



SEX RESEARCH UPDATE

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This instalment of *Sex Research Update* summarizes recent research on Canadian sexually transmitted infection rates; sexual behaviour and condom use of Canadian young people; the effect of correct and consistent condom use on chlamydia and gonorrhea prevention among adolescents; testosterone, sexual offence recidivism and treatment of male sex offenders; sexually inappropriate sexual behaviour among elderly people with dementia; and the association between circulating androgen levels and sexual function in women.

Public Health Agency of Canada (2005). Supplement: 2002 Canadian sexually transmitted infections surveillance report. *Canada Communicable Disease Report*, 31S2.

This report from the Public Health Agency of Canada provides the most recent (i.e., up to the end of 2002) epidemiological data on nationally reportable sexually transmitted infections (STI) (chlamydia, gonorrhea, syphilis). Other, very common STI, such as human papillomavirus (HPV) and genital herpes (HSV) are not reportable to public health authorities and therefore are not included in the report. The authors caution that "Many STI are asymptomatic, therefore some infections may go unnoticed, undiagnosed, and unreported" (p. iii) and "among symptomatic individuals, only those who seek testing or medical care will be captured by this passive surveillance system. Because of these limitations, the counts in this report are likely to be an under-estimate of the true burden of disease" (p. iii). In general, this latest report confirms the upward trend in STI rates that began in 1997 and that has continued up to 2002.

Chlamydia remains the most common reportable STI in Canada. National data for chlamydia has been available since 1990 and from that year until its lowest point in 1997, the chlamydia rate had declined steadily.

According to the Public Health Agency, "The picture has changed drastically over the last 5 years. The rate of chlamydia in Canada has reached an all-time high of 179.3 per 100,000 in 2002, compared with 113.9 per 100,000 in 1997" (p. 1). Between 2001 and 2002, the chlamydia rate increased by 11.1%. As in past years, in 2002, the majority of cases were in 15- to 24-year-olds and 69% of reported cases were among females. The highest rate was among 20- to 24-year-old females at 1,376.6 per 100,000 closely followed by 15- to 19-year-old females at 1,362.0. For both groups the rates have increased every year from 1997 to 2002. In 2002, the total chlamydia rates per 100,000 for each of the provinces and territories were as follows: Nunavut (2853.2); Northwest Territories (1448.1); Yukon (468.1); Saskatchewan (362.9); Manitoba (291.7); Alberta (235.6); British Columbia (187.2); New Brunswick (175.0); Nova Scotia (168.5); Quebec (149.3); Ontario; (148.8); Prince Edward Island (105.8); Newfoundland (100.5).

Gonorrhea also reached its lowest rate in Canada in 1997 (14.9 per 100,000) and steadily increased to 2002 (23.5). In 2002, as in past years, the gonorrhea rate was considerably higher in males (29.6) than in females (17.5). Among males, the highest rate was in the 20-24 age group (102.0), followed by those aged 25 to 29 (76.5) and those aged 30 to 39 (55.6). The pattern for females was different. Among females, the highest rate was in the 15 to 19 age group (101.3), followed by those aged 20 to 24 (83.0), and those aged 25 to 29 (36.2). In the more populous provinces (Quebec, Ontario, British Columbia) males account for the majority of reported cases of gonorrhea. For example, in 2002, 84% of the reported cases of gonorrhea were among males. However, in other less populous jurisdictions (Nunavut, Nova Scotia, New Brunswick, Saskatchewan) the rate is higher among females than males. In view of the higher number of reported gonorrhea cases among males compared to females in Canada, the authors



note that "...males are more likely to be symptomatic than females and therefore would be more likely to present to the health care system for diagnosis and treatment" (p. 14).

Infectious syphilis is the least commonly reported STI in Canada. However, like chlamydia and gonorrhea, the syphilis rate has increased since 1997. Specifically, the syphilis rate rose from 0.4 per 100,000 in 1997 to 1.5 per 100,000 in 2002, an increase of 285%. In 2002, the rate of infectious syphilis was considerably higher in males (2.4) than in females (0.6). The highest rate was among males aged 30 to 39 (6.6) followed by males aged 40 to 59 (3.2) and males aged 25 to 29 (2.8). For females, the highest rate was among those aged 25 to 29 (2.6) and 20 to 24 (2.2). Together, Ontario, Quebec, and British Columbia accounted for 94% of all reported cases of syphilis in Canada. In effect, the national syphilis rate is largely driven by specific outbreaks in these three provinces. The authors also note that the disproportionately high number of reported cases of syphilis in males is likely the result of transmission occurring among men who have sex with men.

Rotermann, M. (2005). Sex, condoms and STDs among young people. *Health Reports*, 16, 38-45.

This report from Statistics Canada provides data on sexual intercourse experience, number of sexual partners, condom use at last intercourse, and self-reports of STD diagnosis among 15- to 24-year-old Canadian youth living in private households. The data came from the 2003 Canadian Community Health Survey (CCHS), based on a sample of 18,084 weighted to be representative of the household population aged 15 to 24 in 2003.

Among 15- to 17-year-olds, the percentage who reported that they had experienced sexual intercourse at least once was 28.8% for females and 27.4% for males. For 18- to 19-year-olds the corresponding percentages were 63.8% for females and 65.6% for males. Among 20- to 24-year-olds, the percentages were 81.1% for females and 79.4% for males. Overall, the average age of first sexual intercourse was 16.5 years for both males and females. The percentage of sexually active 15- to 17-year-olds who reported having more than one sexual partner in the

previous year was 41% for males and 29% for females. For the 18- to 19-year-old age group the corresponding percentages were 39% for males and 31% for females and in the 20- to 24-year-old age group the percentages were 35% for males and 23% for females.

Younger respondents were significantly less likely to report that they did not use a condom at last intercourse. For the overall sample, 21.5% of sexually active 15- to 17-year-olds reported that they did not use a condom at last intercourse compared to 32.5% for 18- to 19-year-olds and 43.6% for 20- to 24-year-olds. The author suggests that lower rates of condom use among the older age groups may be because "... long-term relationships with one partner are more common in the older age group and thus condom use is perceived to be less of a concern" (p. 40). For all three age groups, females were significantly more likely than males to report not using a condom at last intercourse. Respondents who reported having one sexual partner in the previous year were less likely to report using a condom at last intercourse compared to those reporting multiple partners.

Among 15- to 24-year-olds who had experienced intercourse at least once in their lives, 4% reported that they had been diagnosed with an STD. The author notes that "Because of the lack of symptoms and/or a lack of awareness, these figures likely represent only a fraction of the actual number of infections in this age group" (p. 41).

