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.[3]

Alcohol: Alcohol is commonly believed to be a powerful sexual facilitator and aphrodisiac due to its disinhibiting properties.[4]Alcohol abuse is the leading cause of impotence and other disturbances in sexual dysfunction.[5]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).[6]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.[7]

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.[8]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.[9] Erectile dysfunction and reduced sexual desire are frequently seen to be coexisting.[10,11,12,13] Alcohol-induced sexual dysfunction may not be reversible with abstinence.[14] Long-term alcohol abuse interferes with the HPA axis, resulting in reduced testosterone and feminization in men, and thus reducing sex drive and performance.[15]

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.[16,17]Ethanol increases the inhibitory activity mediated by gamma-aminobutyric acid-A (GABA-A) receptors and decreases the excitatory activity mediated by glutamate receptors.[18] GABA-A receptor activation mediates many of the behavioral effects of ethanol including motor incoordination, anxiolysis and sedation.[19] 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.[20]

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.[21,22] However, the Massachusetts Male Aging Study found that the incidence of ED doubled in a sub-group of men smokers free from vascular-disease.[23] 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.[24] 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.[25,26] There is little evidence regarding recovery from ED after stopping smoking. Stopping smoking may improve ED in a considerable

proportion of smokers.[27]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.[25] Regular exposure to passive smoking increases the risk of coronary heart disease and therefore, passive smoking may cause ED by causing vascular diseases.[28] 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.[29,30]

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.[31] 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.[32,33] 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.[34]

Cannabis: Derived from the cannabis sativa plant, dela-9-tetra-hydrocannabinol, the primary active ingredient in cannabis.[35] 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.[36]

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.[37,38,39,40,41,42] 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.[43] 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.[46]

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.[47,48,49,50] 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.[51,52] 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.[53] 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.[54] 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.[55] 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.[56]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.[57]

Cocaine appears to have two opposite effects on sexual functioning according to its acute or choric abuse. New or infrequent cocaine users may report that cocaine induces spontaneous erection and ejaculation.[58] 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.[59]

MacDonald et al. (1988) found that of men who had used cocaine for 1 year or longer, 66% reported to have erection difficulties.[60]With chronic abuse of cocaine, sexual dysfunction is attributed mainly to hyperprolactinemia and downregulation of the hypothalamic dopaminergic receptors.[61] There are reports of cases of priapism associated with intracavernosal injection of cocaine.[62]

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.[63.64,65] 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

androgenic progestin component. However, OCPs are also known to suppress free testosterone. 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. Polydrug 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:

1. The world health report 2002: Reducing risks, promoting health life. World Health Organization: Geneva, 2002.
2. Babor TF, Caetano R, Casswell S, Edwards G, Giesbrecht N, Graham K, et al. Alcohol: no ordinary commodity-research and public policy. Oxford: Oxford University Press, 2003.
3. Cummins T, Miller S. The effects of drug abuse on sexual functioning. In: Levine SB (editor). Handbook of clinical sexuality for mental health professionals. New York: Brunner-Routledge; 2003:p.443-456.
4. Rosen RC. Alcohol and drug effects on sexual response: human experimental and clinical studies. Ann Rev Sex Res 1991; 2:119-79.
5. Mendelson JH, Mello NK. Medical progress, Biologic concomitants of Alcoholism. N Engl J Med 1979; 301:912–921.
6. Mirone V, Ricci E, Gentile V, BasileFasolo C, Parazzini F. Determinants of erectile dysfunction risk in a large series of Italian men attending andrology clinics. Eur Urol. 2004; 45:87–91.
7. Gelder M, GathD, Mayon P, Cowen P. Etiology of sexual dysfunction. In: Oxford Text Book of Psychiatry. 3rd ed. Oxford University Press: Oxford, UK; 1996.
8. Arackal BS, Benegal V. Prevalence of sexual dysfunction in male subjects with alcohol dependence. Indian Journal of Psychiatry 2007; 49(2):109-112.

