Chapter 2
Risk for Cancer in Gay, Bisexual and Transgender Men via Infection

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Abstract A number of infections play a significant role in cancer risk for gay, bisexual and transgender (GBT) men. The association between Human Immunodeficiency Virus (HIV), the virus that attacks and degrades the immune system, frequently leading to Acquired Immune Deficiency Syndrome (AIDS) and cancer, has been studied extensively. In addition, human papillomavirus (several types), human herpes virus (also several types), and Hepatitis virus (primarily Hep B and Hep C), each contribute to additional cancer risk in the form of infection-related cancers. This chapter reviews these infections and associated cancers among GBT men including viral transmission and prevalence both independently and in comparison to other men. It further explores the risk of cancer associated with these infections and, given the wide varieties and potential cancer sites, where elevated cancer rates have been observed. In response to these elevated risks, efforts to improve screening, educate GBT men about the increased risk, and new treatment strategies have been implemented. This chapter explores how programs to address infection-related cancer in GBT men have fared to date, including efforts to reduce transmission of infectious agents, early intervention and screening, cost effectiveness of screening, advances in cancer treatment itself, and changes in knowledge, attitudes and behavior among GBT men. The chapter ends with unique challenges with respect to treatment of GBT men with infection related cancers.

1. Prevalence of Cancer-related STIs (HIV, HPV, Herpes, Hepatitis, Epstein-Barr)

a. Human Immunodeficiency Virus (HIV)

HIV has been associated with GBT men since the onset of the AIDS epidemic [1], as well as with cancer–Kaposi's sarcoma (KS). KS, along with Pneumocystis Carinii Pneumonia, was the presenting illness among one of the initial cohorts of AIDS patients, before the disease had a name or its cause was even known [2–3]. Gay and bisexual men, referred to as "men who have sex with men" or MSM by the federal Centers for Disease Control, are 42 times more likely to be living with HIV

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compared to other men [4]. Cancer risk is elevated among people living with HIV/AIDS (PLWH) for three AIDS-defining cancers (ADCs) including Kaposi's sarcoma, non-Hodgkin Lymphoma (NHL) and cervical cancer as well as for several non-AIDS-defining cancers (NADCs) including lung cancer, Hodgkin lymphoma, anal cancer, and liver cancer [5].

HIV risk for GBT men is well reported with risk related to anal intercourse and confounded by co- infection with other STIs including syphilis, gonnorhea, and human papillomavirus. A report based upon the CDC's National HIV Behavioral Surveillance system collected cross-sectional data in 21 U.S. cities in 2008 and found 19% of gay and bisexual men to be infected with HIV [6].

b. Human Papillomavirus (HPV)

HPV is the most common sexually transmitted infection and can be transmitted through oral, anal and vaginal sex [7]. Using DNA sequencing, more than 100 HPV types have been identified, of which 40 types infect the genital epithelia [8]. A person does not need to be symptomatic to transmit the virus [7]. Various health problems are associated with HPV including genital warts and cancers. However, most HPV infections do not cause symptoms or disease and are cleared by the body (Table 2.1).

HPV has been found to increase risk for oropharangeal, penile and anal cancers in men, including both HIV-negative and HIV-positive GBT men. Chin-Hong et al. [9] found anal HPV infection in 57% of a sample of urban HIV-negative men. Infection in this cohort was correlated with receptive anal intercourse and greater than five sex partners, both in the past 6 months. Rates of HPV among GBT men with HIV infection have been found to be as high as 93%–97.9% [10]. Two HPV types, HPV 16 and 18, are considered to carry the highest risk for cancer [11]. Reviewing

Table 2.1 List of common acronyms

ADC	AIDS Defining Cancers
EBV	Epstein-Barr Virus
HAART	Highly Active Antiretroviral Therapy
HBV	Hepatitis B Virus
HCV	Hepatitis C Virus
HHV	Human Herpes Virus
HL	Hodgkin Lymphoma
HPV	Human Papillomavirus
KS	Kaposi's sarcoma
NADC	Non-AIDS Defining Cancer
NHL	Non-Hodgkin Lymphoma
PLWH	People living with HIV/AIDS
SIR	Standardized Incidence Ratio

incident cases of anal cancer among men from 2004 to 2007, CDC attributed 93% to HPV infection and 87% specifically to HPV 16 and 18 [12].

c. Human Herpesvirus (HHV)

There are eight types of human herpesviruses including varicella/zoster (VZV/HH3), Epstein- Barr virus (EBV/HHV4), Cytomegalovirus (CMV/HHV-5) and Kaposi's sarcoma herpesvirus (KSHV/HHV-8) [13]. HHVs are transmitted both sexually and non-sexually with evidence of sexual transmission for HH2, EBV, CMV and KSHV [14].