Paz-Bailey, G., Koumans, E., Sternberg, M., et al. (2005). The effect of correct and consistent condom use on chlamydial and gonococcal infection among urban adolescents. *Archives of Pediatric and Adolescent Medicine*, 159, 536-542.

The authors begin their research report by noting that females aged 15 to 24 have high rates of chlamydia and gonorrhea and that these infections are among the leading causes of pelvic inflammatory disease, infertility, ectopic pregnancy, and chronic pelvic pain. "Because of the cost and serious sequelae, prevention of these infections is of great public health importance" (p. 536). Although previous research has established that latex condoms provide an effective barrier against both chlamydia and gonorrhea, the degree to which consistency and correctness of



condom use, particularly among adolescents, affects the effectiveness of condoms in infection prevention is largely unknown. The purpose of the Paz Bailey et al. study was to evaluate the relationship between self-reported correct and consistent condom use and the incidence of chlamydial and gonococcal infection among sexually active adolescent women.

The sample for the study consisted of 509 sexually active (i.e., intercourse in the previous three months) females with an average age of 16.6 years attending an adolescent health clinic in Atlanta, Georgia between January 1999 and August 2002. Study participants completed questionnaires that included items on sexual behaviour and condom use. For the purposes of the study, consistent condom use was defined as using a condom for every act of vaginal intercourse in the previous three months. Participants were asked about the occurrence of condom errors such as starting intercourse without a condom and then putting one on, taking the condom off before finishing sex, putting the condom on inside out and then flipping it over and putting it on again, slippage, and breakage. In addition, participants provided urine samples for detection of *C tracomatis* and *N gonorrhoea*.

Most (87%) of the participants reported using a condom at least once in the previous three months. "Only 176 (35%) reported using condoms consistently and 80 (16%) reported consistent use with no condom errors (correct use)" (p. 537-538). Among condom users, 71% reported at least one condom error with starting sex without a condom being the most common error, reported by 43%. Chlamydia was detected in 21% of participants, gonorrhoea in 7%, and 4% had both infections. "The prevalence of chlamydia with no use of condoms, inconsistent use, and consistent use was 25%, 23%, and 16% respectively ($p = .15$); the prevalence of gonorrhoea was 7%, 9%, and 4% respectively ($p = .12$)" (p. 538). None of the condom use errors was significantly associated with either infection except putting on a condom after sex which was associated with chlamydia infection. Overall, the data indicated that correct and consistent condom use reduced the risk of gonorrhoea by 90% and the risk of chlamydia by 60%. In summarizing their findings, the authors state: "We found that consistent and correct use of condoms provided significant protection against both chlamydia and gonorrhoea" (p. 539). In the

concluding discussion of their research report Paz-Bailey et al. note that,

....condoms remain the best STD and HIV prevention approach for persons whose sexual behaviors place them at risk of STDs. Although messages directed at adolescents should encourage delaying initiation of sexual activity, many are already sexually active, and STDs are particularly common among this group. Thus, aggressive condom promotion must remain a key to reducing STDs and HIV (p. 541).

Studer, L.H., Aylwin, A.S., & Reddon, J.R. (2005). Testosterone, sexual offense recidivism, and treatment effect among adult male sexual offenders. *Sexual Abuse: A Journal of Research and Treatment*, 17, 171-181.

There is well established evidence that the hormone testosterone, with its active metabolite dihydrotestosterone, influences both aggression and sex drive in human males. As a result, testosterone is a relevant consideration in the study of sexual offending, sexual offense recidivism, and treatment of sexual offenders. The purpose of the Studer, Aylwin, and Reddon study was to examine the relationship between serum testosterone and sexual violence in a sample of adult male sex offenders attending an in-hospital treatment program.

The final sample for the study consisted of 501 adult male sex offenders voluntarily admitted to the Phoenix Program, an inpatient sex offender treatment program in Edmonton, Alberta. All participants had been convicted of some type of sexual offense (i.e., adult or child victims, intra- or extrafamilial victims, exhibitionism, etc.). Offenses were coded in a range from one (non-contact) to six (severe violence). Serum testosterone samples were collected within the first week of admission into the program. Participants receiving antiandrogenic pharmacotherapy were excluded from the analysis. The program was highly intensive, consisting of 35-40 hours per week of group psychotherapy over a 10-13 month period. For the purposes of the study, recidivism was defined as conviction for a sexual offense after being discharged from the program. As



part of the program's evaluation process, reoffending was tracked through the Canadian Police Information Centre (CPIC). The average length of time from discharge to follow-up through the CPIC was 106.7 months and the authors note that "...the length of the follow-up period is very substantial and long enough to capture a great majority of those offenders who may be prone to reoffend" (p. 175).

In reporting their results, the authors point out that it is estimated that 2.5% of the general male population would be expected to have a serum testosterone level of 30.0 nmol/L or higher (designated in the study as "above normal") whereas 14.5% of the study participants had levels of 30.0 nmol/L or higher. For study participants, there was a clear and statistically significant association between testosterone levels and sexual reoffending. For the combined group of program completers (n=270) and program noncompleters (n=231), "...it was observed that sexual recidivism was demonstrably higher in those with higher serum testosterone" (p. 176). It was also found that higher levels of testosterone were significantly correlated with increased severity (i.e., violence) of reoffending. The authors suggest that this finding seems to indicate that testosterone levels are not just associated with aggression and sex drive as separate entities but also to sexual aggression specifically. When the analyses were conducted separately for those who completed the program and those who did not, an important finding emerged. That is, for those participants who completed the program, higher levels of testosterone was no longer predictive of the likelihood of sexually reoffending. In other words, the intensive group therapy provided by the program "...appears able to intercede in the influence of testosterone on sexually deviant behavior" (p. 171). Studer, Aylwin and Reddon conclude their research report with the following:

We postulate that consistent behavioral changes, which demonstrate a cognitive, overlay, affective restraint, and personal insight into impulses and drives, could account for the amelioration of the predictive influence on recidivism. Perhaps, after all, Plato was correct when he suggested that reason can rule man's passions (p. 179).

Alagiakrishnan, K., Lim, D. Brahim, A., et al. (2005). Sexually inappropriate behaviour in demented elderly people. *Postgraduate Medical Journal*, 81, 463-466.

The authors of this report introduce their study with an important observation:

Sexuality is part of human nature throughout life. Being elderly and sick does not necessarily mean that there is a decline in sexual desire. Patients with dementia may become sexually disinhibited as cognitive deficits progress. Caregivers who are taking care of demented elderly people should expect sexual behaviours to occur and they should be ready to respond appropriately (p. 463).

