

# The Infectious Disease Syndemics of Crack Cocaine

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## Abstract

Compared to the users of other illicit drugs, mortality rates among crack cocaine users have been found to be conspicuously high. Several factors contribute to this pattern. This article argues that one of the primary factors involved in drug-related deaths among crack cocaine users is the role this drug plays in fostering infectious disease syndemics, including adverse interactions among HIV, TB, and various STIs. Notably, syndemic disease interactions like the kinds described in this manuscript are promoted by social conditions of marginalization, poverty, and living in stressful and often traumatic social conditions.

Keywords: infectious disease, syndemics, crack cocaine, HIV, tuberculosis, sexually transmitted diseases

## Introduction

Illicit drug use, directly or indirectly, is involved in about 1.5% of deaths in the U.S. annually (Heron & Smith 2007). Worldwide, it is estimated that in the year 2000 there were over 200,000 deaths attributable to the use of illicit drugs (Degenhardt et al., 2004). As Mokdad et al. (2004) note, however, “Several studies have reported an undercount of the number of deaths attributed to drugs by vital statistics...” (p. 1242). Notably, illicit drug use is an especially significant cause of premature mortality among young adults (Degenhardt et al., 2004). Further, analyses of vital statistics suggest that fatalities ultimately associated with the use of illicit drugs have a variety of immediate causes, including self-inflicted and street violence, vehicular injury, drug overdose, chronic diseases, and infectious diseases. Although drug-related death statistics involve numerous illicit drugs, mortality rates among crack cocaine users have been found to be conspicuously high.

In a longitudinal study of over 1600 HIV-positive women enrolled in the U.S. multi-city Women’s Interagency Study, Cook et al. (2008) found that, among HIV-1 infected individuals, those who were persistent crack cocaine users (i.e.,

reporting crack use at every interview) were over three times as likely as non-users to die from AIDS-related causes. Moreover, while approximately 23.5% of non-users and intermittent users of crack cocaine enrolled in the study died of all causes by the follow-up period, among persistent users the percentage that died was 68%. Similarly, in São Paulo, Brazil, Ribeiro et al. (2006) found that mortality among crack users (1992-1994) was more than seven-fold than seen in the general population of the city for the same period. High mortality rates among Brazilian crack/cocaine-dependent individuals in Brazil have also been described by Dias et al. (2011).

Violence is one factor that has been closely linked to high mortality rates among crack cocaine users (Bungay et al., 2010; Ribeiro et al., 2007).

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In addition, the advent of crack cocaine use has contributed to increased mortality through the spread of infectious disease syndemics. Syndemics entail the clustering of diseases in populations as a consequence of adverse social conditions, deleterious interactions of various kinds among comorbid diseases, and resulting heightened rates of morbidity as well as mortality. The syndemics of crack cocaine use involve several identifiable pathways of harmful disease interaction ushered in by the development of widespread use of this drug, namely: 1) crack cocaine use prompted involvement in risky sexual practices that accelerated the spread of several sexually transmitted infections (STIs), and the consequent adverse interaction of STIs with each other, with HIV, and with non-STI infectious diseases (e.g., tuberculosis [TB]) as well; 2) by reducing adherence to HIV treatment and by biochemically advancing HIV/AIDS disease progression, cocaine contributed to enhanced HIV viral loads and the acquisition of co-infections that further damaged the health of affected populations; and 3) crack cocaine use complicated access to health care among individuals infected with TB, and may have increased infectivity and susceptibility to infection, thereby supporting the injurious interaction of TB with other infectious diseases.

Examination of these multiple, entwined routes of infectious disease interaction, as the focus of this article, helps to clarify why crack cocaine users have been found to have such high rates of mortality and suggests the public health value of a syndemics approach to drug-related illness and death.

### **Syndemics of Drug Use**

The term “comorbidity” was introduced into the medical literature by Feinstein (1970) to denote the presence in a patient of one or more diseases (or other disorders) in addition to a specific index disease of primary concern. The term has proved to be useful in medicine as a means of allowing clinicians to develop a framework for treating patients with multiple diseases, especially in helping them make critical decisions about which disease to treat first and whether treating one disease by itself will outweigh the negative effects of not treating other co-present conditions. Feinstein’s approach reflected the prevailing conception of comorbid diseases as independent entities. This strategy, however, failed to draw attention to the importance

of syndemic processes and outcomes of comorbid disease interactions.