In 1994, Chang [15] found that human herpesvirus 8 (HHV-8) was the cause of Kaposi's sarcoma. In one study, Del Mistro [16] compared rates of HHV-8 and HPV among three groups of PLWH–MSM, heterosexual men, and women, finding higher rates of both HHV-8 and HPV among gay and bisexual HIV+ men.

Epstein-Barr Virus (EBV) is another name for human herpesvirus 4 and one of the most common human viruses. Most people are infected in childhood and do not develop symptoms or have very minor symptoms [17]. EBV was identified over 40 years ago in a biopsy of Burkitt's lymphoma, becoming the first infectious agent to be directly associated with a human cancer 18].

While there is limited epidemiological data on rates of EBV infection in gay and bisexual men, one study found a higher prevalence of EBV type 2 among gay men compared to heterosexual men associated with HIV infection and a higher number of sexual partners [19]. Additional epidemiological research would be helpful to know more about prevalence rates of EBV among gay/bisexual/transgender and heterosexual men in a variety of geographic locations (unlike parts of Africa where EBV is endemic). EBV is associated with a diverse group of lymphomas and carcinomas including Burkitt's lymphoma, Hodgkin's disease, Post-transplant lymphoproliferative disease, AIDS- associated lymphoma, and nasopharyngeal and gastric carcinoma [17].

d. Hepatitis B (HBV) and Hepatitis C (HCV)

Hepatitis B and C are disproportionately found among gay and bisexual men [19]. Hepatitis B is spread in a manner similar to HIV, i.e. through blood or semen. Hepatitis B is considered to be 50–100 times more infectious than HIV [20]. Hepatitis C is primarily spread through sharing of needles and syringes. Over time, Hepatitis B and C attack the liver, causing a variety of liver diseases including liver cancer. It is estimated that 90% of liver cancers in less developed countries and 40% of liver cancers in more developed countries are attributable to HBV or HCV infection [22]. Approximately 20% of gay and bisexual men account for new Hepatitis B infections [20], disproportional to their 4% representation in the general population.

2. Reasons for different rates of cancer than in the heterosexual community

Why Higher Rates of Infection Lead to Higher Rates of Cancer

HIV can increase the risk of GBT men for rare cancers like non-Hodgkin Lymphoma, Kaposi sarcoma, and liver cancers. Additionally it has been shown that immunosuppression and infection with other viruses related to HIV/AIDS puts those infected at higher risk of anal, liver, lung cancer. HPV infection impacts disparate cancer rates in GBT men. HPV infection leads to penile cancer for men, cervical cancer for women, and cancers of the mouth, throat, and anus for people of both genders. Hep B and C can also put people at risk for liver cancer and lymphomas.

HIV-positive Patients Have Higher Risk for Some Cancers

The burden of cancer on PLWH has been well documented. However, the nature of this burden has shifted, specifically as a result of the introduction of highly active antiretroviral therapy (HAART) in 1996. For example, in a 2011 study, Shiels [23] found that during 1991–2005, an estimated 79,656 cancers occurred in the population of people living with AIDS in the United States. However, comparing the periods 1991–1995 and 2001–2005, the estimated number of AIDS-defining cancers decreased by greater than threefold from 34,587 to 10,325 cancers. In contrast, the number of non-AIDS-defining cancers increased by approximately threefold from 3193 to 10,059. An earlier meta-analysis by Shiels [24] found that the standardized incidence ratios (SIR) of NADC was approximately 2-fold higher risk for all NADC among PLWH compared with the general population. However, individual cancer types associated with infectious agents had different SIRs including anal cancer (SIR = 28), Hodgkin lymphoma (SIR = 11), and liver (SIR = 5.6).

