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SOX2 regualted FOXE1 in head and neck squamous cell carcinoma and correlated with patient outcome

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A high incidence of Head and neck cancer is seen all over the world. Decisions about treatments are usually complex. Although improvements have been achieved in treatments of this disease, the overall survival of patients has limitedly improved. It needs more efforts to investigate in the molecular markers for prognosis since more and more evidences linked tumor etiology with molecular events.
Progressive acquisition of genetic and epigenetic alterations plays an important role in the cause of HNSCC. It was found that 3q26 is the most common amplification locus in this disease. From the microarray and chip-sequence analysis we found out SOX2 as an active element amplified in HNSCC. But information is preliminary and limited. Based on these findings, the first major aim of this master thesis was to explore the expression level of SOX2 in HNSCC tissues and cell lines and tried to find the expression status between SOX2 with the most important prognostic factors such as TNM stage, site and lymph node metastasis, which is not detailed described on published paper. Another prominent and related aim is the identification of the downstream targets of SOX2 proteins. To date, this information in adult human tissue, as well as their potential roles in carcinogenesis in HNSCC, is also limited.
Therefore, at the beginning I constructed tissue microarrays of HNSCC from hospital and used immunohistochemistry method to examine the expression of SOX2 and tried to find the relationships between SOX2 expression and clinic data using statistic software. It was revealed a significant correlation between SOX2 protein levels and tumor site, tumor stage, and lymph node metastasis. In the same time I also test the expression level of SOX2 in HNSCC cell lines which provide groundwork for later genomic modified research. Cooperate with my colleagues, next I obtained an *In silico* analysis report which show that FOXE1 was one of the possible target gene downstream of SOX2 in HNSCC. Then I verified this assumption through establishing gain of function and loss of function experiments, and tried to establish directly relationship between these two factors. The experiments data is coincidence with the in computational analysis that SOX2 regulated FOXE1 in HNSCC. Finally, I inspected the relationship of SOX2 and FOXE1 under hypoxia situation. The assumption is verified again.
In summary, these data revealed SOX2 regulated FOXE1 in human HNSCC and correlated with patient outcome. The information will be important in understanding the oncogenic potential of SOX2 in HNSCC then finally contribute to the treatment and improve the survival of the patients.