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Molecular Biology of Oral Cancer and Role of Cyclin D1 in Oral Premalignant Lesions and Oral Squamous Cell Carcinoma – A review of original Study

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Carcinogenesis is a complex, multi-step process in which genetic events within signal transduction pathways governing normal cellular physiology are quantitatively or qualitatively altered (Vogelstein and Kinzler, 1993). The genetic basis of cancer is now well-established. Under normal conditions, these tightly controlled excitatory and inhibitory pathways regulate oral keratinocyte biology. Aberrant expression of the proto-oncogene epidermal growth factor receptor (EGFR)/c-erb1, members of the ras family, as well as cmyc, int-2, HST, Cyclin- D1, and bcl-1, is believed to contribute to oral cancer development. Cyclin D1 is a protein derived from PRAD1 or CCND1 or Bcl-1gene located on chromosome 11q13 and it acts as a positive regulator of the cell cycle in normal cells as well as in neoplasia. Over expression of cyclin D1 may lead to shortening of G1 phase, increased cell proliferation and reduced dependency on growth factors. Our immunohistochemical study results showed that the alteration of cyclin D1 is frequent in oral precancer and oral squamous cell carcinoma. Expression of cyclin D1 was significantly altered from oral epithelial dysplasia to oral squamous cell carcinoma indicating that over expression of cyclin D1 may be an early event in oral cancer development.

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