Robbins [25], exploring data from 1996 to 2010, looked at cancer trends for three ADCs and seven NADCs to see if demographic changes for HIV positive individuals, changes in relative risks, and/or background incidence in the general population had an effect, and if so which. Table 2.2 includes a summary of changes identified by Robbins. Simard [26] had similar findings looking at data from 1980 to 2006.

Each of the ADCs has a viral cause suggesting that the advent of HAART in 1996 has had an effect on cancer reduction by improving the immune system's ability to manage the viral infection [25]. At the same time, demographic shifts were likely related to the increase in liver cancer and prostate cancer; specifically, the increase in liver cancer reflected additional years living with Hepatitis B and C viruses. Curtrell [27] explores several factors related to the increase in NADC including oncogenic effects of HIV, immunosuppression, chronic inflammation and immune activation, exposure to HAART, higher rates of oncogenic viral coinfections and traditional cancer risk factors. The same study found that when standard cancer therapy is given, PLWH have the same outcomes as the non-HIV population [27].

Other types of NADC have been identified among PLWH. Silverberg [28] compared a California cohort of HIV-infected persons, of whom 74% were MSM, and compared them with a demographically similar group non-HIV-infected persons. He found adjusted rate ratios, coming HIV-infected with HIV-uninfected persons

Table 2.2 Trends in Cancer incidence among HIV infected persons [25]. (Source: Robbins et al. AIDS 2014 Mar 27)

Type of cancer	Trend 1996–2010 (unless otherwise indicated)		Summary
Kaposi's sarcoma (ADC)	1996–2000 2000–2010	−29.3% −7.8%	Decreasing
NHL (ADC)	1996–2003 2003–2010	−15.7% −5.5%	Decreasing
Cervical cancer (ADC)	−11.1%		Decreasing
Anal cancer (NADC)	3.8%		Increasing
Liver cancer (NADC)	8.5%		Increasing
Prostate cancer (NADC)	9.8%		Increasing
Hodgkin Lymphoma (NADC)	−4.0%		Decreasing
Lung cancer (NADC)	−2.8%		Decreasing

of 37.7 for ADC, 9.2 for infection-related NADC, and 1.3 for infection-unrelated NADC. The rates for individual NADC included anal squamous cell (rate ratio = 101.6), Hodgkin lymphoma (rate ratio = 19.4), penis (rate ratio = 5.8) liver (rate ratio = 2.7) and HPV-related oral squamous cell cancers (rate ratio = 2.0) [27]. Among infection- unrelated NADC there were increased rates for people with HIV infection for other anal (rate ratio = 35.3, nonmelanoma skin (rate ratio = 10.6), other head and neck (rate ration = 2.7), lung (rate ratio = 1.9) and melanoma (rate ratio = 1.5). HIV-infected persons also had a lower rate of prostate cancer (rate ratio = 0.7). HIV infection was not associated with higher rates of other infection-unrelated NADC [27]. Similarly, Grulich et al. [29] demonstrated that other cancers not known to be associated with an infection were also elevated in both immunosuppressed populations, that is, HIV-infected persons and transplant recipients, including lung and kidney cancers, multiple myeloma, and leukemia. Yanik [30] found decreasing rates from 2000 to 2011 of NHL among a cohort of HIV-infected individuals in North Carolina.

Silverberg [31] found that HIV-infected patients had a twofold higher incidence rate of non- melanoma skin cancers compared with non-HIV-infected subjects. Squamous cell cancers but not basal cell cancers were associated with immunodeficiency. Shebl [32] concluded that chronic pulmonary inflammation arising from infection contributes to recurrent pneumonia which puts PLWH at greater risk of lung cancer, independent of higher smoking rates. Similarly, Sigel [33] found HIV infection was an independent risk factor for lung cancer when controlling for potential confounders, including smoking and surveillance bias.

Persson [34] looked at data for 596,955 person with AIDS from 16 US population-based HIV/AIDS and cancer registries. Risk of stomach and esophageal malignancies in people with HIV/AIDS were compared with those of the general population using standardized incidence ratios (SIRs). People with HIV/AIDS had increased risk of carcinomas of the esophagus (SIR, 1.69) carcinoma of the stom-

ach (SIR, 1.44), esophageal adenocarcinoma (SIR, 1.91), squamous cell carcinoma (SIR, 1.47), and carcinomas of the gastric cardia (SIR, 1.36) and noncardia (SIR, 1.53) compared with the general population. Rates of NHL decreased from 1980 to 2007 with HAART, but incidence of carcinomas remained consistent over time [34].

