HHS Public Access

Author manuscript

Oral Surg Oral Med Oral Pathol Oral Radiol. Author manuscript; available in PMC 2016 July 01.

Published in final edited form as:

Oral Surg Oral Med Oral Pathol Oral Radiol. 2015 July ; 120(1): 34–42. doi:10.1016/j.oooo.2015.04.004.

Self-reported Oral Health, Oral Hygiene, and Oral HPV Infection in At-Risk Women in Ho Chi Minh City, Vietnam

Thanh Cong Bui, Dr.P.H.[1], **Ly Thi-Hai Tran, Ph.D.**[2], **Christine M. Markham, Ph.D.**[3], **Thuy Thi-Thu Huynh, M.D., Ph.D.**[4], **Loi Thi Tran, M.D., Ph.D.**[5], **Vy Thi-Tuong Pham, M.D.**[6], **Quan Minh Tran**[6], **Ngoc Hieu Hoang, M.D.**[7], **Lu-Yu Hwang, M.D.**[2], and **Erich Madison Sturgis, M.D., M.P.H.**[8]

[1]Department of Behavioral Science, The University of Texas MD Anderson Cancer Center, Houston, Texas, United States of America

[2]Department of Epidemiology, School of Public Health, The University of Texas Health Science Center at Houston, Houston, Texas, United States of America

[3]Department of Health Promotion and Behavioral Sciences, School of Public Health, The University of Texas Health Science Center at Houston, Houston, Texas, United States of America

[4]Tu Du Hospital of Obstetrics and Gynecology, Ho Chi Minh City, Vietnam

[5]Department of Obstetrics and Gynecology, Faculty of Medicine, Vietnam National University in Ho Chi Minh City, Ho Chi Minh City, Vietnam

[6]Pham Ngoc Thach University of Medicine, Ho Chi Minh City, Vietnam

[7]Biochemistry Department, University of Mississippi Medical Center, Jackson, Mississippi, United States of America

[8]Department of Head and Neck Surgery, Division of Surgery, The University of Texas MD Anderson Cancer Center, Houston, Texas, United States of America

Abstract

Corresponding author: THANH CONG BUI, Instructor, Department of Behavioral Science, The University of Texas MD Anderson Cancer Center, P.O. Box 301439, Houston, Texas 77230-1439, USA, tcbui@mdanderson.org, thanh.bui@aya.yale.edu, T: 713-745-5542, F: 713-745-4286.

Conflict of Interest Disclosures: The authors declared no potential conflicts of interest relevant to this study.

Prior or upcoming presentation of abstracts at meetings regarding the study: None.

Author contributions: *Study design:* Bui, Ly Tran, Loi Tran, Huynh. *Data collection:* Bui, Ly Tran, Pham, Quan Tran, Loi Tran, Huynh. *Analysis and interpretation of data:* Bui, Ly Tran, Markham, Hwang, Sturgis. *Writing, review, and/or revision of the manuscript:* Bui, Ly Tran, Markham, Loi Tran, Huynh, Hwang, Sturgis. *HPV testing:* Hoang. *Administrative, technical, or material support:* Markham, Loi Tran, Huynh, Hwang. Bui had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Objectives—This study aimed to examine the relationship between self-reported oral health, oral hygiene practices, and oral human papillomavirus (HPV) infection among women at risk for sexually transmitted infections in Ho Chi Minh City, Vietnam.

Study design—Convenience and referral sampling methods were used in a clinic-based setting to recruit 126 women aged 18–45 years between August–October 2013. Behavioral factors were self-reported. Oral-rinse samples were tested for HPV DNA of two low-risk and 13 high-risk genotypes.

Results—A higher unadjusted prevalence of oral HPV infection was associated with poorer self-rated overall oral health (p=.001), reporting oral lesions/problems in the past year (p=.001), and reporting a tooth loss not because of injury (p=.001). Higher unadjusted prevalence of oral HPV infection was also associated with two measures of oral hygiene: lower frequencies of toothbrush per day (p=.047) and gargling without toothbrush (p=.037). After adjusting for other factors in multivariable logistic regression models, poorer self-rated overall oral health remained statistically associated with oral HPV infection (p=.042); yet, the frequency of toothbrush per day did not (p=.704).

Conclusion—Results corroborate the association between self-reported poor oral health and oral HPV infection. The effect of oral hygiene on oral HPV infection remains inconclusive.

Keywords

oral HPV infection; oral health; oral hygiene; oral sex; oropharyngeal neoplasms; oral cancer; head and neck cancer

INTRODUCTION

Research has shown that oral infection with high-risk (i.e. oncogenic) types of human papillomavirus (HPV) is etiologically associated with oropharyngeal squamous cell carcinoma.[1–3] In a systematic review, oral HPV was also associated with potentially malignant disorders, such as leukoplakia, oral lichen planus, or epithelial dysplasia.[4] The prevalence of oral HPV infection ranges from 1.3–9.2% in the general population,[5–9] and is 2–3 times higher in HIV-positive populations.[10–13] HPV 16 is often the most common type identified.[5, 6] Risk factors for oral HPV infection which have been consistently identified in several studies include cigarette smoking, number of lifetime sex partners, and performing oral sex behaviors.[2, 3, 5, 6, 13–16] Other less consistently found risk factors are age, biological sex, alcohol consumption, and open-mouth kissing.