With this in mind, the purpose of the Alagiakrishnan et al. study was to collect data on problematic sexual behaviour among elderly patients with dementia. The study utilized patient chart data from a long-term care psychiatric consulting service, a community-based geriatric psychiatric centre, and an inpatient dementia behavioural unit, all of which were located in Edmonton, Alberta. A total of 2,278 charts were screened to determine the prevalence, aetiology, and treatment profile for problematic sexual behaviour among patients attending these services.

Overall, the prevalence of sexually inappropriate behaviour was low (1.8%). The average age of cognitively impaired subjects exhibiting inappropriate sexual behaviour was 78 years and 92.7% were male. About half of those exhibiting inappropriate sexual behaviour were living in nursing homes and the rest were living in the community. Among these patients, 54% had vascular dementia, 22% had been diagnosed with Alzheimer's, and 9% had mild cognitive impairment. Among those patients with documented inappropriate sexual behaviours, 87.7% exhibited physically inappropriate behaviour and 67% exhibited verbally inappropriate behaviour. Verbally inappropriate sexual behaviour was more common in the community group (81%) than in the nursing home group (50%). Twenty six (63.4%) had behavioural treatment, 43.8% were taking antipsychotics, 14.6% were being given cholinesterase inhibitors, and 7.3% were receiving hormonal therapy. Behavioural

treatments was more commonly used in the community sample (81%) than in the nursing home sample (45%).

In their discussion, the authors make a number of important points regarding the management of sexually inappropriate behaviour among elderly people with dementia. For example, they point out that inappropriate verbal behaviour such as sexually crude jokes or excessive sexual innuendo among elderly people with dementia do not necessarily signal an intent towards sexually aggressive behaviour and that such patients may not fully comprehend the meaning of what they are saying. In addition, they suggest that,

Inappropriate sexual behaviours are often better managed by non-pharmacological means, as patients may be less responsive to psychoactive therapies. Behavioural therapy includes redirecting the behaviour verbally or if necessary physically. Firmly but gently identify the behaviour and point out that it is unacceptable. Remind the subject who you are, especially if the resident is confused. Exposing and fondling genitals and public masturbation may be minimized by choosing clothing that opens in the back and by assigning manual activities such as folding towels. If behavioural interventions fail, pharmacological therapy may be necessary (p. 465).

Davis, S.R., Davison, S.L., Donath, S. & Bell, R.J. (2005). Circulating androgen levels and self-reported sexual function in women. *Journal of the American Medical Association*, 294, 91-96.

Low desire is the most commonly reported female sexual dysfunction. Although it is clear that levels of sexual desire and arousal are impacted by a multitude of health and psychosocial factors, it has been increasingly assumed that endogenous androgen levels are significant and independent determinants of female sexual behaviour. In addition, several studies have reported that some women with clinically defined hypoactive sexual desire disorder have benefited from testosterone supplementation. However, as Davis et al. point out, "...evidence that a low serum testosterone level distinguishes women with low

sexual function from others, and that androgen deficiency syndrome can be defined biomedically, is lacking" (p. 91). Thus, the purpose of the author's study was to examine whether women reporting low sexual desire and low sexual satisfaction were more likely to have low serum androgen levels than women without low desire or low satisfaction.

The final sample of the study consisted of 1,021 women aged 18 to 75 randomly selected from the electoral roll data base for Victoria, Australia. Participants completed the Profile of Female Sexual Function (PFSF) and provided blood samples tested for serum levels of total and free testosterone, androstenedione, and dehydroepiandrosterone sulfate (DHEAS). The PFSF measures desire, arousal, orgasm, pleasure, sexual concerns, responsiveness, and self image. The authors used receiver operating characteristic curves (ROC) to determine if each of the androgens was useful for discriminating between women with and without low sexual desire.

The ROC analysis indicated that levels of total or free testosterone were not useful for discriminating between younger (18 to 44) or older (45-75) women with or without low sexual functioning for any of the seven domains of the PFSF. However for older women, a lower domain score for sexual responsiveness was associated with higher odds of having DHEAS levels below the 10th percentile for that age group and for younger women, low scores on the desire, arousal and responsiveness domains were associated with higher odds of having DHEAS levels below the 10th percentile for that age group. Nevertheless, the authors are careful to point out that although there was a statistical association between DHEAS and sexual function, most of the women with low DHEAS did not have low sexual function.

In discussing the finding that it was DHEAS, not free testosterone, that showed some association with sexual function, Davis et al. suggest that,

This is most likely due to differing circulating levels of these steroids and the complexity of androgen metabolism. DHEA is the most abundant sex steroid in women and circulating DHEA and its sulfate, DHEAS, provide a large precursor reservoir for the intracellular



production of both estrogens and androgens (p. 95).

The authors also suggest that their findings do not necessarily conflict with the use of testosterone supplementation in the effective treatment of some women with hypoactive sexual desire disorder. However, Davis et al. do suggest that,

...our data, taken together with what is already known about the endocrine physiology, suggest that sex steroids influence female sexual function, but that there is no serum androgen level that defines female androgen insufficiency. The measurement of serum testosterone, free testosterone, or DHEAS in individuals presenting with low sexual function is not informative and levels of these hormones should not be used for the purpose of diagnosing androgen insufficiency in women (p. 96).



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SEXUALITY AND SUBSTANCE USE: THE IMPACT OF TOBACCO, ALCOHOL, AND SELECTED RECREATIONAL DRUGS ON SEXUAL FUNCTION

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INTRODUCTION

Throughout history and across cultures, alcohol, recreational drug use and sexuality have been closely intertwined. For example, alcohol has often been considered to be "... a powerful facilitator, promoter, disinhibitor, and common accompaniment to sexual behavior of all types" (Rosen, 1991, pp. 120-121). Smoking cigarettes has traditionally been associated with glamour, sophistication, and enhanced attractiveness. Advertising often attempts to associate alcohol consumption with sexual attractiveness. Many recreational drugs are thought to be aphrodisiacs and, in general, substance use is often considered a facilitating social-psychological prelude to sexual activity. While there is a well established mythology concerning the relationship between sexuality and substance use, many people, including educators and health professionals, are less certain about the current medical/scientific understanding of the impact of substance use on sexual functioning.

The purpose of this article is to review and summarize the available scientific literature on the impact of

consumption of tobacco, alcohol, methamphetamine, cocaine, and marijuana on sexual function. The findings of the available research are summarized in the context of the immediate impact of consumption and in terms of the impact that may result from regular, prolonged use of a substance. This information will be of general interest but also of particular use to educators and counsellors in assisting their students and clients in making informed decisions about substance use.