Thus while HAART has reduced the risk of cancer related to HIV and EBV i.e. NHL, other cancer risks continue to be elevated related to HIV infection and immunologic status, possibly unrelated to other infections.

Infection with HPV Raises Cancer Risk for GBT Men and Especially Those Co-infected with HIV

Beachler [35] found HIV infected gay and bisexual men to have higher rates of anal HPV infections compared to HIV-infected heterosexual men. In addition, anal HPV infection rates were higher than oral HPV infection rates, contributing to the higher burden of anal HPV associated cancer in HIV-infected individuals. Berry [36] described the progression from anal high-grade squamous intraepithelial lesions (HSIL) to anal squamous cell cancer.

Incident anal cancer has increased by 96% in men and 39% in women since the 1980 primarily due to the HIV epidemic [37]. Nearly all anal cancers in gay and bisexual men are associated with HPV [38]. The rate of abnormal anal cytology in a cohort of 60 young gay and bisexual men (mean age = 21.2 years) was found to be comparable to the rate among adult MSM [39]. Increased risk for developing anal cancer among PLWH was associated with prolonged survival and increasing immunosuppression [40].

The prevalence of cirrhosis and hepatocellular carcinoma in patients with human immunodeficiency virus infection. The Hepatitis C (HCV) epidemic has driven up the rate of cirrhosis and hepatocellular carcinoma (HCC) amongst HIV-infected persons. Among patients co-infected with HIV and HCV, there was a dramatic increase in the prevalence of cirrhosis (3.5–13.2%), decompensated cirrhosis (1.9–5.8%), and HCC (0.07–1.6%). Little increase was observed among patients without HCV co-infection in the prevalence of cirrhosis [41].

However, HIV Infection Does Not Lead to Higher Risk or Different Types of Risk for all Kinds of Cancer

HIV does not appear to impact some of the most common cancer including colorectal and prostate cancer among GBT men. Shiels [42] found a reduced risk of prostate cancer among HIV-infected men, though attributed that primarily to lower rates of screening. There were no differences between rates of distant stage prostate cancer between people with AIDS and the general population, giving strength to this argument [42]. Incidence rates of head and neck squamous cell cancers were higher among HIV-infected patients, compared with other gay and bisexual men. However, the risk factors for head and neck cancer were similar for HIV-infected persons and the general population [43].

3. Interventions for risk reduction, include efforts to raise knowledge/awareness of how infections raise cancer risk, the role of vaccination in preventing infection and how screening and early treatment may reduce cancer incidence

a. Current Interventions and treatments for for STIs and HIV

Interventions to reduce STIs including HIV include behavioral interventions, use of highly active antiretroviral therapy (HAART), and both HPV and HBV vaccines, screening, and treatment, are frequently targeted to gay and bisexual men. In many situations, people with any STIs are recommended to undergo the same cancer screening and, if diagnosed, receive the same treatment regimens for both the infection and the cancer as those without infection. However this is not always the case, and there are many studies in progress to find more effective ways of treating these populations.

b. Interventions specific to infection and cancer risk

I. Knowledge, awareness and perceived risk

Gilbert [44] found there not to be much of a difference between HIV(+) and (−) men, but that overall there was acceptability for the vaccine, little understanding of how HIV increases risk for HPV-related diseases, and other misperceptions about the vaccines. This information can inform awareness/prevention efforts for gay men. Blackwell [45] conducted a descriptive study to assess knowledge of HPV, anorectal carcinoma, and anorectal screening in a sample of MSM in Orlando, FL. The 89 participants demonstrated low levels of knowledge with an average score on knowledge items of 38 % correct. Of the 49 participants who had heard of anal Papanicolau (Pap) smears, only five (10.2 %) discussed screening with a physician, while eight (16.3 %) had discussed it with a nurse, and 16 (32.7 %) with another health care professional.

Rosa-Cunha [46] found that only 54 % of men who have sex with men (MSM) reported discussing anal health with their HIV providers in the prior 12 months. Rates for MSM and heterosexual men were 5.56 times and 2.31 times more likely, respectively, than women to have to discuss anal health with their HIV provider. Interestingly, having reported unprotected sex with a partner who was HIV negative or whose HIV status was unknown was inversely related to having a discussion about anal health with their primary care provider [46].

