Oral human papillomavirus (HPV) infection is the principal underlying cause of a dramatic increase in oropharyngeal cancer incidence over the last several decades in the United States.[1-3] If recent incidence trends continue, the annual number of HPV-positive oropharyngeal cancers is expected to surpass the annual number of cervical cancers by the year 2020.[4-6] Approximately 70% of cancers of the oropharynx in the United States are linked to HPV.[7]

This article has an accompanying online continuing education activity available at: http://jada.ada.org/ce/home.

Oral HPV 16 infection confers an approximate 20-fold increase in risk of developing oropharyngeal cancer.[8] However, the clinical usefulness of oral HPV detection for screening for oropharyngeal cancer is unknown. Investigators in population-based studies of oral HPV infection in the United States have identified age, being male, current smoking, and high number of lifetime sexual partners as factors associated with oral HPV infection.[9,10] HPV infection was greatest among African-American participants, those who smoked more than 20 cigarettes daily, current marijuana users, and those who reported 16 or more lifetime vaginal or oral sex partners.[11] The point prevalence in the United States of an oral HPV infection by a cancer-causing HPV type is approximately 7.3% in men and 1.4% in women, corresponding to approximately 8.6 million people.[10,11]

The recognition that oral HPV infection plays a role in the pathogenesis of oropharyngeal cancer has resulted in a paradigm shift in concepts of the risk of developing cancer of the oral cavity.[12] Young people without a history of tobacco or alcohol use nevertheless may be at risk of developing oral cancer. The dental community is well positioned to play an important role in the development of clinical algorithms for secondary prevention of HPV-positive oral cancers. Oral disease detection is generally the responsibility of dentists and dental hygienists. Assessing for oral cancer risk factors is recommended as part of a clinical protocol.[13] In the case of another HPV-caused cancer, cervical cancer, screening through detection of cytologic alterations induced by HPV infection (the Papanicolaou test) and, direct HPV deoxyribonucleic acid detection, has reduced the incidence of cervical cancer in the United States by 80% over the last several decades.[14] Whether analogous screening interventions—namely, screening for a risk factor (HPV) rather than the disease of primary concern (cancer)—are able to help stem the rising tide of HPV-positive oral cancer is unknown.