A presentation and discussion of research on the impact of substance use on sexuality must take into account several important considerations. First, many substance users report a beneficial impact of consumption on sexuality (e.g., increased sexual enjoyment). Second, all the substances considered in this review are, or can be highly addictive and prolonged, heavy consumption of these substances carries a high risk for significant detrimental health outcomes. Third, this important reality should be factored into informed decision-making around substance use. Fourth, it should be noted explicitly that with the exception of tobacco and alcohol,



possession of the substances considered in this article is illegal.

There are some significant methodological issues in the research on this topic which should induce a good measure of caution in drawing definitive conclusions about the impact of substance use on sexuality. First, the impact of specific substances on specific phases of human sexual response has, in many cases, not been directly examined in the scientific literature. Second, as other reviewers have noted, much of the research in this area relies on small, non-representative samples and self-report data from volunteer users obtained in uncontrolled studies (Peugh & Belenko, 2001). It has also been noted that the effects of substance use on sexuality are mediated by, among other things, dosage level, duration of use, history and characteristics of the user, and the social/environmental context in which the substance is consumed (Rosen, 1991). With these caveats in mind, there is sufficient research available to make some observation, albeit tentatively, about the impact of different substances on sexual function.

THE CONCEPTUAL DISTINCTION BETWEEN SEXUAL ENJOYMENT/PLEASURE AND THE AROUSAL AND ORGASM PHASES OF SEXUAL RESPONSE: AN IMPORTANT CONSIDERATION IN THE EXAMINATION OF THE IMPACT OF SUBSTANCE USE ON SEXUALITY

In order to clearly articulate the known impact of substances on sexual function it is important to clarify what is meant by sexual function and its relationship to sexual pleasure/enjoyment. Various models of human sexual response have been proposed (Hyde, Delameter & Byers, 2004). These models range, in terms of complexity, from very basic to highly intricate. In reality, human sexual response is multifaceted and subject to a wide array of biological, psychological, contextual, and interpersonal factors (Basson, 2004). The degree to which the substances considered in this review interact with each of the interconnected facets of human sexual response is largely unknown. Thus, for the purposes of this review, Kaplan's (1979) triphasic model of sexual response will be used to delineate, in basic terms, current medical/scientific understandings of how different substances impact upon key parameters of sexual response. Most sexologists agree that healthy sexual functioning is dependant to some degree on

the three components of the triphasic model of sexual function: desire, arousal, orgasm (Winze & Carey, 2001) (Table 1). This review identifies the phases of sexual response that research suggests are impacted by each of the substances considered.

Table 1 Triphasic Model of Sexual Response

Desire: An interest in sexual activity which leads an individual to initiate sex or be receptive to it.

Arousal: Typically includes feelings of sexual excitement accompanied by erection in the male and vaginal lubrication in the female.

Orgasm: Usually includes a series of pelvic muscle contractions that release sexual tension.

SEXUALITY AND SUBSTANCE USE: GENERAL OBSERVATIONS

As described below, many new or infrequent users of some substances report that they increase desire (i.e., they are aphrodisiacs) (e.g., alcohol, methamphetamine, cocaine, marijuana) or that they enhance sexual pleasure/enjoyment (e.g., methamphetamine, cocaine, marijuana). For the purposes of this review it is important to note that feelings of sexual enjoyment and pleasure are highly subjective and thus are quite likely to be influenced by the psychoactive agents in recreational drugs. However, commonly used recreational drugs, in their modes of action, do not typically directly target the arousal or orgasm phases of sexual response. (By way of contrast, the drug sildenafil [Viagra], in its mode of action, does target the arousal phase of sexual response by directly facilitating erection). This distinction is of particular importance in the assessment of the impact of substance use on sexuality. One of the potential pitfalls of drug use is that the initial perception of sexual benefits from consuming a drug may contribute to prolonged and/or heavy use which in turn may have a significant negative impact on health and thereby on sexual function which in turn may make sexual pleasure/enjoyment difficult to achieve. As the authors of a previous literature review on alcohol, drugs, and sexual function noted,



The available research does suggest that alcohol and most drugs often have deleterious acute and chronic effects on normal sexual functioning. A moderate alcohol drinker or a new user of some illicit drugs may experience sexual pleasure or facilitation connected to their substance use. However, with higher doses and long-term use, alcohol and drugs can impair sexual response, reduce sexual desire, and contribute to sexual dysfunction (Peugh & Belenko, 2001, pp. 229-230).

Based on the research reviewed in this article, Table 2 provides an overview of the potential negative impact on sexual function of prolonged regular use of selected substances. In addition to the cautions about the methodology and reliability of available research on the effects of illicit drugs on sexuality noted above, readers should also be aware that in many cases, there is insufficient research to draw even very tentative conclusions about the impact of a particular substance on sexual function. For example, although there is a considerable and growing body of research on the impact of smoking on male erectile function, there has been little or no research that has attempted to investigate the impact of smoking on female sexual function. Thus, our assumptions about this and other under-investigated effects of substance use on sexual function remain speculative.

Table 2 Potential Negative Impact of Prolonged, Regular Use of Selected Substances on Sexual Response

	Desire	Arousal	Orgasm
Tobacco		✓×	
Alcohol	✓×*	✓*	✓*
Methamphetamine		✓	
Cocaine	✓	✓×	✓
Marijuana			✓0

- ✓ = Research suggests substance impairs phase of sexual functioning
 × = Impact is demonstrated in males only
 * = Impact is evident only when high doses are consumed
 0 = Some evidence to suggest a negative impact but contrary evidence exists as well

TOBACCO

In terms of the short-term impact of smoking on sexual function, it is important to understand that the physical aspects of the arousal phase of the human sexual response cycle (i.e., erection in the male, swelling of the clitoris and genital engorgement in the female) requires efficient blood flow to the genital area and that smoking reduces the efficiency of blood circulation throughout the body. In men, smoking interferes with several important biological processes that are needed to produce and maintain an erection (e.g., vasodilation, corporal smooth muscle relaxation) (Celermajer, Sorenson, Georakopoulos et al., 1993; Powell, 1998). In short, smoking impairs the function of the blood vessels that are needed to get and keep an erection (Virag, Bouilly, & Frydman 1985). A population-based study of men in Finland found that cigarette smoking was clearly linked to a higher risk of erectile dysfunction (Shiri, Koskimaki, Hakama et al., 2004). A study that measured the hardness and number of minutes that erections lasted while men slept (nocturnal penile tumescence), found that the more cigarettes a man smoked during the day, the less rigid and long lasting his erections were at night (Hirshkowitz, Arcasoy, Karacan et al., 1992). In another study of heavy smoking men, not smoking for 24 hours resulted in harder erections (Guay, Perez, & Heatly, 1998). In sum, it is clear that smoking can have an immediate negative impact on a man's ability to get and maintain a strong erection and that not smoking, even for a short period of time, can improve erectile capacity. While there has been no research on the direct impact of smoking on female sexual function, it is clear that smoking reduces blood flow to the lower extremities, and to the extent that female sexual arousal involves blood flow to the genital area, it is logical to speculate that cigarette smoking may inhibit physical sexual arousal among women.