Burkhalter [47] explored perceived risk of cancer in a large urban community center and found that men associated a higher number of sexual partners with a higher risk for cancer. Sanchez [48] found that a quarter of MSM attending a sexually transmitted disease clinic in New York City did not know that HPV is transmitted through anal sex and 77 % were unaware of the link between HPV and anal cancer.

II. Vaccinations

Vaccinations are available to reduce exposure to HPV and Hep B. In 2009, the FDA licensed the use of quadrivalent vaccine for the prevention of genital warts in males ages 9–26 and in 2010, its use was extended for prevention of anal cancer in the same group [49]. On October 25, 2011, the Advisory Committee on Immunization

Practices (ACIP) recommended routine use of quadrivalent HPV vaccine in males aged 11–12 and vaccination with HPV4 for males aged 13–21 who have not been previously vaccinated or did not complete the three dose series [50]. Males aged 22–26 may also be vaccinated [50]. To date, update of HPV vaccination among adolescent males generally has been limited. Reiter [51], looking at a nationally representative sample of adolescents, found that HPV vaccine initiation among males ages 13–17 increased from 1.4 % in 2010 to 8.3 % in 2011. Parents were more likely to get their sons vaccinated against HPV if they received a recommendation from their healthcare provider [51]. Gay and bisexual men have been found to have greater willingness to receive HPV vaccines as well as higher levels of concern about HPV-related diseases [52].

For GBT males, perceived benefits and barriers were the most proximate predictors of intention to be vaccinated against HPV, while knowledge and perceived threat exerted an indirect influence [53]. One study found 73 % of gay and bisexual men were willing to receive the HPV vaccine [52]. Another study of young gay and bisexual men found that 36 % were likely to be vaccinated based upon perceived stronger physical and psychological benefits [54]. Kim [55] found HPV vaccination to be cost effective using the standard measure of costs per QALY below $ 50,000. With respect to Hepatitis B vaccination, a study of 3,432 MSM age 15–22, found only 9 % immunization coverage and 11 % infection rates [56].

III. Screening innovations

Given the increasing rates of anal cancer, substantial effort has been placed on screening for anal intraephitelial neoplasia (AIN), primarily using high-resolution anoscopy. High resolution anoscopy (HRA) was developed in England in the 1980s and uses a colposcope to explore the anal mucosa. A swab, soaked in 5 % acetic acid, is inserted through the anoscope and applied topically for 1–2 min. Lesions reacting to the application are identified and biopsied [57]. Anal-rectal cytology collects non-gynecological specimens via exfoliative cytology tests which are then interpreted by a qualified pathologist. The smear is the same technique as a Pap test, whereby the exfoliated cells are quickly smeared and fixed onto a glass slide [58]. Cachay [59] found that despite the availability of several modalities for treatment of precursors of anal cancer, evidence that current treatment modalities favorably alter the natural history of human papillomavirus oncogenesis in the anal and perianal regions is still inconclusive. However, there is sufficient evidence to state that the accuracy of anal cancer screening procedures (cytology and high-resolution anoscopy directed biopsy) is comparable to the accuracy of those used in screening for cervical cancer precursors. More research is needed to assess the efficacy of anal cancer screening programs on reducing morbidity/mortality in the HIV-infect population [59].

Darragh [60] looked at inter-rater reliability in the reading of Papanicolaou-stained liquid based cytology cells being used for anal cancer screening among high risk populations of gay and bisexual men. Two observers had an overall agreement of 66 % and this increased to 86 % for dichotomized cytology results. Thus review-

ers were able to detect which lesions were precancerous and which were not, similar to the methodology used for cervical cancer screening. A high rate of acceptability of screening was found at a Veteran's Affairs HIV Clinic [61]. When approached during a routine care visit to participate in the study by obtaining an anal Pap smear, 82% of HIV-patients agreed to do so. Another clinic was established at an HIV clinic in New York to comply with New York State AIDS Institute guidelines for anal cancer screening and treatment in HIV-positive persons. The intent is to reduce morbidity and mortality in young, HIV-infected persons [62].