Both oral health and oral hygiene have been associated with oral and oropharyngeal cancers.[3] An increased risk of these cancers is associated with indicators of poor oral health (e.g. tooth loss, irregular dental check-ups),[3] and with indicators of poor oral hygiene (e.g. less frequent tooth brushing, having visible plaque, having dental caries).[3, 17, 18] Using a sample of 3,439 participants aged 30–69 years from the 2009–2010 National Health and Nutrition Examination Survey in the United States, our previous study showed that poor oral health also elevated the odds of oral HPV infection, independent of smoking status and oral sexual behaviors.[19] Through epithelial wounds in the oral cavity, HPV enters the basal layer of epithelium to establish the infection.[20] Poor oral health, which may include ulcers,

mucosal disruption, or chronic inflammation, may create an entry portal for HPV or may increase the epithelium's susceptibility to HPV.

The relationship between oral hygiene and oral HPV infection remainsrelatively unexplored. Hypothetically, because oral hygiene is fundamental to improve oral health, maintaining good oral hygiene practices may reduce oral health problems and thus may have a protective effect against oral HPV infection. To our knowledge, only one cohort study of 212 male university students (18–24 years old) examined this relationship; and it found a non-significant association between frequency of tooth brushing per week and oral HPV incidence.[8] However, only one indicator of oral hygiene (i.e. tooth brushing) was measured in this study. Another possible mechanism for the relationship between oral hygiene and oral HPV infection is the physical effect of oral rinse in removing HPV or exfoliated cells that contain HPV after exposure. This proposition is supported by the thought that the continuous flow of saliva possibly contributes to a commonly lower HPV prevalence and incidence in the oral region, compared with anogenital sites.[9] If this physical effect is real, using oral rinse shortly after performing oral sex may help wash away HPV DNA or exfoliated cells which contain HPV DNA at the point of exposure, and may reduce subsequent oral HPV infection. This effect is particularly more observable in high-risk populations who have several oral sex partners.

This study aimed to examine the relationship between self-reported oral health, self-reported oral hygiene practices, and oral HPV infection among women at risk for STIs in Ho Chi Minh City (HCMC), Vietnam. Results of this research are important for future interventions to prevent oncogenic HPV infection in the oral cavity.

MATERIALS AND METHODS

Study Population

Women at risk for sexually transmitted infections (STIs) were recruited from a clinic-based cross-sectional study at two gynecology clinics at a national obstetrics/gynecology hospital in HCMC. We employed convenience sampling to recruit all eligible women aged 18–45 years who came to these clinics between August–October 2013. Eligibility criteria, which defined being at risk for STIs, included at least *one of the following*: (1) had ≥ 3 different lifetime sexual partners, (2) had ≥ 2 different sexual partners in the past month, (3) was diagnosed with any STI ≥ 2 times or with ≥ 2 types, (including chlamydia, gonorrhea, syphilis, trichomoniasis, granuloma inguinale, Herpes Simplex Virus, HPV, HIV, and Hepatitis B Virus; and including an STI diagnosis at the time of recruitment), and (4) ever exchanged sex for money or other goods. Even in urban areas in Vietnam, like HCMC, it is uncommon for women in the general population to have multiple sexual partners (e.g. mean lifetime number of sexual partners = 1.1, SD = .5),[21] and to have premarital sex (e.g. about 2.6% in a national Survey Assessment of Vietnamese Youth).[22] All eligible women were invited to take part in the study; three refused to participate due to time conflicts. We additionally employed a snow-ball sampling technique by asking those participants who reported ever trading sex to refer other female sex workers in their network to participate in this study. The total sample size was 126. The study protocol was approved by a local

institutional review board (IRB) (QD/BVTD-2424) and the IRB of The University of Texas Health Science Center at Houston (HSC-SPH-13-0297).

Data collection

Prior to data collection, all participants went through an informed consent process and provided a written consent. Participants underwent a 45-minute face-to-face interview, and then provided an oral rinse specimen for oral HPV testing. Interviewers were nurses and physicians who were trained on how to conduct the interviews and on all related ethical issues. A structured questionnaire, which had been pre-tested with a convenience sample of eight healthcare professionals and 10 women in Vietnam, was used for the interviews. All interviews were conducted in Vietnamese, in private clinic rooms. Each participant received the equivalent of $7 US dollars in compensation for their time. After the interview, participants were instructed to gargle with 10 mL of a common commercial mouthwash for 20 seconds, and then expectorated the specimen into a sterile collection cup. Specimens were transported to the hospital's Biological & Genetic Testing Lab on a daily basis for HPV genotyping. The principal investigators (TB and Ly T) directly supervised all data collection activities in the clinics.

