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Assesment of celecoxib in inhibition of nicotine-induced invasion of human oral squamous cancer cells

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Background and Aims: Cigarette smoke has been documented to be related to the development of cancer. However, the exact mechanism for the carcinogenic action of cigarette smoke is still unknown. Nicotine is recognized to be the major compound in cigarette smoke and has been suggested to play a role in oral cancer via cyclooxygenase /prostaglandin dependent pathway. Besides, extracellular signal-regulated kinases (ERK1/2) belong to the MAPK family and are crucial in the control of cell growth and cell differentiation. The present study was designed to evaluate the action of nicotine in the oral cancer cell, and to further examine whether ERK and COX-2 are responsible for tumor-associated angiogenic vascular endothelial growth factor (VEGF) expression *in vitro*.
Methods: Viability of human oral squamous cancer cells (BHY) was measured using MTT assay. Protein expression was determined by western blot and immunoassay kits.
Results: We found that exposure of BHY cells to nicotine (200 µg/mL for 6 h) resulted in 2.9-fold induction of COX-2 expression as well as 4-fold increase in VEGF level compared to control group. Pre-treatment with celecoxib, inhibited nicotine induced change in VEGF and COX-2 expression.
Conclusions The results suggest that stimulation of COX-2 and VEGF expression can contribute as the important factors in the tumorigenic action of nicotine in oral cancer progression. This effect can be blocked by celecoxib, suggesting interaction of nicotine and COX-2 pathways.

Keywords: Nicotine; Celecoxib; COX-2; Cancer