New study introduces the prospect for concurrent antiangiogenic/antitumorigenic therapy

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Today, during the 39th Annual Meeting of the American Association for Dental Research, convening at the Walter E. Washington Convention Center in Washington, DC, lead researcher M. Tong, The Ohio State University, will present a poster of a study titled "Epithelial-to-Endothelial Transition: An Epithelial Phenotypic Modulation Facilitating Oral-Squamous-Cell Carcinoma Progression."

Tong and a team of researchers have reported that oral squamous cell carcinoma (OSCC) cells endogenously produce exceptionally high levels of vascular endothelial growth factor (VEGF). In addition to its proangiogenic function, they observed that VEGF fulfills an autocrine-paracrine role in OSCC by directly promoting OSCC cell proliferation and invasion.

Despite their epithelial origin, cultured OSCC cells express both VEGFR1 and VEGFR2 and serve as both targets and effectors for VEGF. Furthermore, the recognized angiostatic agent, Endostatin, inhibits OSCC cell migration and invasion. Therefore, the researchers of this study hypothesize that at least a subpopulation of OSCC cells have the capability to undergo an "epithelial-to-endotheliod transition" and this process facilitates OSCC progression.

The results of the study imply that OSCC cells undergo a phenotypic transition that endows endotheliod characteristics which augment VEGF

production (with associated autocrine-paracrine functions) and facilitate OSCC cell mobility, enabling angiogenesis, tumor progression and metastases. These OSCC-endothelial cell commonalities introduce the prospect for concurrent antiangiogenic/antitumorigenic therapies.

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