As Mustanski et al. (2007) aptly point out:

...it is possible for two disorders to be comorbid, but not ... syndemic (that is, the disorders are not epidemic in the studied population or their co-occurrence is not accompanied by additional adverse health consequences). Beyond the focus on disease clustering and interaction, the term syndemic also implies a focus on health disparities and the social conditions that perpetrate them. (p. 40).

Such an approach, emphasizing the importance of examining how co-occurring diseases influence each other and, in turn, are influenced by their encompassing social environment, offers a fully biosocial framework capable of broadened epidemiologic understanding and suggesting promising directions for improving global public health efforts (Nichter 2008; Singer and Erickson 2013; Syndemics Prevention Network, 2005). As Littleton and Park (2009) stress, studying syndemic relationships is critical because of the global extent of clustering of diseases and noxious social conditions. As a result, syndemics theory offers “a way of assessing and developing inter-sectoral approaches to health and disease prevention and to understanding the way risks are concentrated and buffered over time in particular people and communities” (Littleton & Park 2009, p. 1679). With regard to prevention, the syndemic approach “focuses on the connections among health-related problems, orients developing health policies, and aligns with forces for social change” (Rock et al., 2009; p. 991). For example, the HIV/AIDS pandemic has not exacted its considerable toll on humanity by acting alone; its effects, including those that propelled it into global headlines and continue to cause intense global concern, are the result of ill-fated interactions, on the one hand, with a wide range of opportunistic and non-opportunistic diseases as well as various economic, gender, sexual, and ethnic social inequalities that collectively have come to be called structural violence, on the other.

As an analytic concept in public health, the term “syndemic” was first used to characterize the three-way interaction found among substance abuse, physical violence and HIV/AIDS among inner city poor in the U.S. (Singer 1994; Singer 1996). Subsequently, a growing number of researchers have described drug-influenced syndemics among ethnic minorities, men who

have sex with men, women, and other populations (Bastos et al., 1999; Burke et al., 2010; Feingold, 2009; Gielen et al., 2007; Gonzalez-Guarda, 2009; Halkitis et al. 2013; Senn et al., 2010; Singer, 2006a; Stall et al., 2007; Walkup et al., 2008). Although the role of syringes in mediating drug use syndemics has been stressed in the literature (e.g., Bulled & Singer, 2011), as discussed below, non-injection crack cocaine use also plays a critical role in promoting syndemic disease interaction and enhanced disease burdens.

### **Syndemics of Crack Cocaine Use**

A number of syndemics have been linked to the use of crack cocaine. These are examined more closely below.

#### ***Risky Sexual Practices and STI Disease Interaction***

##### **Association of crack cocaine to risky sex.**

Critical to the role crack cocaine plays in infectious disease interaction and its spread is its impact on risky sexual behaviors (Mackesy-Amitia et al, 2010). Jones et al. (1998) identified three linkages between using crack cocaine and involvement in risky sexual practices: 1) individuals who develop a dependency/addiction to crack cocaine may initiate commercial sex activity; 2) individuals who previously had engaged in commercial sexual exchanges may return to this behavior; and, 3) active commercial sex workers may increase their level of involvement. In all of these cases, sex workers under the influence of crack cocaine “may be less careful when choosing sexual practices or partners” than when not using the drug (Jones et al. 1998, p. 188). These researchers found that crack-involved commercial sex workers frequently reported having sex with persons they believed were HIV positive or knew to be injection drug users. Similarly in a study of street drug users in three U.S. cities, Booth et al. (1993) found that “Crack smokers... reported more sex partners and more acts of unprotected sex; they also were more likely to have exchanged sex for drugs and/or money and to have used drugs more often before or during sex” (p. 1147). These and related findings on the sexual risk patterns of crack cocaine users have been consistently replicated in multiple other studies in and outside of the U.S. with various populations over the last two decades (Balslem et al., 1992; Baseman et al., 1999; Campsmith et al., 2000; Clements-Noelle et al., 2008; Fullilove et al., 1993; Inciardi et al, 1993; Latkin et al., 1996;

Lejuez et al., 2005; Malta et al., 2008; Oliver-Velez et al., 2003; Ross et al., 2002; Sterk, 2000; Timpson et al., 2010; Williams & Ekundayo 2001; Word & Bowser 1997). Notably, the frequency of these patterns has been found to correlate with the quantity and intensity of crack cocaine consumption (Hoffman et al., 2000). Binge users of crack cocaine--those who consume as much of the drug as possible over several days in a row--for example, were found by Harzke et al. (2009) to score higher on a risk-taking measure than crack cocaine users who did not engage in bingeing. Specifically, binge users had more sexual partners and were more likely to have never used a condom during sex.