Measures

Primary independent variables included oral health and oral hygiene practices, collected through *self-report* in the interviews. Oral health was measured by self-rated overall oral health on a 5-point Likert scale (poor, fair, so-so, very good, and excellent), number of times having oral lesions/problems in the past year, and having a tooth lost not because of injury.[23] Variables measuring oral hygiene practices comprised the average number of times of toothbrushing per day in the past year, frequency of gargling without toothbrushing in the past year (i.e., beside times of toothbrushing; from 1=never to 5=very often), and the average number of toothbrushing or gargling shortly after performing oral sex (i.e. the woman's mouth contacted male partner's genitals) per 10 occasions of performing oral sex in the past year. Because the distribution of this last variable was either very uncommon (0–3 times) or very common (8–10 times), with very few cases in between, it was dichotomized as always brushing teeth or gargling after performing oral sex or not (yes=8–10 times vs. no) in this analysis. We additionally asked for the number of hours since last tooth brushing or gargling in order to control for potential bias in HPV detection. The primary dependent variable was oral infection with any HPV type(s) (see below). Covariates included age, education level, cigarette smoking status, alcohol use, drug use, ever traded sex, oral sex behaviors, frequency of using a protection (condom/dental dam) in oral sex, lifetime number of vaginal/oral sex partners, and HIV status.

HPV DNA Detection Technique

We used the automated Kingfisher system with DynaBead® (Invitrogen) and detergents (Triton X100, Guadinin thiocyanate - Merck) to extract DNA from collected specimens. DNA-binding beads were then washed by ethanol to remove contaminants. To screen for the existence of HPV DNA, nested polymerase chain reaction (PCR) was used with consensus primers designed on the L1 gene of the HPV DNA (MY09/M11 PCR). After amplification,

PCR products were analyzed by electrophoresis on 2% agarose gels staining with GelRed (Biotium). HPV-positive samples were then genotyped. Amplicons were hybridized onto ELISA plates which were coated with streptavidine and specific genotyped probes in each well (genotypes 6, 11, 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, & 68). Genotype-specific probes bound to complementary denatured amplicons. The resulting hybrids were detected by tetramethyl benzidine coloring after incubation with horseradish-peroxidase - binding monoclonal antibody to digoxigenin. Finally, absorbance was read using the iMark™ Microplate Reader (Biorad) at 450nm. The variable of oral HPV infection was coded as positive if any of the 2 low-risk (6 & 11) or 13 high-risk (the remaining in the above list) HPV DNA types were detected.

Data analysis

Bivariate associations between demographic or behavioral variables and oral HPV infection were examined using chi-square tests or binary logistic regression. Due to small numbers of cases responding to some values of self-rated overall oral health, responses to this variable were recoded into three categories: poor-fair, so-so, and very good-excellent. Separate multivariable logistic regression models were used to examine the adjusted associations between primary independent variables (oral health and oral hygiene practices, respectively) and oral HPV outcomes. A directed acyclic graph was used to select covariates to be controlled for in multivariable logistic regression models.[24] A two-sided p-value of <.05 was considered statistically significant.

RESULTS

In our sample, 95.2% were Kinh ethnicity, the major ethnicity in Vietnam. The mean age of participants was 31.9 years (S.D.= 6.2; median= 32). About half of them had not attended high school (Table 1). Seventy-two percent had ever performed oral sex, and 37.3% reported ever trading sex for money, drugs, or other in-kind exchange. The prevalence of those who currently smoked and ever used drugs was 16.7% and 13.0%, respectively; most of these were in the subgroup reporting ever traded sex (all p values <.001, data not shown). The prevalence of HIV-positive participants was 6.3% (1.3% in those who never traded sex, and 14.9% in those who ever traded sex, p=.009). The prevalence of self-rated overall oral health was 17.6% for poor or fair, 43.2% for so-so, and 39.2% for very good or excellent. The majority of participants (68.3%) brushed their teeth on average >1–2 times per day in the past year; 11.9% brushed their teeth 1 time or less per day. In addition to toothbrushing, 22.2% participants reported that they gargled sometimes and 35.7% gargled often or very often. Among those who gargled without toothbrushing (n=81), 75.3% used water only, 17.3% used water with salt, and the remaining (7.4%) used commercial mouthwash. Among those who had ever performed oral sex (n=91), 58.9% always brushing teeth or gargling after performing oral sex.

The overall prevalence of oral infection with any HPV type was 24.6% (31/126), and with any high-risk HPV type (i.e. excluding 6 & 11) was 16.7% (21/126). In the subgroup of those who ever traded sex, the prevalence of oral HPV infection was 48.9% for any type and 38.3% for high-risk types. Among those who had any-type oral HPV infection, one case was

infected with two types and one case was infected with three types. Among those who had oral infection with a high-risk type of HPV (n=21), the most common types were 52 (n=10, 47.6%), 58 (n=7, 33.3%), and 16 (n=3, 14.3%). No participant had prior HPV immunization.