9. Van Thiel DH, Lester R. The effect of chronic alcohol abuse on sexual function. The Clin Endocrinol Metab 1979; 8:499–510.
10. Jensen SB, Gludd C. Sexual dysfunction in men with alcoholic liver cirrhosis: A comparative study. Liver 1985; 5:94–100
11. Fahrner EM. Sexual dysfunction in male alcohol addicts, prevalence and treatment. Arch Sex Behav 1987; 16:247–57
12. Fagan PJ, Schmidt CW, Wise TN, Derogatis LR. Alcoholism in patients with sexual disorders. J Sex Marital Ther. 1988; 14:245–252
13. Gumus B, Yigitoglu MR, Lekili M, Vyanik BS, Muezzinoglu T, Buyuksu C. Effect of long term alcohol abuse on male sexual function and serum gonadal hormone levels. IntUrolNephrol. 1998; 30:755–759.
14. Schiavi RC, Stimmel BB, Mandelli J, White D. Chronic alcoholism and male sexual function. Am J Psychiatry. 1995; 152:1045–1051.
15. Emanuele MA, Emanuele NV. Alcohols effects on male reproduction. Alcohol Health Res World 1998; 22:195–201.
16. McKay A. Sexuality and substance use: The impact of tobacco, alcohol and selected recreational drugs on sexual function.Can J Hum Sex 2005; 14:47-56.
17. Jacobs MR, Fehr KOB. Drugs and drug abuse: a reference text. 2nd ed. Toronto: Addiction Research Foundation, 1987.
18. McBride WJ. Central nucleus of the amygdala and the effects of alcohol and alcohol-drinking behavior in rodents.PharmacolBiochemBehav2002; 71:509-515.
19. Grobin AC, Matthews DB, Devaud LL, Morrow AL. The role of GABA(A) receptors in the acute and chronic effects of ethanol. Psychopharmacology (Berl) 1998; 139:2-19.
20. Nordmann R, Ribière C, Rouach H. Ethanol-induced lipid peroxidation and oxidative stress in extrahepatic tissues.Alcohol1990; 25:231-237.
21. Johannes CB, Araujo AB, Feldman HA, Derby CA, Kleinman KP, McKinlay JB. Incidence of erectile dysfunction in men 40 to 69 years old: Longitudinal results from the Massachusetts Male Ageing Study. J urol 2000; 163:460-463
22. Moreira ED Jr, Lbo CF, Diament A, Glasser DB. Incidence of erectile dysfunction in men 40 to 69 years old: results from a population-based cohort study in Brazil. Urology 2003; 61:431-436.
23. Feldman HA, Johannes CB, Derby CA, Kleinman KP, Mohr BA, Araujo AB, McKinlay JB. Erectile dysfunction and coronary risk factors: Prospective results from the Massachusetts Male Aging Study. Prev Med 2000; 30:328-38.
24. Parazzini F, MenchiniFabris F, Bortolotti A, Chatenoud L, Colli E, Landoni M, Lavezzari M, Turchi P, Sessa A, Mirone V. Frequency and determinants of erectile dysfunction in Italy. EurUrol 2000; 37: 43-49.
25. Bortolotti A, Parazzini F, Colli E, Landoni M. The epidemiology of erectile dysfunction and its risk factors. Int J Androl 1997; 20:323-334.
26. Derby Ca, Mohr BA, Goldstein I, Feldman HA, Johannes CB, McKinlay JB. Modifiable risk factors and erectile dysfunction: can lifestyle changes modify risk? Urology 2000; 47:277-286.
27. Pourmand G, Alidaee MR, Rasuli S, Maleki A, Mehrsai A. Do cigarette smokers with erectile dysfunction benefit from stopping? A prospective study. BJU Int 2004; 94:1310-1313.

