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Researchers find link between tobacco use and viral infection that causes oral cancers

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Johns Hopkins scientists have shown a strong association between tobacco use or exposure and infection with oral human papillomavirus type 16 (HPV16), the sexually transmitted virus responsible for mouth and throat cancers worldwide. The numbers of such cancers have increased 225 percent in the United States over the past two decades.

HPV16 is found in 80 percent of cancers located in the back of the throat and is transmitted through oral sex. "The practice of oral sex is common, but this cancer is rare. So there must be cofactors in the process that explain why some people develop persistent HPV16 infections and HPV-positive oropharyngeal cancers when most other people don't," says Gypsyamber D'Souza, Ph.D., M.S., M.P.H., an associate professor of epidemiology at the Johns Hopkins Bloomberg School of Public Health and Johns Hopkins Kimmel Cancer Center.

The research team found that HPV16 infection is more common among people who have recently used or been exposed to tobacco, independent of their sexual behavior, according to authors of the new study published in the Oct. 7 issue of *JAMA*.

"It appears that tobacco exposure increases the likelihood of having oral HPV16 infection, and although we do not yet know why, we suspect that the virus may not be cleared from the body as easily in people who use tobacco," says D'Souza.

But the researchers caution that although the study shows an independent

relationship between tobacco and HPV16 infection, they cannot fully rule out the possibility that people who use more tobacco might also have more oral sex, and therefore have a higher risk of HPV16 infection.

Previous studies have shown an association between oral HPV16 infection and cigarette use. The new study takes this reported association a step further by looking at oral HPV16 infection among people who tested positive for the presence of tobacco-linked chemicals in their blood or urine, which can come from any tobacco source – even secondhand smoke, says Carole Fakhry, M.D., M.P.H., an assistant professor of otolaryngology-head and neck surgery at the Johns Hopkins University School of Medicine.

While the current study links tobacco use and exposure as a risk factor for oral HPV16 infection, the researchers emphasize that smoking or other forms of tobacco use do not directly cause HPV16 infection, and that nonsmokers can still get HPV16 infections.

The study's 6,887 participants were drawn from the National Health and Nutrition Examination Survey, which comprises a representative sample of the U.S. population. They included 2,012 who were current tobacco users at the time of the study and 63 who were infected with HPV16.

The virus was detected by a 30-second oral rinse and gargle that collected mouth and throat cells. The participants described their tobacco use in a survey and also had their blood and urine tested for two tobacco-related chemicals, cotinine and NNAL (4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol).

People with higher levels of the tobacco-related biomarkers in their blood and urine were more likely to have oral HPV16 DNA, compared with those who had no detectable levels of the compounds, says Fakhry.

"We found that increasing levels of tobacco exposure were associated with higher odds of oral HPV16 prevalence," she adds.

Each increase in the blood level of cotinine, equivalent to three cigarettes per day, increased the odds of HPV16 prevalence by 31 percent. Each rising level of NNAL detected in urine, the equivalent of four daily cigarettes, increased the odds of HPV16 prevalence by 68 percent. This so-called dose-response curve is considered to be scientifically strong evidence of association between two events.

"These results may provide an additional reason for smoking cessation and suggest that even modest amounts of tobacco use are associated with higher oral HPV prevalence," says Fakhry.

Provided by Johns Hopkins University School of Medicine