However, a review of the literature found that screening for anal cancer in HIV-positive gay and bisexual men as well as HIV-positive women was not cost-effective [63]. Given the number of false-positives, results with treatment for high-grade AIN, there were no models that showed a 50% probability of cost-effectiveness to a quality-adjusted life year (QALY) gained reaching the value of 50,000 British pounds. This is contrary to earlier reports that found it to be cost effective to screen for anal squamous interepithelial lesions in gay and bisexual HIV-infected men [64].

Routine HIV testing, in the form of standard "opt-out" protocols is recommended by the CDC in all health care settings. This has not been widely implemented, but should be for cancer patients [65], in order to maximize effective HIV management during cancer treatment and improve clinical outcomes.

IV. Treatment innovation/cancer risk reduction

A review of recently published literature on the heightened risk for cancer in PLWH explored whether early HAART treatment can lower their risk [66]. The findings were that immunodeficiency still appears to be the key factor; however, there is emerging evidence that HIV may have direct oncogenic effects through inflammation and coagulation that HAART only partly normalizes. Analysis of studies comparing the impact of early versus delayed HAART was inconclusive [66]. Chiao [67] looked at a cohort of US veterans with HIV and in a multivariate analysis found that those with controlled (i.e. undetectable) viral load at 61–100% of follow-up time had significantly decreased risk of squamous cell anal cancer compared to those with undetectable viral load less than 20% of the time.

Compared to high-resolution anoscopy alone, it is more beneficial to health outcomes as well cost effective to use combined HRA and anal cytology at 6 and 12 months as a method of surveillance for HIV-positive MSM treated for high-grade anal intraepithelial neoplasia to prevent anal cancer and to maximize QALYs [68]. Use of HPV vaccine in HIV-infected children and adult men is safe and highly immunogenic [69]. More research is needed on the role of HPV vaccination for older adults living with HIV who have ongoing HPV infections [69].

A California-based study found an inverse association between statin use and risk of NHL in HIV(+) persons, and though there were limitations in the study design this may be an area for additional research [70].

4. Unique challenges

a. Effect of HAART

Although HAART has led to reductions in the incidence of Kaposi's sarcoma and non-Hodgkin lymphoma among HIV-infected individuals, it has not reduced the incidence of cervical cancer, which has essentially remained unchanged. Moreover, the incidence of several other cancers, particularly Hodgkin lymphoma and anal cancer, has been increasing among HIV-infected individuals since the introduction of HAART. The influence of HAART on the risk of these other cancer types is not well understood [71], [72], [40].

Research continues to show that in the post-HAART era, PLWH continue to be at increased risk for cancer late after AIDS onset [73] and that cancer-related mortality for PLWH is significant [74]. However, early treatment with HAART has been shown to lower cancer risk generally [28]. Overall, use of HAART was not associated with increased ADC or NADC risk, except for one NADC, anal cancer [75].

With respect to the leading cause of cancer-related death among HIV-infected individuals, Gopal [76] found that, over time, HIV-associated lymphoma is changing with less immunosuppression and greater HIV control at diagnosis. Both stable survival and increased mortality for lymphomas occurring on HAART need more research to improve outcomes [76].

b. Understanding mechanisms associated with infection and cancer

For some infections it is unclear exactly what the mechanism is that leads to cancer. For example, both HBV and HCV have been demonstrated to relate to increased risk, but the exact mechanisms are unknown and likely different for both viruses. Jiang [77] reports that while the integration of HBV into the host genome has been reported, the scale, impact and contribution to HCC development are not clear. With respect to HCV, there are a range of lympho-proliferative disorders that required clinical, pathological and molecular findings to establish diagnosis and treatment [78].

c. Special treatment concerns for management of cancer in HIV-infected individuals

Treatment of cancer in people with HIV needs to be cognizant of managing two complex treatment regimens simultaneously. Hadjuandreou [79] finds the key to controlling resistance is the optimal management of the frequency and magnitude of treatment interruptions. As we continue to learn about the interactions of multiple infections, clinicians need to ensure that innovations in treatment for HIV and other infections don't come with negative side effects. People with STIs or HIV may have not just higher risk, but also worse prognoses once diagnosed with cancer. For example, even when the stage of presentation and use of treatment was the same for HIV-infected and non-infected persons, HIV-infected person with non-small cell lung cancer (NSCLC) lived 6 months compared with 20 months for non- infected persons, suggesting this cancer might behave more aggressively in the presence of HIV [33]. Another study found people with HIV and NSCLC had more complica-

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