28. Kawachi I, Colditz GA, Speizer FE, Manson JE, Stampfer MJ, Willett WC, Hennekens CH. A prospective study of passive smoking and coronary heart disease. Circulation 1997; 95:2374-2379.
29. Natali A, Mandaini N, Lombardi G, Del Popolo G, Rizzo M. Heavy smoking is an important risk factor for sexual dysfunction in young men. Int J Impot Res 2005; 17:227-230.
30. Moreira ED Jr, Beastane WJ, Bartolo EB, Fittipaldi JA. Prevalence and determinants of Erectile dysfunction in Santos, southeastern Brazil. Sao Paulo Med J 2002; 120: 49-54.
31. Cocores JA, Miller NS, PottashAC, Gold MS. Sexual dysfunction in abusers of cocaine and alcohol. Am J Drug Alcohol Abuse 1988; 14:169–173.
32. Zemishlany Z, Aizenberg D, Weizman A. Subjective effects of MDMA ('ecstasy') on human sexual function. Eur Psychiatry 2001; 16:127–130.
33. Passie T, Hartmann U, Schneider U, Emrich HM, Kruger TH. Ecstasy (MDMA) mimics the post-orgasmic state: impairment of sexual drive and function during acute MDMA-effects may be due to increased prolactin secretion. Med Hypotheses 2005; 64:899–903.
34. Schilder AJ, Lampinen TM, Miller Ml, Hogg RS. Crystal methamphetamine and ecstasy differ in relation to unsafe sex among young gay men. Can J Public Health 2005; 96:340–343.
35. Peugh J, Belenko S. Alcohol, drugs and sexual function: a review. J Psychoactive Drugs2001; 33:223-232.
36. World Health Organization. Cannabis (facts and figures). Geneva: WHO; 2008. Available at: http://www.who.int/substance_abuse/facts/cannabis/en/(accessed March 13, 2013).
37. Abel EL. Marihuana and sex: a critical survey. Drug Alcohol Depend 1981; 8:1–22.
38. Arvidson M, Kallings I, Nilsson S, Hellberg D, Mårdh PA. Risky behavior in women with history of casual travel sex. Sex Transm Dis 1997; 24:418–421.
39. Boyer CB, Shafer MA, Teitle E, Wibbelsman CJ, Seeberg D, Schachter J. Sexually transmitted diseases in a health maintenance organization teen clinic: Associations of race, partner's age, and marijuana use. Arch PediatrAdolescMed 1999; 153:838–844.
40. Clark T, Robinson E, Crengle S, Watson P. Contraceptive use by Maori youth in New Zealand: Associated risk and protective factors. N Z Med J 2006; 119:U1816.
41. Guo J, Stanton B, Cottrell L, Clemens RL, Li X, Harris C, Marshall S, Gibson C. Substance use among rural adolescent virgins as a predictor of sexual initiation. J Adolesc Health 2005; 37:252–5.
42. Johnson SD, Phelps DL, Cottler LB. The association of sexual dysfunction and substance use among a community epidemiological sample. Arch Sex Behav 2004; 33:55–63.
43. Smith AMA, Ferris JA, Simpson JM, Shelley J, PittsM, and Richters J. Cannabis use and sexual health. J Sex Med 2010; 7:787–793.
44. Mirin SM, Meyer RE, Mendelson JH, Eellingboe J. opiate use and sexual function. Am J Psychiatry 1980; 137:909–915.
45. Bliesener N, Albrecht S, Schwager A, Weckbecker K,Lichtermann D, Klingmuller D. Plasma testosterone andsexual function in men receiving buprenorphine maintenance for opioid dependence. J Clin Endocrinol Metab 2005; 90:203–206.
46. Palha AP, Esteves M. A study of the sexuality of opiate addicts. J Sex Marital Ther 2002; 28:427–437

