number of approaches have been tried to relieve sexual dysfunction, including behavioral strategies to modify sexual technique. In individuals who are unable to reduce their substance intake, it may be necessary to institute a drug to treat the sexual dysfunction (adjuvant therapy), such as sildenafil (Viagra), tadalafil (Cialis), vardenafil (Levitra) or yohimbine. In appropriate cases, psychosexual therapy should be offered and, with all patients, attention should be paid to the effects on the whole person.

In men, the primary treatment for opioid-induced endocrine deficiency resulting in hypogonadism is testosterone supplementation. There has been far less research regarding opiate-induced endocrine deficiencies in women than in men. Hypothetically, androgen treatment would relieve clinical symptoms and reduce risks of osteoporosis in affected women. In younger women, oral contraceptive pills (OCPs) might have benefit; particularly an OCP with a relatively
Another approach might be the administration of DHEAS. Although the potential value of DHEAS therapy in women remains controversial, it may be the most appropriate treatment option for those with opioid-induced endocrine deficiency. The highest quality studies evaluating DHEAS treatment support its use in women with adrenal insufficiency.66,67,68 Usually, DHEAS supplementation of 50 to 100 mg/day will sufficiently raise androgen levels to normal or near normal levels.

**DISCUSSION:** The relationship between psychoactive substances and sexual behavior are complex, not always direct. Caution should be taken when interpreting research on the effects of drugs on sexual function. Psychological, physiological, environmental, or cultural factors may be associated with use of a particular substance, and may have independent or intervening effects on sexual functioning.35 There is a wide variation in the dosing frequency, amount and duration of drug abuse, as well as in the purity of substance that may influence the occurrence and severity of ED; but these factors were hardly taken consideration in the studies. Polidydrug use is not uncommon in men with illicit drug abusers. In the study of 701 illicit drug users, 92.5% were active smokers, 6.6% had alcoholism and 19.0% used more than one kind of illicit drugs.69 However, the synergic effect of polydrug use on ED is rarely investigated.

Psychoactive substance dependence is a predominantly male activity. Illicit drug use is even more prevalent among young people than in older age groups.70 Despite these limitations and knowledge gaps, the available research does suggest that alcohol, tobacco and illicit drugs have deleterious effects on men's erectile function. Virtually, alcohol and illicit drugs are known to have inhibitory effects on libido and ejaculation.35,69,71 Issues concerning sexuality have always attracted attention from the media and general population. Sexual dysfunction like ED can be used as a new weapon in the war on tobacco, alcohol and illicit drugs.

**REFERENCES:**


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