Five years ago, the International Agency for Research on Cancer (IARC) conducted a case-control study in 9 sites (Italy, Spain, Northern Ireland, Poland, India, Cuba, Canada, Australia, and Sudan) of 1415 participants with cancer of the oral cavity and 255 with oropharyngeal carcinoma.1 Markers for human papillomavirus (HPV) infection were evaluated, including antibodies against HPV16 E6 and E7 proteins, which are common in cervical cancer, and HPV DNA in biopsy samples, detected by polymerase chain reaction assay. HPV was documented in biopsy specimens from 18.3% (range, 12.0% to 24.7%) of patients with oropharynx cancers and 3.9% (range, 2.5% to 5.3%) of those with oral cavity tumors.1 The fact that these lesions were detected less frequently among tobacco smokers, paan (tobacco plus betel leaf and areca nut) chewers, and those with heavy alcohol intake—common risk factors for cancers of the oral cavity and oropharynx in general1,2—and were more common among persons who reported having more than one sexual partner or who practiced oral sex led to the conclusion that they represent a special category of tumor, and yet another form of sexually transmitted cancer. This observation was replicated by a study in the United States conducted between 2000 and 2006 that found HPV16-associated cancer of the head and neck was positively correlated with the number of oral sex partners as well as with marijuana use.3 Cancers of the oral cavity and oropharynx are major worldwide health concerns, with more than 400,000 new cases diagnosed annually.4 In addition to the IARC study, data from 9 US Surveillance, Epidemiology, and End Results (SEER) program registries collected from 1973 to 1994 were reviewed to explore links between HPV and oral squamous cell carcinomas.4 The data showed that 38.5% of the 45,679 tumors examined appeared to be HPV-related. They were diagnosed at younger ages (61.0 vs 63.8 years; P < .001), and their incidence increased significantly over the past 3 decades, with an annual percentage change of 0.80.4 In contrast, oral cancers not linked to HPV declined from 1983 to 2004.4 HPV-related disease was diagnosed in more men than women (72.6% vs 61.9%; P < .001), and sites of the HPV-related tumors included a higher prevalence at the base of the tongue and the tonsil.4 One hopeful finding was that for all stages of oral cancer treated with radiation, patients with HPV-related disease had higher survival rates. This difference, confirming earlier studies, is believed to arise from enhanced sensitivity to radiation and chemotherapy, associated with increased expression of p16, a cyclin-dependent kinase inhibitor that blocks cell cycle progression.5 Although the number of oral cancers for which HPV16 appears to be the cause is relatively low in the United States—about 6000 per year versus 11,000 per year for cervical cancer—there has been a 5% increase per year in HPV-associated head and neck cancers since 2000.6 These findings also have particular significance in the setting of HIV infection. There is an excess risk of HPV-linked cervical cancer—an AIDS-defining malignancy—as well as HPV-associated penile and anal canal malignancies among HIV-positive persons, with no decline in incidence for many of these tumors despite the introduction of highly active antiretroviral therapy.7,8 Dr Maura Gillison of Johns Hopkins University, Baltimore, an author of several of the studies cited above, felt that these new findings “should give people optimism that the [HPV] vaccine that was approved largely for women and for cervical cancer
could have broader implications, and also for other cancers that occur in both men and women.\textsuperscript{9} Indeed, others have suggested that HPV vaccination may aid in the local control of HPV-linked anal cancer.\textsuperscript{8}

Extension of HPV vaccination to adolescents is clearly worthy of consideration, although there are several practical obstacles to its implementation and actual use. First, despite its availability for the past 2 years and its promotion through many school systems, only 49% of mothers in the United States said they would vaccinate a daughter if she was 12 years or younger.\textsuperscript{10} The CDC recommends vaccination when girls are 11 to 12 years old, with a "catch-up" immunization between ages 13 and 26.\textsuperscript{10} Second, rates of vaccination for hepatitis B virus (HBV) infection among young men who have sex with men, who are at very high risk for acquisition of HBV, are appallingly low in the United States, at 17.2%, despite access to regular sources of health care.\textsuperscript{11}

References:

References

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