47. Daniell HW. DHEAS deficiency during consumption of sustained-action prescribed opioids: Evidence for opioid-induced inhibition of adrenal androgen production. J Pain 2006; 7:901-907.
48. Daniell HW, Lentz R, Mazer NA. Open-label pilot study of testosterone patch therapy in men with opioid-induced androgen deficiency. J Pain 2006; 7:200-210.
49. Brown RT, Zuelsdorff M, Fleming M. Adverse effects and cognitive function among primary care patients taking opioids for chronic nonmalignant pain. J Opioid Manag 2006; 2:137-146.
50. Ruan X. Drug-related side effects of long-term intrathecal morphine therapy. Pain Physician 2007; 10:357-366.
51. Facchinetti F, Comitini G, Petraglia F, Volpe A, Genazzani AR. Reduced estriol and dehydroepiandrosteronesulphate plasma levels in methadone-addicted pregnant women. Eur J ObstetGynecolReprodBiol 1986; 23:67-73.
52. Woody G, McLellan AT, O'Brien C, Persky H, Stevens G, Arndt I, Carroff S. Hormone secretion in methadone-dependent and abstinent patients. NIDA Res Monogr 1988; 81:216-223.
53. Daniell HW. Hypogonadism in men consuming sustained-action oral opioids. J Pain 2002; 3:377-384.
54. Bliesener N, Albrecht S, Schwager A, Weckbecker K, Lichtermann D, Klingmuller D. Plasma testosterone and sexual function in men receiving buprenorphine maintenance for opioid dependence. J Clin Endocrinol Metab 2005; 90:203-206.
55. Daniell HW. Opioid endocrinopathy in women consuming prescribed sustained-action opioids for control of nonmalignant pain. J Pain 2008; 9:28-36.
56. Ensrud KE, Blackwell T, Mangione CM, Bowman PJ, Bauer DC, Schwartz A, Hanlon JT, Nevitt MC, Whooley MA. Study of Osteoporotic Fracture Research Group. Central nervous system active medications and risk for fractures in older women. Arch Intern Med 2003; 163:949-957.
57. Ritz MC, Cone EJ, Kuhar MJ. Cocaine inhibition of ligand binding at dopamine, norepinephrine and serotonin transporters: a structure-activity study. Life Sci1990; 46:635-45.
58. Buffum J. Pharmacolosexology: the effects of drugs on sexual function: a review. J Psychoactive Drugs 1982; 14:5-44.
59. Buffum J, Moser C, Smith D. Street drugs and sexual function. In: Sisten JMA, ed. Handbook of sexology. Volume 6: the pharmacology and endocrinology of sexual function. New York: Elsevier Science Publishers,1988.
60. MacDonald PT, Waldorf D, Reinarman C, Murphy S. Heavy cocaine use and sexual behavior.J Drug Issues1988; 18:437-455.
61. Saso L. Effects of drug abuse on sexual response.Ann Ist Super Sanità2002; 38:289-96.
62. Mireku-Boateng AO, Tasie B. Priapism associated with intracavernosal injection of cocaine.UrolInt2001; 67:109-10.
63. Daniell HW. Opioid-induced androgen deficiency. CurrOpinEndocrinol Diabetes. 2006; 13(3):262-266.
64. Endocrine Society. Androgen therapy in men. J Clin Endocrinol Met. 2006; 6(91):1995-2010.
65. Endocrine Society. Androgen therapy in men. J Clin Endocrinol Met. 2006; 10(91):3697-3716.
66. Gurnell EM, Hunt PJ, Curran SE, et al. A randomised, controlled trial of long-term DHEA replacement in Primary Adrenal Insufficiency. J Clin Endocrinol Metab. 2007[Epub ahead of print].

67. Morales AJ, Hubris RH, Hwang JY, Asakura H, et al. The effect of six months treatment with a 100 mg daily dose of dehydroepiandrosterone (DHEA) on circulating sex steroids, body composition and muscle strength in age-advanced men and women. Clin Endocrinol. 1998; 49(4):421-432.
68. Panjari M, Davis SR. DHEA therapy for women: effect on sexual function and wellbeing. Hum Reprod Update. 2007; 13(3):239-248.
69. Bang-Ping J. Sexual dysfunction in men who abuse illicit drugs: a preliminary report. J Sex Med2007.
70. Halikas J, Weller R, Morse C. Effects of regular marijuana use on sexual performance. J Psychoactive Drugs1982; 14:59-70.
71. Palha AP, Esteves M. Drugs of abuse and sexual functioning. Adv Psychosom Med2008; 29:131-49.

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