##### **The influence of crack cocaine on risky sex.**

Why is crack especially associated with high levels of sexual risk? Various explanations of this relationship have been offered, including: the chemical impact of the drug on sexual arousal (Oliver-Velez et al., 2003; Ross et al., 2002) and sexual disinhibition (Booth et al., 1993); the influence of the drug on judgment and safer sexual decision-making (Falck et al., 1997); the predisposition (e.g., level of impulsivity) of people who get mostly heavily involved in the use of the drug (Lejuez et al, 2005); cultural patterns of sexual behavior that developed historically among crack cocaine users (Edlin et al., 1994; Ratner, 1993); involvement in social networks with established high risk patterns (Latkin et al., 1996; Sena et al., 2007); and, the impact of socio-economic structures that bring together traditionally subordinated and marginalized populations with an inexpensive (if short acting) form of a previously high status drug that carries with it a reputation for enhancing sexual experiences and the emergence of a sex for drugs street economy (Singer, 2006b). In fact, all of these factors may play a role in increasing sexual risk among crack cocaine users. Even injection drug users, a group at considerable risk because of multiple person use of injection equipment, have been found to be at even higher levels of disease risk if they engage also in certain sexual practices like having unprotected sex with multiple partners (Chiasson et al., 1991). Whatever the cause(s), one of the significant consequences is that use of crack cocaine has repeatedly been found to be associated with elevated rates of STIs, including HIV. Hence, injection drug users who are concurrent crack cocaine users have amplified rates of infection over injection drug users who do not use crack cocaine (DeBeck et al., 2009).

Miller and co-workers (2008), for example, sampled 200 women involved in drug use in New

York. Participants were tested for a range of STIs, including herpes simplex virus-2, syphilis, gonorrhea, chlamydia, trichomoniasis, and HIV. High rates of previously undiagnosed STIs were identified among participants, especially, herpes, trichomoniasis, and chlamydia. Further, women who reported crack cocaine use were more likely than the other women in the sample to suffer from multiple STIs. A similar array of STIs linked to crack cocaine use was found by Ross et al. (2002) in their examination of patients at three drug treatment facilities in Texas, including HIV, syphilis, chlamydia and herpes simplex-2. This type of clustering of diseases in a population creates the biological potential for the development of an infectious disease syndemic (Singer et al., 2006).

**STI clustering.** Patterns of STI clustering among crack cocaine users vary by location and time period. Several studies conducted in the Bahamas illustrate one such pattern. One of the earliest known locations of cocaine use by smoking—first involving freebase cocaine and later crack cocaine—was the Bahamas. Beginning in the early 1980s, as crack cocaine consumption began to spread among drug users, the Bahamas experienced a series of sequential epidemics of genital ulcer disease (GUD) and HIV infection. GUD, involving lesions in the groin region and other symptoms (e.g., enlarged lymph nodes), is caused by several STIs, including genital herpes, syphilis, chlamydia, and chancroid. Introduction of crack in the Bahamas was followed by a 12-fold increase in the diagnosis of GUD in clinic patients between 1983 and 1987. Shortly after the jump in GUD cases began, there was a dramatic rise in sexually transmitted HIV cases.

To investigate the relationship between crack cocaine use, GUD and HIV, Gomez et al. (2002) conducted a retrospective case-control study of patients diagnosed with GUD at a public STI clinic in the capital city of Nassau. They found that among men, crack use was strongly associated with secondary syphilis, GUD, and HIV, although a strong association with GUD did not hold for women. This research team also discovered a related rise in lymphogranuloma venereum, an STI caused by the pathogen *Chlamydia trachomatis*, among crack users (Bauwens et al., 2002).

A second pattern, involving age/gender defined social networks, has been described by Ellen et al. (1996) based on a study of STI clinic patients in three U.S. cities. These researchers identified two distinct networks: syphilis/crack networks and gonorrhea/crack networks. The former network contained older male and female

crack cocaine users who consumed the drug during sex, often including the exchange of crack for sex. The latter network consisted of older men who did not use crack or other drugs but had sex without a condom with women who were crack cocaine users. Although gonorrhea/crack networks involved the exchange of drugs and money for sex, the men did not define their sexual partners as commercial sex workers. As this study suggests, different STIs may be introduced to and spread in different social networks even in the same location, leading to multiple independent crack cocaine syndemics. Over time, because of social mixing and the behavior of individuals who bridge otherwise separate networks, multiple STIs may circulate and interact within social networks of crack cocaine users.

