

January 1994  
VOLUME 40 NO.1

EGYPTIAN DENTAL JOURNAL

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# **DISTRIBUTION OF KERATIN POLYPEPTIDES IN ADULT, JUVENILE AND RAPIDLY PROGRESSIVE PERIODONTITIS**

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*Inflammation of human gingival tissues including the periodontal structures occurs in response to infection in crevicular sulcus caused by microorganisms which first attach to the surface of oral squamous epithelium, penetrate into inner gingival epithelium and finally through the basement membrane of the underlying connective tissue (Murase et al, 1985).*

*Other authors have long held the view that periodontitis is not a homogenous disease entity but rather a group of family related diseases manifesting similar features and different etiologic factors. Immunological mechanism have long been implicated in pathogenesis of chronic periodontitis. Defect of host mechanism may have a relationship in development of rapidly progressive periodontitis (RPP) (Page et al, 1983).*

The influence of inflammation on gingival epithelium has been subjected to several reports which shows a relationship between connective tissue inflammation and epithelial proliferation, rates of cell turnover or surface keratinization (Ouhayoun et al, 1990).

The epithelial lining of the oral cavity is highly cellular. These cells are capable of producing and retaining many secretory products. As an example keratinocytes manufacture an intermediate filament protein from which layers of keratin arise (Stanback, 1987).

Keratin are a family of water insoluble protein which contains 19 cytoskeletal polypeptides, with molecular weight ranging from 40 to 68 KD. A given epithelium express certain subset of cytokeratin polypeptides and thus specific cytokeratin pattern characterize a given epithelium (Frank et al, 1981 & Quinlan et al, 1985). Moll et al (1982) have described a catalogue of the human cytokeratins numbered 1-19, according to their coordinates on separation by two-dimensional gel electrophoresis.

Keratin distribution of a given epithelium appears to depend on the type of epithelium, its stage of development and differentiation, extrinsic factors and pathological conditions (Cooper et al, 1985). Thus keratinized epithelia of attached gingiva and hard palate expressed identical pattern and resembled that pattern found in skin. Although the normally non-keratinized epithelium expressed pattern which is distinctly different from that present in the skin (Clausen et al, 1986).

It is now established that the expression of keratin in given epithelium is not random and model has been proposed which provide a simple framework for their expression in term of certain rules. While different keratin may be present during histogenesis of normal epithelium additional keratin may supplement this basic repertoire in pathological states (Morgan et al, 1987).

Pathological changes, various environmental factors and the subepithelial connective tissue can influence the pattern normally expressed. Examination of changes in keratin

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