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Mastermind-like 1 (MAML-1) as a new metastatic marker in esophageal squamous cell carcinoma

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Deregulation of the normal cellular processes that mediate cell proliferation, differentiation and death programs, have been seeing in various tumor cell types [1]. Notch signaling pathway that plays fundamental roles in definition of the cell fate and self-renewal is frequently deregulated in human malignancies, by up or down regulation, where it may act either as an oncogene or as a tumor suppressor depending on the cell context [2,3]. The MAML proteins are transcriptional co-activators of notch signalling that are not only essential for related responses, but also have some regulatory functions in other signaling pathways, including p53 and Wnt/beta-catenin, and therefore have a central role in signaling cross talk of diverse cellular processes such as cell proliferation, differentiation and survival [4].

In current study, the expression of MAML-1 and EGFR genes were analyzed in 44 esophageal squamous cell carcinoma (ESCC) patients with the quantitative (real-time) RT-PCR. Overexpression of these genes was found in 45.5% and 38.6% of ESCC samples respectively. Relative expression of MAML-1 was significantly associated with the lymph node metastasis of tumor cells ($P < 0.05$).

Since MAML-1 is an essential transcription coactivator of notch signaling pathway and downstream related genes expression, we extrapolated that, notch signaling may contribute in tumor development and metastasis in ESCC, however further studies are required. We report for the first time that MAML-1 gene is overexpressed as a metastasis-related gene in ESCC samples. This indicates that MAML-1 may be a possible metastatic marker of ESCC and a possible good therapeutic target in order to block tumor cell metastasis.

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