**STI interaction.** Several distinct types of adverse disease interaction occur among STIs. First, STI interaction can involve changes that lead to one or more infections gaining enhanced contagiousness. This occurs, for example, in individuals dually infected with both the bacterium *Treponema pallidum*, which is involved in the development of syphilis, and HIV. Moreover, although oral sex is usually a comparatively low risk behavior for HIV transmission, oral lesions caused by syphilis infection have been found to attract HIV cells in infected individuals, increasing the risk for HIV transmission with recurring unprotected exposures (Fleming & Wasserheit, 1999). High rates of syphilis and HIV co-infection are facilitated by damage caused to the multilayered epithelial barrier and genital-tract ulceration caused by *Treponema pallidum* that, in turn, facilitates the sexual transmission of HIV. As a result, people with syphilis are 2-5 times more likely to transmit or contract HIV than are those who are not infected with an STI (Wasserheit, 1992). Characteristically, dually infected individuals develop multiple or deeper chancres and an overlapping of primary- and secondary-stage features of syphilis (Rofls et al., 1997; Rompalo et al., 2001).

Secondly, HIV interaction with other STIs can produce an acceleration of pathogenic virulence. In individuals dually infected with HIV and syphilis, for example, there can be rapid progression from early syphilis to neurosyphilis, with resulting blindness, loss of hearing, and paralysis. Several studies (Buchacz et al., 2004; Palacios et al., 2007) have found that in HIV-infected individuals, syphilis infection was associated with a significant increase in HIV viral loads and a significant decrease in CD4 cell frequencies—telling markers of HIV progression.

Similar detrimental interactions have been described between HIV and HSV (herpes) including a significant speeding up of HIV pathogenesis (Mole et al., 1997). As Palù et al. (2001) indicated, genital herpes, more than any other sexually transmitted disease, is linked to HIV-1 transmission not only by increasing HIV-1 load, but also by providing a portico for entry and exit of the virus. This increase appears to be caused by the specific effects that the herpes virus has on the pace of HIV viral replication. Various factors may be involved in this process, including specific herpes proteins that boost HIV replication efficacy (Schacker, 2001).

In some cases, alterations of the body caused by one pathogen promotes the disease progression caused by another pathogen. These alterations include changes in biochemistry (e.g., damage or modulation of immune system components), cellular signaling capacity, and the integrity of organ systems. The case of herpes and the production of genital lesions, noted above, is an example of this route of pathogen-pathogen interaction.

One group of pathogens commonly transmitted through sexual contact is the herpes viruses, a group that has attracted increasing public health attention since the 1960s. From a syndemics standpoint, herpes viruses are significant because they tend “not to act alone, and instead require some other agent of disease, genetic weakness, or physiological upset for the development of herpes vesicles, small blister-like sores” (Barnes 2005, p. 31). The interaction of the herpes viruses with HIV has been the subject of a growing body of research (e.g., Chirgwin et al., 1991). Indeed, at least thirty studies have demonstrated that herpes simplex virus type 2 (HSV-2) is associated with a two to four times greater level HIV infection. As Corey et al. (2004) note, “Of all the sexually transmitted diseases (STDs), there appears to be true epidemiologic synergy between these 2 viruses, in that HIV incidence is increased in parallel with HSV-2 prevalence among HIV-1-negative and -positive persons, and HIV-1 prevalence increases HSV-2 incidence” (p. 435).

Notably, genital herpes has been found to enhance the risk of HIV infection even after a sufferer has been treated with oral acyclovir and the healing of genital lesions. Research by Zhu et al. (2009) found that CD4+ T cells, a target of HIV infection, appeared at healed sites of genital HSV-2 lesions at levels 2 to 37 times higher than in unaffected genital skin. Moreover, these researchers found that CD4+ T cells at healed lesion sites expressed higher frequencies of 2 cell-

surface receptors—CCR5 and CXCR4—which are known to be used by HIV to enter T cells. Compared to control tissue, healed genital herpes lesion sites also were characterized by significantly higher concentration of immune cells that are known to carry HIV particles to CD4+ T cells, whether or not the patient had been treated with acyclovir. In additional experiments, these researchers found that HIV replicates 3 to 5 times faster in cultured tissue from the sites of healed HSV-2 lesions than in cultured tissue from control sites. These findings suggest that HSV-2 lesions create an ideal site for the swift spread of HIV infection.

