

Comprehensive genomic analysis identifies alterations in head and neck cancer that could lead to targeted therapy

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Not all head and neck squamous cell carcinomas (HNSCCs) have the same pattern of genomic alterations, but those cancers with certain distinctive patterns could be amenable to specific targeted therapies, according to a researcher who presented the data at the AACR Annual Meeting 2013, held in Washington, D.C., April 6-10.

The study was part of The Cancer Genome Atlas (TCGA), a National Institutes of Health project to catalog the [genetic alterations](#) responsible for several types of cancer, in particular those with a [poor prognosis](#).

"It is likely our study will become a landmark research tool for HNSCC for many years as the secrets included in this massive data set are gradually unlocked," said David N. Hayes, M.D., M.P.H., an associate professor at the University of North Carolina (UNC), who practices otolaryngology at the UNC Lineberger Comprehensive Cancer Center in Chapel Hill, N.C.

Hayes and colleagues conducted comprehensive [genomic analysis](#) of tumor tissue and healthy tissue from 279 patients with previously untreated HNSCC. Eighty percent of the patients reported a history of smoking.

The researchers identified more than 30 sites of significant somatic copy number alteration, or sites of significant change in the number of copies

of a certain gene or genetic region. Most of the sites were identical to those recently identified in lung [squamous cell carcinoma](#).

"HNSCC is a tobacco-related cancer," Hayes said. "We frequently see very altered genomes in other tobacco-related tumors. One of the striking things we observed was a high degree of similarity to other squamous tumors, including lung squamous cell carcinoma. Lessons learned from studying the similarities and differences between tumors, such as these copy number alterations, will be one of the angles researchers will follow to better understand the pathways altered in cancer."

The researchers also identified differences in alterations between tumors infected with the [human papillomavirus](#) (HPV) and those that were negative for the virus. "The current report greatly clarifies an observation that has been made in smaller cohorts of patients with HNSCC that EGFR gene amplification is infrequent in tumors that are infected with HPV but that these same tumors have a high rate of PIK3CA gene mutations," Hayes said.

This finding raises questions about the efficacy of the EGFR inhibitor approved by the U.S. Food and Drug Administration for the treatment of metastatic HNSCC in patients with HPV-positive tumors, according to Hayes. "It also suggests that these patients may benefit from treatment with the inhibitors of PIK3CA that are in development," he added. "However, any treatment conclusions should be based on treatment data, which were not part of the TCGA study."

Provided by American Association for Cancer Research

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