Table 1 displays bivariate associations between any-type oral HPV infection and participants' characteristics. Higher prevalence of oral HPV infection was found in those who currently smoked, drank alcohol in the past 90 days, ever used drugs, ever performed oral sex, first performed oral sex at a younger age, had a higher number of lifetime vaginal sex partners, had a higher number of lifetime partners on whom participants performed oral sex, and ever traded sex. Among those who ever performed oral sex, 94.5% never used any protection; this was not associated with oral HPV infection, which might be due to the small sample of those who ever used protection. Higher prevalence of oral HPV infection was also associated with all three measures of self-reported oral health, including self-rated poorer oral health (p=.001), having oral lesions/problems in the past year (p=.001), and having tooth loss not because of injury (p=.001). Regarding oral hygiene practices, higher frequency of tooth brushing per day (p=.047) and gargling without toothbrushing (p=.037) were associated with a lower risk of oral HPV infection in bivariate analysis. Always brushing teeth or gargling after performing oral sex (p=.175) and time since last tooth brushing or gargling (p=.801) were not associated with oral HPV detection.

We built separate multivariable logistic regression models to further examine the associations between self-reported oral health, oral hygiene, and oral HPV infection when controlling for other factors (Table 2). Self-rated overall oral health (Model 1) was selected to represent oral health in this report, as in our previous work,[19] and average number of toothbrushing per day in past year (Model 2) to represent oral hygiene. Oral hygiene and oral health were examined in separate models because oral health might be an intermediate between oral hygiene and oral HPV infection. Based on *a priori* knowledge and directed acyclic graphs, we controlled for ever traded sex, ever performed oral sex, and smoking status. Smoking was not included in the Model 2 because it cannot be a cause of the primary independent variable of oral hygiene. Lifetime numbers of oral/vaginal sexual partners were not included because these were strongly related to and were a descendant of ever traded sex. Although frequency of toothbrushing per day in the past year was strongly associated with self-rated overall oral health in bivariate analysis (p<.001, data not shown), results in the multivariable logistic regression models indicated that self-rated overall oral health (p=.042) but not frequency of toothbrushing per day (p=.704) remained associated with oral HPV infection.

DISCUSSION

To our knowledge, this study is the first to report on oral HPV infection and associated risk factors in Vietnam. In this group of at-risk women in HCMC, our data showed that oral HPV infection was common. About one-fourth were infected with at least one HPV type, and 16.7% were infected with one of the 13 high-risk types which could be detected by our testing technique. These figures are slightly lower than the prevalence of oral HPV infection in other at-risk populations, such as in the United States (34.0%).[11] However, in the subgroup of those who ever traded sex, the prevalence of oral HPV infection doubled,

mirroring the high prevalence of genital HPV infection (from 42.5%–85.0%) in this specific group in Vietnam.[25, 26]

Risk factors for oral HPV infection in our sample were consistent with risk factors found in previous studies, including smoking status, ever performed oral sex, first performed oral sex at a younger age, lifetime number of vaginal sex partners, and lifetime number of oral sex partners.[5, 11, 14] Most previous studies have documented a strong association between oral HPV infection and HIV-positive status due to immunosuppression (e.g. see Beachler et al., 2012)[11]. However, this association was not statistically significant in our sample, likely due to a very limited number of HIV-positive cases. Given that oral high-risk HPV infection has been established as a cause for a subset of oropharyngeal cancers, and that this infection was significantly more prevalent in those who reported trading sex, interventions for sex-worker groups may be needed to prevent long-term burden of oropharyngeal cancers.

Our results showed that self-reported oral health measures, particularly self-rated overall oral health, were significantly associated with oral HPV infection. The association between self-rated overall oral health and oral HPV infection remained significant in the multivariable model, after controlling for other known risk factors (e.g., performing oral sex, smoking). This suggests that self-reported poor oral health is an independent risk factor for oral HPV infection, regardless of trading-sex status, performing oral sex, and smoking status. This finding is consistent with the results from our previous work, which were the first to suggest the tie between oral health and oral HPV infection, irrespective of smoking and performing oral sex.[19]

Our study provides initial evidence regarding possible links between a variety of self-reported oral hygiene practices and oral HPV infection. As mentioned above, only one previous study examined and found a non-significant association between tooth brushing and oral HPV infection in univariate analysis.[8] Our results showed that lower frequency of tooth brushing or gargling without toothbrushing per day were associated with higher oral HPV prevalence in bivariate analysis; yet these associations no longer existed when controlling for other risk factors. The association between self-reported oral health, but not oral hygiene, and oral HPV infection may be due to the susceptibility directly caused by these two factors. It is presumed that HPV infects the basal layer of epithelium in the oral cavity through epithelial wounds,[20] which may be caused by poor oral health and possibly associated with self-reported poor oral health. Poor oral hygiene, although significantly related to self-reported poor oral health in this analysis, may not directly create an entry portal for HPV such as ulcers or mucosal disruption.