When herpes and HIV are comorbid, the natural course of both diseases is altered. That herpes is a more significant factor in the transmission of HIV than are other STDs is seen in a revealing study in Rakai, Uganda. Gray et al. (2001) examined the probability of HIV-1 transmission per coital event among monogamous, heterosexual, HIV-1-discordant couples (in which the HIV infected partner was not receiving highly active antiretroviral therapy). Although these researchers found that the presence of symptoms of or laboratory-confirmed infection with gonorrhea, chlamydia, and trichomoniasis did not increase the per event risk of HIV transmission, the HIV-negative partner was five times more likely to become infected with HIV during a single sexual contact if he or she was already infected with herpes. This enhanced susceptibility was found to be especially common if the previously uninfected partner displayed herpes symptoms, but was still statistically significant in asymptomatic infected partners. The bidirectionality of this syndemic is seen in the fact that the presence of HIV appears to promote herpes epidemics (Kamali et al., 1999; McFarland et al., 1999). An alternative interpretation of the findings of these studies, however, is that individuals who have contracted HIV engage in more frequent sexual risk than other people do and that this increases their likelihood of also acquiring herpes.

### **Reducing Adherence to HIV Treatment**

One of the ways crack cocaine use appears to contribute to AIDS syndemics is through its impact on patient adherence to antiretroviral therapy. This issue is of considerable public health importance, because the development of AIDS medicines have led to substantially decreased morbidity and mortality in HIV/AIDS patients, but less than full adherence has been found to be associated with higher viral loads, more rapid

disease progression, and the risk of developing multi-drug resistant viral strains, which significantly reduces treatment options (Patterson et al., 1996). In a review of the literature that addresses this issue, Palmer et al. (2010) reported that HIV patients at greatest risk of nonadherence to antiretrovirals are individuals with current alcohol, marijuana or crack-cocaine use combined with psychological stress. Although not found in all research (e.g., Catz et al., 2000), numerous studies affirm an association between reduced antiretroviral adherence and crack cocaine use (Arnsten et al., 2002; Gordillo et al., 1999; Ingersoll, 2004; Power et al., 2003). For example, in a study of 1655 mostly African American (72%) HIV-infected women, Sharpe et al. (2004) found that crack cocaine users (and to a lesser degree self-reported users of other drugs) were significantly less likely than non-users to adhere to their medical regimen as prescribed. By interfering with antiretroviral adherence, especially when couples continued sexual risk behavior, crack cocaine use increases the likelihood of HIV syndemic interactions with other diseases.

#### **Crack as a Factor in HIV Disease Progress and Resulting Syndemic Co-Infection**

Studies have been published for a number of years reporting direct impacts of crack cocaine use on HIV/AIDS disease progression (Duncan et al., 2007, Larrat & Zierler, 1993). Baum et al. (2009), for example, carried out a 30-month longitudinal study of over 200 HIV seropositive men and women to assess the relationship of drug use on HIV CD4 cell count and viral load. They found that users of crack cocaine were over two times more likely to suffer CD4 cell count decline, independent of antiretroviral treatment. Further, crack users had a higher viral load over the 2.5 year study period. Additionally, they found that a significantly smaller proportion of crack cocaine users on Highly Active Antiretroviral Therapy (HAART) had controlled viral loads compared to non-users. This finding suggests a lower level of medication adherence among crack-cocaine users. Crack cocaine users in the study who were not receiving HAART treatment were found to be at greater risk of disease progression compared to nonusers. In a subsequent study (Baum et al. 2010), this research team reported that individuals who daily drank two or more alcoholic beverages mixed with crack-cocaine use were at an increased risk of CD4 cell decline. Similarly, in a study of over 500 HIV-seropositive methadone patients, Webber et al. (1999) found that crack cocaine use

was associated with progression to clinical AIDS based on the criteria of CD4 cell count and having two or more HIV-related symptoms. Moreover, Cook et al. (2008), in the cohort study of HIV-1 infected women noted above, found that both persistent and intermittent users of crack cocaine had higher HIV-1 RNA levels, were more likely to develop new AIDS-defining syndemic illnesses, and suffered greater CD4 cell loss than non-users, all important markers of HIV disease progression, controlling for confounders like other drug use and injection drug use.