There is abundant evidence linking long-term cigarette smoking with an increased risk of erectile dysfunction in men (Derby, Mohr, Goldstein et al., 2000; Feldman, Johannes, Derby et al., 2000; Mannino, Klevens, & Flanders, 1994). A recent study found that the higher the number of cigarettes smoked per day and the higher the number years a man has smoked, the higher his risk of developing erectile dysfunction (Gades, Nehra, Jacobson et al., 2005). Although the long-term impact of smoking on



erectile function is more clearly evident in older men, there is also evidence linking heavy smoking to erectile dysfunction in men under age 45 (Natali, Mondaini, Lombardi et al., 2004).

As noted above, not smoking, even for short periods of time, can improve sexual function. In addition, there is evidence that quitting smoking can have a longer-term benefit on sexual function. A study of male smokers with erectile dysfunction, found that compared to those who did not quit, those who quit were more likely to show improvements in erectile function one year later (Pourmand, Alidaee, Rasuli et al., 2004). As also noted above, there has been no published research on the impact of smoking on female sexual function. However, it is well known that long-term, heavy smoking has a negative impact on many aspects of women's health and so it is quite likely that smoking may also have a negative impact on the physiological aspect of female sexual function. For both men and women, smoking is most likely to have an impact of the arousal phase of the human sexual response cycle.

ALCOHOL

In Western culture, alcohol and sexual behaviour are frequently linked. Put another way, the consumption of alcohol often socially facilitates and precedes sexual activity and it is commonly believed that alcohol is a powerful sexual facilitator and disinhibitor that potentially acts as an aphrodisiac (Rosen, 1991). Although some of these beliefs are held by many people, they are, in some cases, incorrect. While alcohol consumption can act as an disinhibitor, leading some people to be more open or receptive to sexual activity, the belief that alcohol acts as an aphrodisiac, increasing or intensifying sexual response is false. Indeed, as summarized below, alcohol typically has the opposite effect in that consuming alcohol, particularly in large quantities, tends to dampen sexual response.

Consuming small amounts of alcohol is unlikely to have an immediate short-term impact on a person's sexual response. However, it is clear that as the amount of alcohol a person consumes increases, their physiological ability to respond sexually decreases. With respect to its impact on the sexual response cycle, it is important to understand that alcohol is a

central nervous system depressant that slows down brain functioning, respiration, and circulation (Peugh & Belenko, 2001). As a result, alcohol's depressive effect on the central nervous system can, particularly in higher doses, lead to, for example, erectile dysfunction in men and decreased vaginal lubrication in women (Peugh & Belenko, 2001).

Several studies measuring the impact of alcohol on male sexual response have found that small amounts (e.g., one drink) have little or no impact, but that in larger doses, alcohol impairs a male's ability to become aroused and ejaculate (Farkas & Rosen, 1976; Rubin & Henson, 1976). Similarly, studies of women have shown that larger doses of alcohol reduce the ability to become aroused and experience orgasm (Malatesta, Pollack, Crotty, & Peacock, 1982; Wilson, Lawson, 1978). In sum, the available scientific research indicates that higher amounts of alcohol intake have an immediate short-term negative impact on the arousal and orgasm phases of the human sexual response cycle.

Recent research suggests that moderate (1 to 2 drinks per day) long-term alcohol consumption may have beneficial effects on cardiovascular health (De Lange & van de Wiel, 2004). To the extent that light drinking is a component of a heart healthy lifestyle, it is unlikely to interfere with the circulatory-cardiovascular aspects of sexual response.

While light or moderate alcohol consumption may be compatible with a healthy lifestyle, long-term heavy or binge drinking impacts negatively on every organ system in the body including those associated with sexual response and reproduction (Crenshaw & Goldberg, 1996; Peugh & Belenko, 2001). Studies of alcoholics have shown that alcohol consumption can enable some people to overcome sexual inhibitions or feelings of inadequacy (Pinhas, 1980). However, there is a large body of evidence indicating that long-term alcohol abuse is associated with a range of sexual problems and dysfunctions. For women these include difficulties with lubrication, inhibited orgasm, and painful sex (Johnson, Phelps, & Cottler, 2004; Peugh & Belenko, 2001). For men, they include erectile dysfunction, inhibited orgasm, and hypoactive sexual desire disorder (low desire) (Rosen, 1991). In sum, the available scientific



literature indicates that heavy drinking can have a long-term negative impact on the arousal and orgasm phases of the human sexual response cycle for women and on the desire, arousal, and orgasm phases for men.

METHAMPHETAMINE (CRYSTAL METH)

Methamphetamine is a white, odourless crystalline powder that is swallowed, smoked, snorted or injected. Methamphetamine is a highly addictive, powerful central nervous system stimulant that promotes the release of the neurotransmitters dopamine, serotonin noradrenaline and adrenaline (Seiden, Sobol, & Ricaurte, 1993). The immediate effects of methamphetamine use include increased energy, alertness, and sociability as well as feelings of euphoria which may last from 6 to 30 hours (Anglin, Burke, Perrochet et al., 2000). However, these positive effects are often accompanied by, among other things, elevated blood pressure, heart rate and body temperature as well as anxiety, irritability, insomnia, aggressiveness and, in some cases, paranoia or suicidal tendencies (Maxwell, 2005). Overdosing methamphetamine can result in cerebral hemorrhage, stroke, seizure, hyperthermia, arrhythmias, coma, and death (Freese, Miotto, & Reback, 2002).

