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LETTERS

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Abstract

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PERIODONTITIS AND ORAL HUMAN PAPILLOMAVIRUS

We read with great interest Dr. R. Constance Wiener and colleagues' original contribution in June JADA, "Association of Periodontitis and Human Papillomavirus in Oral Rinse Specimens: Results From the National Health and Nutrition Survey 2009–2012" (*JADA*. 2015;146 [6]:382–389).

The authors analyzed data from the 2009–2012 National Health and Nutrition Examination Survey (NHANES) and observed a statistically significant crude effect of periodontitis on odds of oral human papillomavirus (HPV) infection (odds ratio [OR], 2.25; 95% confidence interval [CI], 1.61–3.13). However, once adjusted for potential confounding, the relationship was rendered statistically nonsignificant (adjusted OR, 1.04; 95% CI, 0.63–1.73). Based on this evidence, the authors concluded that periodontitis was unrelated to oral HPV infection in US adults. We agree with this interpretation of results based on this analysis.

Nonetheless, we believe that opportunities were missed for greater analytic scrutiny of this cross-sectional association. For example, rather than collapsing the Centers for Disease Control and Prevention (CDC) and American Academy of Periodontology (AAP) case definitions for chronic periodontitis severity into a binary state, it is more informative to capitalize on their ordinal properties to explore a possible dose-response effect.¹

Our own unpublished analysis of these NHANES data using the CDC and AAP case definitions found a dose-response relationship ($P_{\text{trend}} < .001$) between chronic periodontitis severity and odds of oral HPV that was statistically significant in women, but not in men, suggesting an effect modification (adjusted $P_{\text{effect modification}} = .074$). We found that compared with women without periodontitis, the adjusted odds of oral HPV infection were 2.2 times as great for women with mild or moderate periodontitis (95% CI, 1.1–4.3; $P = .025$). Furthermore, the odds of women with severe periodontitis having detectable oral HPV were 3.5 times as great (95% CI, 1.3–9.6; $P = .018$) as those of women without periodontitis.

By contrast, the odds of men with mild or moderate periodontitis having oral HPV were no greater than those of men without periodontitis (OR, 1.1; 95% CI, 0.7–1.8; $P = .528$) and, likewise, the odds of men with severe periodontitis having oral HPV of 1.2 (95% CI, 0.6–2.6; $P = .615$) were no greater than those without periodontitis. Our analysis adjusted for similar confounding sociodemographic and behavioral risk factors reported by the authors.

Apart from dose-response and effect modification possibilities, a third factor contributing to the null association reported by the authors may be bias arising from nonrandom missing data. The authors do not describe how missing data were handled. According to Table 1 in the article, information was missing on poverty-income ratio for 521 adults, lifetime number of sex partners for 764 adults, and consumption of alcoholic drinks for 1,139 adults. By default, multivariable models omit observations (for example, adults) that are missing data on any variable entered into the model. All 3 variables were used in the third model of the authors (Table 2 in the article) that found a null association. This raises the possibility that these missing data in model 3 accounted for more of the attenuation in effect size from the crude association than did confounding, because these 3 variables were not strongly associated with oral HPV.

We are pleased that Dr. Wiener and her colleagues have raised this important topic. As statistical power increases with the release of forthcoming NHANES cycles, we look forward to new insights gleaned from analysis of other periodontitis traits, their relationships with oncogenic oral HPV strains, and reasons for possible sex differences in this relationship.

Acknowledgments

We thank Drs. Sanders, Patton, and Webster-Cyriaque for their interest, responses, and comments on our article. National Health and Nutrition Examination Survey (NHANES) 2009–2010 data were the first NHANES data sets with full-mouth periodontal data. We combined those years with data from NHANES 2011–2012, and our sample size for human papillomavirus (HPV)-positive participants was 498. We considered our sample size for mild periodontal disease in the participants who were HPV positive to be too small as an ordinal category in polytomous analysis, although we agree that having such information would have been very informative, and we appreciate you sharing your results.

Our interaction studies of sex with dichotomized periodontal disease on HPV status were not significant. In determining the sample, we included participants, aged 30–69 years, who had all periodontal and HPV data. We agree that missing data may influence the results in the adjusted models; therefore, we presented the 3 adjusted models for consideration. We agree with Dr. Sanders and colleagues' statement that as statistical power increases with additional NHANES cycles, we will be able to have more insights about the relationships and hope that with available HPV vaccines, the number of HPV-positive participants will decline.

References

1. Eke PI, Page RC, Wei L, Thornton-Evans G, Genco RJ. Update of the case definitions for population-based surveillance of periodontitis. *J Periodontol*. 2012; 83(12):1449–1454. [PubMed: 22420873]