#### **TB and Crack Syndrome**

Hirche et al. (2002) use the term “crack syndrome” (also known as “crack lung”) to refer to the set of pulmonary complications associated with inhaling cocaine. Features of this condition include shortness of breath, intensive cough that can involve coughing up blood, chest pain, and filling of air spaces in the lung with fluids, airway injury, and the collection of air in the pleural cavity (Forrester et al., 1990; Mançano et al., 2008). Concern has been raised that suffering from crack syndrome increases susceptibility to tuberculosis infection upon exposure (Crane et al., 1991; Leonhardt et al., 1994). Although epithelial cells in the lung resist the Mycobacterium tuberculosis pathogen and components of the immune system (e.g., activity of alveolar macrophage and dendritic cells, cytokine production) kill invasive agents, these protective adaptations may be damaged by lung exposure to cocaine. As a consequence, risk for pulmonary TB upon exposure is enhanced. Such exposure is high among crack cocaine users, especially if they frequent crack houses, where the gathering of multiple individuals in often poorly ventilated locations to prevent police detection and coughing by infected individuals promote pathogen transmission. Story, Bothamley, and Hayward (2008), for example, examined almost a thousand pulmonary patients, 15-60 years of age, undergoing treatment in London in July 2003. They compared crack cocaine users in their sample with other illicit drug users and those who did not report drug use. They found that 86% of crack cocaine users were smear positive (i.e., there were TB bacteria in a patient's sputum) for TB compared to 56% of other drug users, and 36% of patients who did not report drug use. These researchers concluded that there is “a dangerous synergy between TB and crack cocaine” (Story et al., 2008, p. 1468). Similarly Taubes et al. (1998) reported that crack cocaine use during the 30 day

period prior to hospital admission was associated with a significantly higher purified protein derivative skin test reactivity rate. Notably, research by Reyes et al. (1996) designed to assess the prevalence of *Mycobacterium tuberculosis* infection and its association with HIV in a sample of over 700 injection drug and crack cocaine users in Puerto Rico found that participants infected with tuberculosis were more likely to also be HIV seropositive, a finding repeated in other studies (Rodwell et al., 2010; Tran et al., 2007).

## Conclusions

Overall, the mortality patterns of crack cocaine drug users are understudied. Although comparatively high mortality rates in this population have been identified in a number of studies, the specific causes remain to be fully explored. During the relatively brief social history of crack cocaine use globally, high levels of violence—tied often to rivalries stemming from drug distribution—have been noted as an important factor in crack cocaine user mortality. Also of note in this regard are high rates of lethal infection. The specific aim of this manuscript has been to show how syndemic interactions involving HIV, TB, and STIs have contributed to elevated levels of infectious disease-related mortality among crack cocaine users. Notably, syndemic disease interactions like the kinds described above are promoted by social conditions of marginalization, poverty, and living in stressful and often traumatic social settings, or what have been called “risk environments” (Rhodes et al., 2005). These conditions promote disease clustering and adversely impact both overall health and immune competence (Singer, 2009). Not surprisingly, the literature on mortality among crack cocaine users notes the deleterious impact of social conditions on health of study participants (Cook et al., 2008; Fischer et al., 2006).

Although several successful individual HIV risk reduction interventions for crack cocaine users have been described (e.g., Cottler et al., 1998; Sterk, Theall, & Elifson, 2003), review and analysis of the literature on mortality among crack cocaine users underscores the need for structural interventions (Auerbach, 2009; Blankenhip et al., 2006, O’Leary & Martins, 2000) that minimize infectious disease clustering and interaction as well as the socioenvironmental adversities commonly faced by crack cocaine users (Singer et al. 2012). Such interventions target the modification of social structures and conditions,

while promoting health and reducing risk in specific social contexts.

Structural-level interventions, which include efforts to adjust cultural norms and expectations, laws and policies, institutional cultures and everyday practices, and economic relationships, hold promise for affecting large numbers of individuals and the factors that enhance risk for mortality among consumers of crack cocaine. In the case of crack cocaine users, especially needed are structural interventions that address issues like homelessness, reliance on commercial sex exchange as a survival strategy, society reintegration following prison release, family reunification, and comprehensive, medically driven low-threshold drug treatment.

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