Team develops new model for investigating tobacco/oral cancer link

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Although tobacco use is widely understood to be one of the leading causes of oral cancer, research on the prevention of tobacco-related oral cancer in experimental animals has traditionally been limited to examining the impact of synthetic carcinogens manufactured especially for cancer research, rather than on observing the effects of carcinogens that occur in tobacco smoke.

Now, a recently completed study conducted collaboratively by Dr. Joseph Guttenplan, a Professor of Basic Science & Craniofacial Biology at the NYU College of Dentistry, and Dr. Karam El-Bayoumy, a Professor of Biochemistry and Molecular Biology at Penn State University College of Medicine and Associate Director of Basic Research at the Penn State Cancer Institute, has shown that a powerful carcinogen in tobacco smoke can be used for oral cancer research in experimental animals, thus providing a new, more relevant research model with which to understand the initiation, progression, and, ultimately, the prevention of oral cancer. The two-year study was sponsored by the National Institute of Dental and Craniofacial Research, part of the NIH.

In a presentation on April 19 at the 2010 annual meeting of the American Association for Cancer Research (AACR) in Washington, DC, Dr. Guttenplan said the findings could ultimately facilitate research aimed at identifying new approaches to oral cancer prevention.

Oral cancer is a devastating disease that can severely and permanently

compromise one's ability to eat, drink, talk, and even kiss. In the United States, about 100 new cases of oral cancer occur each day, and approximately 7,000 people die annually from the disease. Worldwide, over 640,000 new cases of oral cancer occur annually. In addition to tobacco use, alcohol use and exposure to the HPV-16 virus (human papilloma virus version 16) are the leading causes of oral cancer.

The study described in Dr. Guttenplan's presentation examined the impact of injecting low, medium, and high doses of dibenzo[a,l]pyrene, a powerful carcinogen in tobacco, into the mouths of 104 mice. The researchers examined 24 of the mice for mutagenesis and 80 for carcinogenesis. After 38 weeks, all of the mice in the high-dose mutagenesis group developed excessive numbers of mutations in their oral tissue, and within one year, 31% of the high-dose carcinogenesis group displayed large tumors in their mouths.

"As a result of this study," said Dr. Guttenplan, "we now have a model that is significantly better than past models which relied on synthetic carcinogens. "We plan to use this new model in future studies to examine potential agents for cancer prevention."

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