With respect to the effects of this drug on sexual function, many users report that methamphetamine acts as an aphrodisiac (increasing sexual desire), reduces sexual inhibitions, and increases sensation (Degenhardt & Topp, 2003; Semple, Patterson, Grant, 2002). Among so-called "club drugs" or "party drugs," methamphetamine is the most strongly associated with sexuality and sexual behaviour. In particular, it is the combination of increased social confidence, loss of sexual inhibitions, and heightened physical sensation that underlies the perceived sexual enhancement effects of methamphetamine (Kurtz, 2005). Methamphetamine does not independently or directly target specific aspects of the sexual response cycle. Rather, because methamphetamine is a powerful nervous system stimulant, it enhances a person's general sense of well-being and excitement which, as a result, is likely to intensify and enhance sexual experiences. Methamphetamine is highly addictive in-and-of-itself. But the use of methamphetamine to lower sexual inhibitions and

enhance sexual experiences can also lead to a dependency on the drug.

There is no direct connection between the use of crystal meth and sex. As a stimulant, however, meth stimulates your libido as well as anything else. And crystal can increase your self-confidence and lower your inhibitions. It also enhances sensation. If one uses crystal in a sexually charged situation, the effect will be heightened. Because of this, people mistakenly believe that crystal caused the sexual feelings. It is indeed a very potent mixture. For many people, sex under the influence of meth rapidly leads to an incredibly strong association between the two which is hard to break. One without the other becomes inconceivable (San Francisco AIDS Foundation; www.thebody.com).

In sum, with respect to its short-term impact on sexual function, while methamphetamine does not exert direct, specific effects on the phases of the sexual response cycle *per se*, as a central nervous system stimulant, it generally enhances or intensifies feelings, desires, and sensations, including those related to sexuality.

Long-term use of methamphetamine is associated with increased risk for stroke, cardiac valve sclerosis, heart attack, reduced lung function, pulmonary hypertension, psychosis and paranoia, reduced cognitive functioning and poor mental health, including depression (Maxwell, 2005). Paradoxically, while the initial use of methamphetamine may have perceived benefits for sexual function, prolonged use of amphetamine-based drugs has been associated with erectile dysfunction and delayed ejaculation in men as well as delayed orgasm in women (Peugh & Belenko, 2001). In addition, prolonged use of methamphetamine is strongly associated with a condition called "crystal dick" in which the user maintains a strong libido, has high energy and lowered sexual inhibitions, but is unable to get an erection (Frosch, Shoptaw, Huber et al., 1996).

An assessment of the impact of methamphetamine on sexuality must take account of the strong



association between consumption of the drug and increases in unsafe sexual behaviour that places methamphetamine users at high risk for HIV and other STI. This association is explained by, among other things, the combination of lowered sexual inhibitions, high libido and energy, problems with sexual functioning (i.e., "crystal dick"), and the highly charged sexual context in which methamphetamine use often takes place. Together these factors seem to increase the likelihood for gay, bisexual, and heterosexual methamphetamine users to engage in very high HIV/STI sexual risk behaviours (Frosch, Shoptaw, Huber et al., 2004; Hirschfield, Remien, Walavalker, & Chiasson, 2004; Semple, Patterson, & Grant, 2004).

COCAINE, CRACK COCAINE

Cocaine is derived from the coca plant, the leaves of which are processed into a white powder that can be snorted or melted and then injected. Crack is a further processed form of cocaine that is smoked. The effects of crack are immediate, intense (comparable to injection), but do not last long. In general, cocaine is a central and peripheral nervous system stimulant. In particular, cocaine inhibits the reuptake of the neurotransmitter dopamine (Mateo, Budygin, Morgan et al., 2004). The buildup of dopamine that results from reuptake inhibition sends pleasurable sensations along the neural pathways, leading to feelings of well-being, self-confidence, and alertness. Ingestion of cocaine has an immediate impact on the cardiovascular system, including increased heart rate, which elevates the user's risk for heart attack and sudden death (Frishman, Del Vecchio, Sanal, & Ismal, 2003).

Cocaine does not directly or specifically impact on the human sexual response cycle. However, like for other nervous system stimulants, the feelings of well being that result from taking the drug may intensify, spark, or enhance feelings of sexual desire and sensuality. Often, new or infrequent cocaine users report that cocaine has beneficial sexual effects, most notably in increasing desire (Peugh & Belenko, 2001). In several studies, male cocaine users reported that use of the drug delayed ejaculation (Rosen, 1991).

Regular or long term use of cocaine that is snorted, injected, or smoked (crack) has significant negative

effects on overall health and on sexual functioning. Cocaine use increases an individual's risk of a variety of cardiovascular conditions including heart attack, sudden death, arrhythmia (irregular heart beat), and cardiomyopathy (Frishman et al., 2003). Chronic cocaine use has been found to be associated with reduced cognitive functioning even after the user has stopped using the drug (Bolla, Rothman, & Cadet, 1999). In addition, infants whose mothers use cocaine during pregnancy have a significantly increased risk for delayed mental/cognitive development (Singer, Arendt, Minnes et al., 2002).

While new or infrequent cocaine users may perceive a beneficial impact on sexuality, it is clear that regular or long term use of cocaine is likely to have a negative impact on sexual function. In one study of regular cocaine users, 66% of men who had been using the drug for one year or longer reported that they had difficulty getting erections (MacDonald, Waldorf, Reinerman, & Murphy, 1988). It is common for regular cocaine users to also be heavy drinkers of alcohol. In a study of men who were dually addicted to alcohol and cocaine, 62% reported low sexual desire, 52% reported erectile dysfunction, and 30% experienced delayed ejaculation (Cocores, Miller, Pottash, and Gold, 1988).

Several studies have assessed the impact on sexuality of crack cocaine. A study of male and female crack users found that 57% experienced diminished desire and 63% reported a decreased ability to have an orgasm (37% reported that crack increased their desire and 24% said that it increased their ability to have an orgasm) (Wetherby, Shultz, Chitwood et al., 1992). Contrary to the notion that crack cocaine may act as an aphrodisiac for women, a study of female crack cocaine users found that the drug diminished sexual desire and increased the likelihood of sexual dysfunction (Henderson, Boyd, & Whitmarsh, 1995).

New users of cocaine may perceive a sexual benefit to taking the drug and this effect is likely the result of the overall feelings of well being, confidence, and energy that are associated with the drug. However, it is clear the regular use of cocaine is not only detrimental to various aspects of health, it can negatively impact on the desire, arousal, and orgasm phases of the sexual response cycle.



MARIJUANA

Marijuana is the most commonly used illicit drug in the world. Marijuana and hashish are derived from the hemp plant *cannabis sativa*. The main psychoactive ingredient in marijuana that results in the user becoming "stoned" is delta-9-tetrahydrocannabinol (THC) which produces mild euphoria, relaxation and a general enhancement of sensory experiences (Adams & Martin, 1996). Smoking marijuana can increase heart rate, increase or decrease blood pressure, and, in some users it produces anxiety or panic attacks.