This study is also the first to examine the association between oral hygiene after performing oral sex and oral HPV infection. Although some previous studies have suggested the preventive role of certain sexual hygiene habits on HPV infectivity, such as penile cleaning after sex to reduce genital HPV infection in men,[27] this study did not find an association between tooth brushing or gargling after performing oral sex and oral HPV status. Even when this association was stratified by whether participants had oral lesion(s) in the past year, no significant association was observed ($p>.460$, data not shown). These results suggest that oral hygiene practices may not have a direct effect on oral HPV infectivity.

Alternatively, the non-associations may be due to the fact that most HPV types detected in our test were mucosal, which could not be easily washed away (and hence reduced in viral load) by tooth brushing or oral rinse after sex. Another alternative is the insufficient statistical power to detect such an association due to small sample size. Similarly, the number of hours since the last toothbrushing or gargling was not associated with oral HPV detection (p=.914). On one hand, this suggests that collection of oral samples using oral rinse for HPV testing was not affected by the most recent time of toothbrushing or gargling. On the other hand, this finding suggests that oral HPV infection might not be prevented by one-time tooth brushing or oral rinse, independent of oral health status. Further research, particularly prospective studies, is needed to elucidate the effect of oral hygiene practices on oral HPV infectivity and incidence (rather than prevalence). In future studies, it is also necessary to separate measures of oral rinse after oral sex (which may physically remove HPV DNA or cells containing HPV DNA) and measures of toothbrushing (which may cause micro-abrasions and thus create an entry portal for HPV). Understanding the roles of oral hygiene practices on oral HPV infection is important because these behavioral factors are modifiable.

This study was not without limitations, and the results were preliminary. The cross-sectional design engenders ambiguity in temporal relationships between variables. However, because of its asymptomatic status,[20, 28] oral HPV infection was unlikely to affect self-reported oral health and oral hygiene. Additionally, these cross-sectional data did not enable investigation of newly infected oral HPV (i.e., incidence) and persistent oral HPV infection. Even if oral hygiene practices are not associated with oral HPV prevalence, they may have a preventive effect on HPV incidence. While the HPV type-specific test used in our study was approved as an in vitro diagnostic product for clinical use for cervical HPV testing in Vietnam, no validating or comparative analysis has been performed for its results with oral specimens. The small sample size might have reduced statistical power to examine associations of interest, especially in multivariable analyses. Self-report of oral health may not be as reliable as dental examination indicators to reflect actual oral health status. Regarding smoking, we only asked for cigarette smoking. However, this was the most common tobacco product used in Vietnam, and the prevalence of using other tobacco products (e.g., water pipes, betel) was <1% in Vietnamese women aged <45.[29] Lastly, our findings pertain to this at-risk group of women and may not be generalizable to women in the general or other populations. Despite these limitations, this study provides important findings regarding the possible roles of oral health and oral hygiene practices on oral HPV infection. While the links between oral hygiene, oral health and oral HPV infection are speculative in nature, the observed links in this study point to the potential importance of these factors and thus call for further work on this topic.

CONCLUSION

Overall, these preliminary results corroborate the association between self-reported poor oral health and an increased risk of oral HPV infection. Thus, improving oral health may help prevent oral HPV infection and oral HPV-related diseases. The effect of oral hygiene practices on oral HPV infection, however, remains inconclusive. Further studies, particularly prospective ones, are necessary to elucidate the role of oral hygiene practices on preventing

oral HPV infection. This study also revealed that oral HPV infection was common in Vietnamese women at risk for STIs. Thus, future studies on this topic or on oropharyngeal cancers outcomes in Vietnam are needed.

Acknowledgments

Financial support: This study was supported by the University of Texas Health Science Center at Houston, School of Public Health, Center for International Training on AIDS Research (externally funded by National Institutes of Health - Fogarty International Center, AIDS International Training and Research Program, D43 TW007669), and by the Margaret McNamara Memorial Fund. Thanh C. Bui was supported by the UTHealth Innovation for Cancer Prevention Research post-doctoral fellowship, Cancer Prevention and Research Institute of Texas (CPRIT) grant #RP101503, and is supported by a faculty fellowship from The University of Texas MD Anderson Cancer Center Duncan Family Institute for Cancer Prevention and Risk Assessment. Ly T. Tran's education was supported by the Vietnam Education Foundation Fellowship, Philanthropic Educational Organization International Peace Scholarship, and American Association of University Woman. The content of the manuscript is solely the responsibility of the authors and does not necessarily represent the official views of the funding agencies.

