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ORIGINAL ARTICLE

c-Jun alterations in oral squamous cell carcinoma

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Summary

Oral carcinogenetic process is based on a variety of genomic imbalances (gross chromosome or specific gene alterations) leading normal oral mucosa to its dysplastic epithelial form and finally to the totally malignant tissue transformation. Suppressor genes' downregulation combined with oncogenes' overactivation are crucial genetic events in pre-malignant and malignant neoplastic epithelia. Additionally, deregulation of specific transcription factors negatively affects the normal expression of genes. Among these, c-Jun (chromosome location: 1p32-p31) is critical forming with c-Fos the activator protein-1 (AP-1) complex early acting as a response transcription factor. In the current special molecular article we explored the role of altered c-Jun gene in oral squamous cell carcinoma (OSCC).

Key words: *c-Jun, oncogene, signaling pathway, oral, carcinoma*

Introduction

Head and Neck Squamous Cell Carcinomas (HNSCC) represent a superfamily of pathological entities with specific etiopathogenetic characteristics [1]. Concerning oral cavity, SCC is the prominent pathological type of malignancy. In fact, oral SCCs (OSCC) demonstrate an aggressive phenotype due to their increased capability to locally metastasize combined with distant lymph node metastases due to specific abnormalities in signaling transduction pathways, such as Notch [2]. Extensive molecular analyses have shown that gross chromosome instability (CI- polysomy/aneuploidy) and specific gene alterations (amplification, deletion, point mutations) or epigenetic (aberrant promoter methylation, microRNAs deregulation) are implicated in the development and progression of solid malignancies, including OSCC [3,4]. OSCC demonstrates increased rates in populations characterized by chronic irritating factors including tobacco and alcohol consumption and also viral mediated deregulation [5,6]. Concerning viral oncogenic activity, persistent Human Papilloma Virus (HPV) infection is responsible for malignant transformation of the corresponding oral mucosa [7]. Among the genes that are involved in OSCC development and progression, overactivated proto-oncogenes involved in signaling transduction pathways play a significant role in modifying nuclear micro-environment [8,9]. Especially, deregulation

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