Biomarkers and Genomic Medicine (2014) **6**, 190

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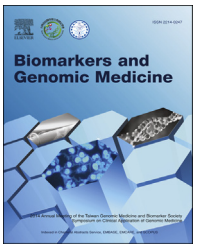

ABSTRACT

Endothelin-1 promotes vascular endothelial growth factor (VEGF) expression through regulating snail of human oral cancer

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Background: Oral cancer is one of the major causes of mortality in humans, and squamous cell carcinoma is the most common type of oral cancer. Endothelin-1 (ET-1) has been implicated in tumor angiogenesis and metastasis. However, the relationship of ET-1 with vascular endothelial growth factor (VEGF) expression in human oral cells is mostly unknown.
Materials and Methods: ET-1-mediated VEGF expression was assessed by qPCR and Western blot analysis. The mechanisms of action of ET-1 in different signaling pathways were studied using Western blotting. Knockdown of proteins was achieved by siRNA transfection.
Results: ET-1 increased VEGF expression through ETAR, integrin-linked kinase (ILK), Akt and sanil signaling cascades. Knockdown of ET-1 decreased VEGF expression and also abolished oral cancer conditional medium-mediated angiogenesis *in vitro*.
Discussion: Taken together, these results indicate that ET-1 occurs through ETAR, ILK and Akt, which in turn activates snail, resulting in the activation of VEGF expression in human oral cancer.

http://dx.doi.org/10.1016/j.bgm.2014.09.010