References

1. Gillison ML, Koch WM, Capone RB, et al. Evidence for a Causal Association Between Human Papillomavirus and a Subset of Head and Neck Cancers. Journal of the National Cancer Institute. 2000; 92:709–720. [PubMed: 10793107]
2. Marur S, D'Souza G, Westra WH, Forastiere AA. HPV-associated head and neck cancer: a virus-related cancer epidemic. The Lancet Oncology. 2010; 11:781–789. [PubMed: 20451455]
3. Gillison ML. Current topics in the epidemiology of oral cavity and oropharyngeal cancers. Head & Neck. 2007; 29:779–792. [PubMed: 17230556]
4. Syrjanen S, Lodi G, Bultzingslowen IV, et al. Human papillomaviruses in oral carcinoma and oral potentially malignant disorders: a systematic review. Oral Diseases. 2011; 17:58–72. [PubMed: 21382139]
5. Gillison ML, Broutian T, Pickard RKL, et al. Prevalence of oral HPV infection in the united states, 2009–2010. JAMA. 2012; 307:693–703. [PubMed: 22282321]
6. Kreimer AR, Bhatia RK, Messeguer AL, Gonzalez P, Herrero R, Giuliano AR. Oral Human Papillomavirus in Healthy Individuals: A Systematic Review of the Literature. Sexually Transmitted Diseases. 2010; 37
7. Kreimer AR, Villa A, Nyitray AG, et al. The Epidemiology of Oral HPV Infection among a Multinational Sample of Healthy Men. Cancer Epidemiology Biomarkers & Prevention. 2011; 20:172–182.
8. Edelstein ZR, Schwartz SM, Hawes S, et al. Rates and Determinants of Oral Human Papillomavirus Infection in Young Men. Sexually Transmitted Diseases. 2012; 39:860–867.10.1097/OLQ.1090b1013e318269d318098 [PubMed: 23064535]
9. Pickard RKL, Xiao W, Broutian TR, He X, Gillison ML. The Prevalence and Incidence of Oral Human Papillomavirus Infection Among Young Men and Women, Aged 18–30 Years. Sexually Transmitted Diseases. 2012; 39:559–566.10.1097/OLQ.1090b1013e31824f31821c31865 [PubMed: 22706220]
10. Beachler DC, D'Souza G, Sugar EA, Xiao W, Gillison ML. Natural History of Anal vs Oral HPV Infection in HIV-Infected Men and Women. Journal of Infectious Diseases. 2013; 208:330–339. [PubMed: 23596319]
11. Beachler DC, Weber KM, Margolick JB, et al. Risk factors for oral HPV infection among a high prevalence population of HIV-positive and at-risk HIV-negative adults. Cancer epidemiology, biomarkers & prevention : a publication of the American Association for Cancer Research, cosponsored by the American Society of Preventive. Oncology. 2012; 21:122–133.
12. D'Souza G, Fakhry C, Sugar EA, et al. Six-month natural history of oral versus cervical human papillomavirus infection. International Journal of Cancer. 2007; 121:143–150.