In the many different cultures where it is consumed, marijuana is often associated with sexual enhancement. However, it is important to note that there has been no direct scientific investigation of the impact of cannabis consumption on the physiological components of the sexual response cycle. It is likely that, similar to the effects of other drugs on sexuality, the general feelings of relaxation and sensory enhancement that often result from smoking marijuana carry over into sexual activity.

Survey and interview research conducted with marijuana smokers suggest that many users perceive that it has a beneficial impact. In the most extensive study of the sexual effects of regular marijuana smoking, most users reported that the drug did not impact on sexual function per se (e.g., increased number of orgasms, or the ability to prolong sexual activity) but 76% of female users and 70% of male users reported that marijuana increased sexual pleasure and satisfaction and 58% of males and 36% of females reported that smoking marijuana enhanced the quality of orgasm (Halikas, Weller, & Morse, 1982). Most marijuana users in this study also reported that it had mild aphrodisiac effects. Other studies have indicated similar effects in that marijuana is perceived by the user to enhance sexual enjoyment but has little or no effect on performance/function (i.e., erection, lubrication, etc.) (Crenshaw & Goldberg, 1996).

The available research suggests that the perceived enhancement of sexual activity from the use of marijuana can be affected by a number of factors. For example, the perceived positive effects are less likely to occur as increasing amounts of the drug's

psychoactive properties are consumed. In one survey, most users (59%) believed that sexual enjoyment was increased after smoking one joint whereas less than half (39%) believed that the sexual experience would be enhanced after two or more joints were smoked (Koff, 1974). It has also been suggested that the enhancement of sexual experiences from marijuana use can be affected by the expectations of users, the setting, personality type, age, and relationship status of the users (Crenshaw & Goldberg, 1996; Rosen, 1991).

The health implications of regular, long-term marijuana use is not well understood and is the subject of considerable debate. However, it should be noted that there is medical research suggesting an association between marijuana smoking and an increased risk for medical conditions including bronchitis, emphysema, lung cancer, impaired immune function, and damage to the reproductive system (Hall & Solowij, 1998; Khalsa, Genser, Francis, & Martin, 2002).

The impact of long-term marijuana use on sexuality and sexual function is unclear as the very limited research on this topic has produced apparently conflicting results. For example, in one study marijuana users were extensively interviewed about their experiences with the drug, then re-interviewed 6 to 8 years later, with the results suggesting that the perceived sexual enhancement associated with marijuana that many experienced at the first interview was maintained at the second interview (Haikas, Weller, Morse, & Hoffmann, 1985). However, another study examining the possible association between substance use and sexual dysfunction found that inhibited orgasm and painful sex among women was independently associated with prior marijuana use (Johnson, Phelps, & Cottler, 2004).

In sum, based on the limited available research on the impact of marijuana smoking on sexuality, it would appear that although the psychoactive properties of marijuana do not directly target the phases of sexual response, smoking the drug may indirectly increase desire in some users. The frequently reported general enhancement of sensory experience felt by many marijuana users may contribute to the sense that sexual activity while under the influence of the drug is more pleasurable.



Research on the long-term impact of marijuana is severely limited but there is preliminary evidence to suggest that regular marijuana use may negatively impact on the orgasm phase of sexual response among some women.

References

- Adams, I.B., & Martin, B.R. (1996). Cannabis: Pharmacology and toxicology in animals and humans. *Addiction, 91*, 1585-1614.
- Anglin, M.D., Burke, C., Perrochet, B. et al. (2000). History of the methamphetamine problem. *Journal of Psychoactive Drugs, 32*, 137-141.
- Basson R. (2004). Recent advances in women's sexual function and dysfunction. *Menopause, 11*, 714-725.
- Bolla, K.I., Rothman, R., & Cadet, J.L. (1999). Dose-related neurobehavioral effects of chronic cocaine use. *Journal of Neuropsychiatry and Clinical Neuroscience, 11*, 361-369.
- Celermajer, D.S., Sorenson, K.E., Georakopoulos, D., et al. (1993). Cigarette smoking is associated with dose-related and potentially reversible impairment of endothelium-dependent dilation in healthy young adults. *Circulation, 88*, 2149-2155.
- Cocores, J.A., Miller, N.S., Pottash, A.C., & Gold, M.S. (1988). Sexual dysfunction in abusers of cocaine and alcohol. *American Journal of Alcohol and Drug Abuse, 14*, 169-173.
- Crenshaw, T.L., & Goldberg, J.P. (1996). Alcohol. In *Sexual Pharmacology: Drugs That Affect Sexual Functioning*, (pp. 151-170). New York, NY: W.W. Norton & Company.
- Crenshaw, T.L., & Goldberg, J.P. (1996). Marijuana and other illegal drugs. In *Sexual Pharmacology: Drugs That Affect Sexual Functioning* (pp. 189-193). New York, NY: W.W. Norton & Company.
- De Lange, D.W., & van de Wiel, A. (2004). Drink to prevent: Review on the cardioprotective mechanisms of alcohol and red wine polyphenols. *Seminars in Vascular Medicine, 4*, 173-186.
- Degenhardt, L., & Topp, L. (2003). "Crystal meth" use among polydrug users in Sydney's dance party subculture: Characteristics, use patterns and associated harms. *International Journal of Drug Policy, 14*, 17-24.
- Derby, C.A., Mohr, B.A., Goldstein, I. et al. (2000). Modifiable risk factors and erectile dysfunction; can lifestyle changes modify risk? *Urology, 56*, 302-306.
- Farkas, G., & Rosen, R.C. (1976). Effects of alcohol on elicited male sexual response. *Journal of Studies on Alcohol, 37*, 265-272.
- Feldman, H.A., Johannes, C.B., Derby, C.A. et al. (2000). Erectile dysfunction and coronary risk factors: prospective results from the Massachusetts Male Aging Study. *Preventive Medicine, 30*, 328-338.
- Freese, T.E., Miotto, K., & Reback, C.J. (2002). The effects and consequences of selected club drugs. *Journal of Substance Abuse Treatment, 23*, 151-156.
- Frishman, W.H., Del Vecchio, A., Sanal, S., & Ismal, A. (2003). Cardiovascular manifestations of substance abuse part 1: Cocaine. *Heart Disease, 5*, 187-201.
- Frosch, D., Shoptaw, S., Huber, A. et al. (1996). Sexual HIV risk among gay and bisexual male methamphetamine abusers. *Journal of Substance Abuse Treatment, 13*, 483-486.
- Gades, N.M., Nehra, A., Jacobson, D.J. et al. (2005). Association between smoking and erectile dysfunction: A population-based study. *American Journal of Epidemiology, 161*, 346-351.
- Guay, A., Perez, J., & Heatly, G. (1998). Cessation of smoking rapidly decreases erectile dysfunction. *Endocrine Practice, 4*, 232-26.
- Haikas, J.A., Weller, R.A., Morse, C.L., & Hoffmann, R.G. (1985). A longitudinal study of marijuana effects. *International Journal of Addiction, 20*, 701.
- Halikas, J., Weller, R., & Morse, C. (1982). Effects of regular marijuana use on sexual performance. *Journal of Psychoactive Drugs, 14*, 59-70.
- Hall, W., & Solowij, N. (1998). Adverse effects of cannabis. *The Lancet, 352*, 1611-1616.
- Henderson, D.J., Boyd, C.J., & Whitmarsh, J. (1995). Women and illicit drugs: Sexuality and crack cocaine. *Health Care for Women International, 16*, 113-124.



