Histology of Oral Mucosal Epithelial Cells

Helena Arja*

Department of Oral Diagnostic Sciences, Institute of Dentistry, University of Eastern Finland, Kuopio, Finland

Received: 04-Apr-2022, Manuscript No. OHDM-22-16284; **Editor assigned:** 07-Apr-2022, Pre QC No. OHDM-22-16284 (PQ); **Reviewed:** 21-Apr-2022, QC No. OHDM-22-16284; **Revised:** 28-Apr-2022, Manuscript No. OHDM-22-16284 (R); **Published:** 05-May-2022, DOI: 10.35248/2247-2452.22.21.995.

Description

The oral mucosal epithelium serves as a barrier between the underlying tissues and their surroundings. The surface stratified squamous epithelium and the deeper lamina propria are the two layers that make up the lamina propria. The epithelium of keratinized oral mucosa consists of four layers: stratum basale, stratum spinosum, stratum granulosum, and stratum corneum. The stratum basale is followed by the stratum filamentosum and the stratum distendum in non-keratinised epithelium. Lining mucosa, masticatory mucosa, and specialised mucosa are three separate phenotypes in the oral mucosa. Lining mucosa is a non-keratinizing squamous epithelium that covers movable tissues such as the soft palate, cheeks, lips, alveolar mucosa, vestibular fornix, and mouth floor. It is extensible and loosely connected to neighbouring structures by an elastin rich connective tissue. The inflexible and tough protective surface of the gingiva and hard palate tightly connected by dense connective tissue to the underlying bone is known as masticatory mucosa. Keratinization is present in this epithelium. The dorsum of the tongue has specialised mucosa, which has a keratinized epithelium and comprises lingual papillae and taste buds as specialised structures. The direct attachment to the tooth surface is maintained by the Junctional Epithelium (JE). The external basal lamina anchors the JE's basal cells to the connective tissue, whereas an internal basal lamina produced by the JE anchors the suprabasal cells to the tooth surface.

Although JE has fewer cell connections than the oral gingival epithelium, it does have well-developed gap junctions and several tiny adherens junctions. The JE possesses large intercellular gaps is highly permeable to water-soluble compounds and is the main channel for polymorph nuclear leukocyte transmigration. Although phenotypic stratification does not exist in JE, the outermost cells seem elongated and align with their long axis parallel to the tooth surface. The ability to respond to a variety of foreign potentially hazardous effects is the result of multiple structural and functional protein interactions in the oral epithelial barrier. To maintain its barrier function, squamous epithelia have structural traits such as keratinocyte stratification and cornification, as well as unique cell-to-cell contacts. It is now widely accepted that epithelial cells are metabolically active and capable of responding to external stimuli by producing a variety of cytokines, adhesion molecules, growth factors, chemokines, and matrix-metalloproteases. Gingival tissues give protection against mastication frictional pressures as well as chemical and microbial attack on soft tissues.

Apoptosis and Cellular Phenotype

The purpose of epithelial tissues is to guard the organism against chemical, microbial, and physical threats which is essential for viability. Oral epithelial cells do this role by following a highly regulated differentiation scheme that culminates in the synthesis of structural proteins that maintain the integrity of epithelial tissues and act as a barrier. Various transmembrane proteins with particular structures and activities connect oral epithelial cells. Desmosomes help keratin filaments adhere to the plasma membrane, forming a three dimensional matrix.

Bacterial Influence and Cell-Cell Contacts

It is well known that pathogenic oral bacteria can influence the expression and configuration of cell-cell junctions. When human keratinocytes are stimulated with bacterial components immune-modulatory receptors are upregulated. Periodontal pathogens such as P. gingivalis are able to suppress oral epithelial innate immune responses and evade host immune responses allowing periodontitis to persist. They can also affect epithelial barrier function by altering the expression and distribution of cell-cell interactions such as Tight Junctions (TJs) and Adherens Junctions (AJs). A highly organised biofilm community transitions from symbiosis to dysbiosis in the aetiology of periodontitis, resulting in damaging local inflammatory reactions.

Cellular Receptors

Pattern Recognition Receptors (PRRs) include cell-surface Toll-Like Receptors (TLRs) and cytoplasmatic Nucleotide-binding Oligomerization Domain (NOD)-like Receptors (NLRs). Microbial components that indicate Pathogen-Associated Molecular Patterns (PAMPs) are recognised by PRRs. After activation, a multimeric complex of proteins known as the inflammasome, which is a subset of NLRs, assembles and releases pro-inflammatory cytokines.

Production and Release of Cytokines

Cytokines and bacterial products can cause tissue damage in the host cell. Interleukin (IL)-1, IL-6, IL-8, and Tumour Necrosis Factor (TNF)-α are among the pro-inflammatory cytokines and chemokines that keratinocytes can produce. Pathogenic bacteria like *Porphyromonas gingivalis (P. gingivalis)* and *Aggregatibacter actinomycetemcomitans (A. actinomycetemcomitans)* can cause these cytokines to be produced in a distinct manner.

Bacterial Infection, Immuno-Modulation, and Cancer Cells

Bacterial infection and cancer are known to be linked. On cancer cells, bacterial components have the ability to up-regulate immune-modulatory receptors. Bacterial interactions with tumour cells could aid malignant transformation in an immune-deficient regulation.

Corresponding author: Helena A, Department of Oral Diagnostic Sciences, Institute of Dentistry, University of Eastern Finland, Finland, E-mail: helena_arja@hotmail.com