13. Kreimer AR, Alberg AJ, Daniel R, et al. Oral Human Papillomavirus Infection in Adults Is Associated with Sexual Behavior and HIV Serostatus. Journal of Infectious Diseases. 2004; 189:686–698. [PubMed: 14767823]
14. D'Souza G, Agrawal Y, Halpern J, Bodison S, Gillison ML. Oral sexual behaviors associated with prevalent oral human papillomavirus infection. J Infect Dis. 2009; 199:1263–1269. [PubMed: 19320589]
15. Chaturvedi AK, Engels EA, Anderson WF, Gillison ML. Incidence Trends for Human Papillomavirus-Related and -Unrelated Oral Squamous Cell Carcinomas in the United States. Journal of Clinical Oncology. 2008; 26:612–619. [PubMed: 18235120]
16. Heck JE, Berthiller J, Vaccarella S, et al. Sexual behaviours and the risk of head and neck cancers: a pooled analysis in the International Head and Neck Cancer Epidemiology (INHANCE) consortium. International Journal of Epidemiology. 2010; 39:166–181. [PubMed: 20022926]
17. Guha N, Boffetta P, Wünsch Filho V, et al. Oral Health and Risk of Squamous Cell Carcinoma of the Head and Neck and Esophagus: Results of Two Multicentric Case-Control Studies. American Journal of Epidemiology. 2007; 166:1159–1173. [PubMed: 17761691]
18. Rosenquist K, Wennerberg J, Schildt EB, Bladstrom A, Goran Hansson B, Andersson G. Oral status, oral infections and some lifestyle factors as risk factors for oral and oropharyngeal squamous cell carcinoma. A population-based case-control study in southern Sweden. Acta Oto-laryngologica. 2005; 125:1327–1336. [PubMed: 16303683]
19. Bui TC, Markham CM, Ross MW, Mullen PD. Examining the Association between Oral Health and Oral HPV Infection. Cancer Prevention Research. 2013; 6:917–924. [PubMed: 23966202]
20. Rautava J, Syrjanen S. Human papillomavirus infections in the oral mucosa. The Journal of the American Dental Association. 2011; 142:905–914. [PubMed: 21804057]
21. Vaccarella S, Franceschi S, Herrero R, et al. Sexual behavior, condom use, and human papillomavirus: pooled analysis of the IARC human papillomavirus prevalence surveys. Cancer epidemiology, biomarkers & prevention : a publication of the American Association for Cancer Research, cosponsored by the American Society of Preventive. Oncology. 2006; 15:326–333.
22. Ministry of Health of Vietnam. Survey Assessment of Vietnamese Youth (SAVY). Hanoi: Ministry of Health of Vietnam, General Statistics Office of Vietnam, United Nations Children's Fund, World Health Organization; 2005.
23. Centers for Disease Control & Prevention. National Health and Nutrition Examination Survey: 2009–2010 Data Documentation, Codebook, and Frequencies: Oral Health. 2012
24. Glymour, M.; Greenland, S. Causal Diagrams. In: Rothman, K.; Greenland, S., editors. Modern Epidemiology. Philadelphia: Lippencott-Raven; 2008. p. 183-209.
25. Hernandez B, Vu Nguyen T. Cervical human papillomavirus infection among female sex workers in southern Vietnam. Infectious Agents and Cancer. 2008; 3:7. [PubMed: 18433504]
26. Hoang HTT, Ishizaki A, Nguyen CH, et al. Infection with high-risk HPV types among female sex workers in northern Vietnam. Journal of Medical Virology. 2013; 85:288–294. [PubMed: 23161344]
27. Widdice LE, Breland DJ, Jonte J, et al. Human Papillomavirus Concordance in Heterosexual Couples. Journal of Adolescent Health. 2010; 47:151–159. [PubMed: 20638007]
28. Edwards S, Carne C. Oral sex and the transmission of viral STIs. Sexually Transmitted Infections. 1998; 74:6–10. [PubMed: 9634307]
29. Ministry of Health of Vietnam, Centers for Disease Control and Prevention. Global Adult Tobacco Survey (GATS) Vietnam 2010. Hanoi, Vietnam: 2010.

STATEMENT OF CLINICAL RELEVANCE

This study suggests that self-reported poor oral health may be associated with an increased risk of oral oncogenic HPV infection. Thus, improving oral health, particularly among high-risk populations, may help prevent oral HPV infection and subsequent oral HPV-related diseases.

Table 1

Prevalence of oral infection with any HPV type by demographic and behavioral characteristics

Characteristics	Total, n (%)	Oral HPV infection	
		n (%)	Unadjusted odds ratios (95% CI)
Total	126	31 (24.6)	
Age			
19–25	20 (15.9)	8 (40.0)	3.22 (.92–11.29)
26–35	71 (56.3)	17 (23.9)	1.52 (.54–4.28)
36–45	35 (27.8)	6 (17.1)	1[a]
p value (for trend)		.163 (.074)	
Education level			
Secondary school or lower	61 (48.8)	17 (27.9)	1
High school or higher	64 (51.2)	13 (20.3)	.66 (.29–1.51)
p-value		.323	
Currently smoked[b]			
Yes	21 (16.7)	11 (52.4)	**4.68 (1.75–12.52)**
No	105 (83.3)	20 (19.0)	1
p-value		**.001**	
Average numbers of drinks per week in the past 90 days			
0	74 (59.7)	12 (16.2)	1
<1 drink/wk	22 (17.7)	8 (36.4)	**2.95 (1.02–8.57)**
1–5 drinks/wk	17 (13.7)	5 (29.4)	2.15 (.64–7.24)
>5 drinks/wk	11 (8.9)	6 (54.5)	**6.20 (1.63–23.63)**
p-value (for trend)		**.020 (.006)**	
Ever used drugs			
Yes	16 (13.0)	9 (56.2)	**5.59 (1.86–16.82)**
No	107 (87.0)	20 (18.7)	1
p-value		**.001**	
Ever performed oral sex			
Yes	91 (72.2)	27 (29.7)	**3.27 (1.05–10.17)**
No	35 (27.8)	4 (11.4)	1
p-value		**.033**	
Age of first performing oral sex[c]			
15–20	18 (19.8)	9 (50.0)	**4.13 (1.28–13.75)**
21–25	32 (35.2)	10 (31.2)	1.88 (.64–5.49)
>25	41 (45.1)	8 (19.5)	1
p-value (for trend)		.060 **(.042)**	
Lifetime number of vaginal-sex partners			
1	49 (38.9)	1 (2.0)	1
2	16 (12.7)	3 (18.8)	**11.08 (1.06–115.53)**
3–10	24 (19.0)	9 (37.5)	**28.80 (3.37–246.21)**
>10	37 (29.4)	18 (48.6)	**45.47 (5.67–364.93)**

