Smoking, HIV Infection, And Gay Men In The United States

David R. Arday, Brian R. Edlin, Gary A. Giovino, David E. Nelson

On 13 August 1992 the Wall Street Journal announced, “The tobacco industry, after being beaten back in its controversial efforts to woo blacks and women, may be turning its marketing muscle on another minority. homosexuals”.1 Philip Morris Inc reportedly planned to unveil its new Benson & Hedges Special Kings brand using advertisements in the national gay press.

Homosexual men in the US have been devastated by the epidemic of human immune-deficiency virus (HIV) infection. Niche marketing of tobacco products to homosexuals may be particularly invidious if smoking enhances the morbidity or mortality of HIV infection. Three questions bear on this possibility. Is cigarette smoking more prevalent among homosexual men than it is among heterosexual men? Does smoking increase the risk of acquiring HIV infection? Does smoking enhance the progression of HIV disease? Unfortunately, little conclusive research has been reported in any of these areas.

Smoking prevalence among gay men

Although the prevalence of smoking has declined in recent years, 2 data from several sources suggest that the prevalence of smoking among gay men is at least as great and probably somewhat greater than among men in the total population. Among a cohort of 202 Washington, DC, and New York City homosexual men (mean age; 34 years) enrolled in a prospective study of risk factors for HIV infection and acquired immune deficiency syndrome (AIDS), 46% (93) reported they were active cigarette smokers at enrollment in 1982.3 By comparison, National Health Interview Survey (NHIS) data showed that 35.1% of US adult men over the age of 18 smoked in 1983.2

In the 1984 population-based San Francisco Men’s Health Study (998 men aged 24-55 years) the smoking prevalence was 41.8% among homosexual/bisexual enrollees, compared with 27.9% among heterosexual enrollees.4 The prevalence of heavy smoking (>2 packs per day) was higher among homosexual and bisexual men (8.3%) than among heterosexual men (2.5%), and it was highest among HIV-seropositive men (9.4%).

In a 1991 San Francisco convenience sample survey of alcohol and other drug use among 416 gay and bisexual men (mean age: 35 years; age range: 18-76 years; 79% white; 88% with 13 or more years of education), 37.3% (155) reported they were current cigarette smokers (J Kelly, EMT Associates, Sacramento, CA, unpublished report, 1991). By comparison, the 1990 NHIS smoking prevalence among all US men over the age of 18 was 28.4%, and among all US white men over the age of 25 with 13 to 15 years of education it was 25.4%. 2 The 1989 smoking prevalence among all men over the age of 18 in San Francisco was 25.5%, according to a study by the State of California.5

Smoking and the risk of HIV infection

Whether smoking affects the risk of acquiring HIV infection is uncertain. Several research teams3.4.6 have reported a higher risk of sexually acquired HIV infection among cigarette smokers, but these studies have been limited in their ability to control for the behavior most likely to be the strongest risk factor for HIV acquisition - unprotected sexual intercourse with an infected partner. Smoking was clearly associated with riskier sexual practices in all these studies. In one study of Haitian women, 6 however, the association between smoking and HIV infection among a sub-group of women living in houses with concrete floors (a marker for higher socioeconomic status) remained after the data were adjusted for the number of sexual partners.
Smoking and immune function
Numerous clinical studies have shown alterations in immune function and inflammatory response among cigarette smokers, including elevated leukocyte counts, alterations in T-lymphocyte subsets, and changes in immuno-globulin levels. Normal T-cell function plays an essential part in the defense against the development of both infection and neoplasia. Analysis of T-lymphocyte subsets in smokers have generally shown increases in total T-cell and CD4+ (T-helper) cell counts in light and moderate smokers, but a relative decrease in the CD4+ subset and an increase in the CD8+ (T-suppressor) subset in heavy smokers, though these changes are generally reversible with smoking cessation. In addition, smokers have a significantly lower proportion of circulating natural killer cells, a decrease that persists despite smoking cessation.

Cigarette smoking's effects on CD4+ cells have been recently studied in both healthy and HIV infected individuals. Smoking increases CD4+ cell counts in healthy whites, though the reverse may be true in blacks. CD4+ cell counts have been noted to fall in smokers who quit and to increase in non-smokers, compared with non-smokers, in studies of men and with recent HIV infection; but these counts initially fall faster in smokers, and the difference between the counts for smokers and non-smokers is markedly reduced within 2 years after seroconversion. Such effects may require clinicians and researchers who measure CD4+ T-lymphocyte counts in HIV-infected individuals to consider smoking status when interpreting CD4+ cell levels in recently infected persons.

Smoking and the course of HIV disease
Despite their initially higher CD4+ T-lymphocyte counts, smokers with HIV infection do not fare better than non-smokers, and they may do worse. Two recent prospective studies have examined the effects of smoking status on dates of HIV seroconversion. In a study of male sexual contacts of men with AIDS, smoking status at enrollment was not associated with the contacts later progression to AIDS. The subjects were not stratified by amount of cigarette consumption, however, so an effect limited to heavy smokers might not have been detected. In a more detailed analysis, Burns similarly reported that smoking was not associated with a subsequent increased risk of either Pneumocystis carinii pneumonia (PCP) or other AIDS-defining conditions. A number of these subjects reduced or discontinued their smoking during the study, however, and the study may not have had enough power to control for this and identify a meaningful dose effect.

In two other analyses, smoking was associated with an increased risk of AIDS or PCP. These studies controlled for CD4+ T-lymphocyte cell counts rather than time of seroconversion, and the findings might have resulted from higher CD4+ cell counts in smokers at seroconversion rather than faster progression to AIDS. In a third analysis, researchers found no association after adjusting for duration of HIV infection.

Conclusions
In summary, homosexual men do appear to have a higher prevalence of smoking than do men in the general population, though no firm conclusions can be drawn about the association between smoking and HIV-associated morbidity. What is certain, however, is that smoking is associated with many other diseases and that it adversely affects many organ systems, including the immune system. Clearly, any increase in smoking would be expected to be harmful to the gay population.

As early as 1985, Newell called for smoking cessation as a possible means of reducing the risk of homosexual men acquiring AIDS. While it is not clear that smoking increases the risk of acquiring HIV infection, given the high smoking prevalence among gay men, smoking cessation efforts targeted to this population are certainly needed for a variety of health reasons. With AIDS ravaging gay communities in the US, niche marketing of tobacco products to gays only adds further insult to a population already at risk.

1 Lipman, J. Philip Morris to push brand in gay media. Wall Street Journal, August 13, 1992. 81
Addendum

At this time of Tobacco Control was going to press, a study on the relationship between cigarette smoking and the progression of AIDS was published (Nieman RB, et al. The effect of cigarette smoking on the development of AIDS in HIV-1-seropositive individuals. AIDS 1993; 7. 705-10). The study population included 84 individuals with AIDS seen at the genitourinary medicine outpatient department of St. Mary's Hospital in London, UK, who provided information on their smoking behavior before their AIDS-defining diagnosis.

The authors reported the following findings: “Progression time to AIDS (all diagnoses) was significantly reduced in HIV-1-seropositive smokers: median time to AIDS was 8.17 months for smokers (n = 43) and 14.50 months for non-smokers (n = 41) (p = 0.003). Smokers developed Pneumocystis carinii pneumonia (PCP) more rapidly than non-smokers, with a median time to PCP of 9.0 months, compared with 16.0 months for non-smokers (p=0.002). Smoking had no significant effect on progression time to AIDS when not due to PCP.”-Ed.