- Hirshfield, S., Remien, R.H., Walavalkar, I., & Chiasson, M.A. (2004). Crystal methamphetamine use predicts incident STD infection among men who have sex with men recruited online: A nested case-control study. *Journal of Medical Internet Research*, 29, e41.
- Hirshkowitz, M., Arcasoy, M., Karacan, J. et al. (1992). Nocturnal penile tumescence in cigarette smokers with erectile dysfunction. *Urology*, 39, 101-107.
- Hyde, J.S., Delameter, J.D., & Byers, E.S. (2004). *Understanding Human Sexuality: Second Canadian Edition*. Toronto, ON: McGraw-Hill Ryerson.
- Johnson, S.D., Phelps, D.L., & Cottler, L.B. (2004). The association of sexual dysfunction and substance use among a community epidemiological sample. *Archives of Sexual Behavior*, 33, 55-63.
- Kaplan, H.S. (1979). *Disorders of Sexual Desire*. New York, NY: Simon and Shuster.
- Khalsa, J.H., Genser, S., Francis, H., & Martin, B. (2002). Clinical consequences of marijuana. *Journal of Clinical Pharmacology*, 42, 7S-10S.
- Koff, W.C. (1974). Marijuana use and sexual activity. *Journal of Sex Research*, 10, 194-204.
- Kurtz, S.P. (2005). Post-circuit blues: Motivations and consequences of crystal meth use among gay men in Miami. *AIDS and Behavior*, 9, 63-72.
- MacDonald, P.T., Waldorf, D., Reinerman, C., & Murphy, S. (1988). Heavy cocaine use and sexual behavior. *Journal of Drug Issues*, 18, 437-455.
- Malatesta, V.J., Pollack, R.H., Crotty, T.D., & Peacock, L.J. (1982). Acute alcohol intoxication and female orgasmic response. *The Journal of Sex Research*, 18, 1-17.
- Mannino, D.M., Klevens, R.M., & Flanders, W.D. (1994). Cigarette smoking: An independent risk factor for impotence? *American Journal of Epidemiology*, 140, 1003-1008.
- Mateo, Y., Budygin, E.A., Morgan, D. et al. (2004). Fast onset of dopamine uptake inhibition by intravenous cocaine. *European Journal of Neuroscience*, 20, 2838-2842.
- Maxwell, J.C. (2005). Emerging research on methamphetamine. *Current Opinion in Psychiatry*, 18, 235-242.
- Natali, A., Mondaini, N., Lombardi, G. et al. (2004). Heavy smoking is an important risk factor for erectile dysfunction in young men. *International Journal of Impotence Research*, 17, 227-330.
- Peugh, M.A., & Belenko S. (2001). Alcohol, drugs, and sexual function: a review. *Journal of Psychoactive Drugs*, 33, 223-232.
- Pinhas, V. (1980). Sex guilt and sexual control in women alcoholics in early sobriety. *Sexuality and Disability*, 3, 256-271.
- Pourmand, G., Alidaee, M.R., Rasuli, S. et al. (2004). Do cigarette smokers with erectile dysfunction benefit from stopping?: A prospective study. *BJU International*, 94, 1310-1313.
- Powell, J.T. (1998). Vascular damage from smoking: Disease mechanisms at the arterial wall. *Vascular Medicine*, 3, 21-28.
- Rosen, R.C. (1991). Alcohol and drug effects on sexual response: human experimental and clinical studies. *Annual Review of Sex Research*, 2, 119-179.
- Rubin, H.B., & Henson, D.E. (1976). Effects of alcohol on male sexual responding. *Psychopharmacology*, 47, 123-134.
- San Francisco AIDS Foundation. Frequently asked questions about crystal methamphetamine. *AIDS Hotline.org*. www.thebody.com.
- Seiden, L., Sobol, K., & Ricaurte, G. (1993). Amphetamine: Effects on catecholamine systems and behavior. *Annual Reviews of Pharmacology and Toxicology*, 33, 639-674.
- Semple, S.J., Patterson, T.L., & Grant, I. (2002). Motivations associated with methamphetamine among HIV-positive men who have sex with men. *Journal of Substance Abuse Treatment*, 22, 149-156.
- Semple, S.J., Patterson, T.L., & Grant, I. (2004). The context of sexual risk behavior among heterosexual methamphetamine users. *Addictive Behaviors*, 29, 807-810.



- Shiri, R., Koskimaki, J., Hakama, M. et al. (2004). Effect of life-style factors on incidence of erectile dysfunction. *International Journal of Impotence Research*, 16, 389-394.
- Singer, L.T., Arendt, R., Minnes, S. et al. (2002). Cognitive and motor outcomes of cocaine-exposed infants. *Journal of the American Medical Association*, 287, 1952-1960.
- Virag, R., Bouilly, P., & Frydman, D. (1985). Is impotence an arterial disorder? A study of arterial risk factors in 440 impotent men. *Lancet*, 1, 181-184.
- Wetherby, N.L., Shultz, J.M., Chitwood, D.D. et al. (1992). Crack cocaine use and sexual activity in Miami, Florida. *Journal of Psychoactive Drugs*, 24, 373-380.
- Wilson, G.T., & Lawson, D.M. (1978). Expectancies, alcohol, and sexual arousal in women. *Journal of Abnormal Psychology*, 87, 358-367.
- Winze, J.P., & Carey, M.P. (2001). *Sexual Dysfunction: A Guide for Assessment and Treatment*. 2nd Edition. New York, NY: Guilford Publications.

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