Characteristics	Total, n (%)	Oral HPV infection	
		n (%)	Unadjusted odds ratios (95% CI)
p-value		**<.001 (<.001)**	
Lifetime number of partners on whom participants performed oral sex[C]			
1	41 (45.1)	3 (7.3)	1
2	8 (8.8)	3 (37.5)	**7.6 (1.19–48.44)**
3–10	17 (18.7)	7 (41.2)	**8.87 (1.94–40.60)**
>10	25 (27.5)	14 (56.0)	**16.12 (3.91–66.43)**
p-value		**<.001 (<.001)**	
Ever traded sex			
Yes	47 (37.3)	23 (48.9)	**8.51 (3.36–21.52)**
No	79 (62.7)	8 (10.1)	1
p-value		**<.001**	
History of vaginal STIs (excluding HIV)			
Yes	34 (27.0)	21 (22.8)	1.41 (.58–3.41)
No	92 (73.0)	10 (29.4)	1
p-value		.446	
HIV status			
Positive	8 (6.3)	2 (25.0)	1.33 (.24–7.39)
Negative	65 (51.6)	13 (20.0)	1
Unknown/undisclosed	53 (42.1)	16 (30.2)	1.73 (.74–4.03)
p-value		.442	
Self-rated overall oral health			
Poor-fair	22 (17.6)	11 (50.0)	**7.17 (2.17–23.68)**
So-so	54 (43.2)	13 (24.1)	2.27 (.79–6.54)
Very good-excellent	49 (39.2)	6 (12.2)	1
p-value		**.003 (.001)**	
Number of times having oral lesions/problems in past year			
None	50 (40.3)	6 (12.0)	1
1–2 times	38 (30.6)	8 (21.1)	1.96 (.62–6.21)
3–4 times	16 (12.9)	7 (43.8)	**5.70 (1.55–21.03)**
5 times or more	20 (16.1)	9 (45.0)	**6.00 (1.76–20.45)**
p-value		**.006 (.001)**	
Having a tooth lost not because of injury			
Yes	38 (30.2)	17 (44.7)	**4.28 (1.82–10.09)**
No	88 (69.8)	14 (15.9)	1
p-value		**.001**	
Average number of toothbrushing per day in past year			
1 or less	15 (11.9)	6 (40.0)	**4.89 (1.00–23.93)**
>1–2	86 (68.3)	22 (25.6)	2.52 (.69–9.25)
>2	25 (19.8)	3 (12.0)	1
p-value (for trend)		.129 **(.047)**	
Frequency of gargling without toothbrushing			

Characteristics	Total, n (%)	Oral HPV infection	
		n (%)	Unadjusted odds ratios (95% CI)
Never/rarely	53 (42.1)	18 (34.0)	**2.79 (1.04–7.49)**
Sometimes	28 (22.2)	6 (21.4)	1.48 (.44–5.00)
Often/very often	45 (35.7)	7 (15.6)	1
p-value		.098 **(.037)**	
Always brushing teeth or gargling after performing oral sex[c]			
Yes	53 (58.9)	13 (24.5)	.53 (.21–1.33)
No	37 (41.1)	14 (37.8)	1
p-value		.175	
Number of hours since last tooth brushing or gargling			
≤2	23 (18.3)	5 (21.7)	.83 (.23–3.09)
3–4	44 (34.9)	11 (25.0)	1.00 (.34–2.99)
5–6	31 (24.6)	8 (25.8)	1.04 (.32–3.38)
≥7	28 (22.2)	7 (25.0)	1
p-value (for trend)		.988 (.801)	

[a] An OR=1 indicates the reference group.

[b] Defined as ever smoked ≥ 100 cigarettes lifetime and smoked in the past 30 days.

[c] Among those who ever performed oral sex, n=91.

Table 2

Adjusted odds ratios (OR) for oral HPV infection in multivariable logistic regression models[a]

	Model 1		Model 2	
Independent variables	**OR (95% CI)**	**p-values**	**OR (95% CI)**	**p-values**
Ever traded sex				
Yes	4.53 (1.48–13.92)	**.001**	7.64 (2.85–20.52)	**<.001**
No	1[b]		1	
Ever performed oral sex				
Yes	3.21 (.83–12.45)	.090	2.35 (.69–8.00)	.173
No	1		1	
Currently smoked				
Yes	1.49 (.43–5.12)	.530		
No	1			
Self-rated overall oral health		**.042**[c]		
Poor-fair	4.12 (1.05–16.15)	**.042**		
So-so	2.01 (.65–6.23)	.227		
Very good-excellent	1			
Average number of toothbrushing per day in past year				.704[c]
1 or less			1.45 (.25–8.50)	.681
>1–2			1.80 (.44–7.38)	.412
>2			1	

[a] Dependent variable: oral infection with any HPV type, 1=yes.

[b] An OR=1 indicates the reference group.

[c] p-value for trend when all values in the according independent variable were considered ordinal.