Volume 07, Issue 02 (March-April 2024), PP 67-72 **www.ijmsdr.org**
ISSN: 2581-902X

International Journal of Medical Science and Dental Research

Unveiling the Guardian of Periodontal Health: Junctional Epithelium in Periodontics

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Abstract: In the intricate world of periodontium, the junctional epithelium stands as a pivotal guardian, delineating the delicate boundary between the tooth surface and the oral environment. Its location makes it a unique tissue where the soft tissue directly attaches to the hard tissue in the oral cavity. This epithelium helps in the barrier defense mechanism and plays a pivotal role in maintaining periodontal health. This review explores the multifaceted roles of the junctional epithelium, shedding light on its structure, function, and significance in periodontal health and disease.

Keywords –*barrier, junctional epithelium, pathogenesis, pocket epithelium.*

I. INTRODUCTION

The junctional epithelium's structural framework comprises specialized cell types organized into distinct layers, fortified by intricate adhesion complexes such as hemidesmosomes and desmosomes. This structural arrangement not only confers mechanical stability but also facilitates dynamic cellular interactions essential for its physiological functions.[1] Functionally, the junctional epithelium serves as a dynamic barrier, regulating the exchange of molecules and cells between the oral environment and the underlying periodontal tissues. Through selective permeability mechanisms mediated by tight junctions and intercellular adhesion molecules, it modulates the passage of ions, nutrients, and immune cells, thereby contributing to tissue homeostasis and defense against microbial challenges. [2]

Immunologically, the junctional epithelium acts as a sentinel interface, orchestrating immune surveillance and response mechanisms in the periodontal microenvironment. Its strategic location enables rapid recruitment of neutrophils and other immune effectors to sites of microbial invasion, thus playing a crucial role in early host defense against periodontal pathogens. Despite its pivotal role, the junctional epithelium is susceptible to perturbations that can compromise its integrity and function. In the context of periodontal diseases, factors such as microbial dysbiosis, host immune response dysregulation, and mechanical trauma can disrupt the epithelial barrier, leading to epithelial attachment loss and subsequent tissue destruction.

II. STRUCTURE AND COMPOSITION:

Situated at the interface of the tooth enamel or cementum and the gingival connective tissue, the junctional epithelium is a specialized stratified squamous epithelium. It comprises a unique cellular arrangement, with hemidesmosomes anchoring its basal cells to the tooth surface, while

desmosomes and tight junctions maintain intercellular connections. [3]

The junctional epithelium is a specialized epithelial tissue that forms the interface between the tooth surface and the gingival connective tissue. Its unique structure and composition play a crucial role in maintaining periodontal health and function.

1. Cellular Composition:

The junctional epithelium is composed of several layers of epithelial cells, each with distinct functions and characteristics:

- Basal Cells: Located at the base of the epithelium, basal cells are cuboidal or columnar in shape and are responsible for anchoring the epithelium to the tooth surface. They contain hemidesmosomes, specialized cell junctions that attach the basal cells to the underlying basement membrane.
- Superficial Cells: The outermost layer of the junctional epithelium is comprised of flattened squamous cells. These cells are constantly renewed through the process of desquamation and are involved in regulating the permeability of the epithelial barrier.[4]

Structural Features:

Basement Membrane: The junctional epithelium is separated from the tooth surface by a specialized basement membrane, which consists of extracellular matrix proteins such as collagen and laminin. This basement membrane provides structural support and serves as a barrier to bacterial invasion.

Hemidesmosomes: Basal cells of the junctional epithelium are anchored to the basement membrane by hemidesmosomes, which are protein complexes that connect the cell cytoskeleton to the extracellular matrix. Hemidesmosomes play a critical role in maintaining the integrity of the epithelial attachment to the tooth surface.

III. Development of junctional epithelium:

As the tooth crown emerges into the oral cavity, the junctional epithelium is formed. Before the tooth emerges fully, the reduced enamel epithelium, comprising reduced ameloblasts and the remaining cells from all other layers of the enamel organ, is responsible for discovering the enamel surface. The stratum intermedium cells of the reduced enamel epithelium and the oral epithelial cells undergo proliferation after the breakdown of the interposed connective tissue these two epithelia eventually merge to form a mass of epithelial cells. As the tips of the cusps or the incisal edge of the crown penetrate the oral mucosa, or shortly before the initial contact between the reduced enamel epithelium and the oral gingival epithelium, a gradual cell transformation process begins. This process starts orally and extends to the cemento-enamel junction within 1 to 2 or 3 to 4 years. The reduced enamel epithelium gradually changes into junctional epithelium, which is a multilayered non-keratinizing squamous epithelium. During this transformation, the reduced ameloblasts transition from short columnar to flattened cells oriented parallel to the enamel surface. Additionally, the cells surrounding the reduced ameloblasts undergo structural changes. Unlike the reduced and transformed ameloblasts, these surrounding cells regain mitotic activity. The transformed ameloblasts move coronally, are shed at the base of the sulcus, and are eventually replaced by the cells adjacent to the reduced/transformed ameloblasts. It has been suggested that the junctional epithelium, initially derived from the reduced enamel epithelium, might be replaced

over time by a junctional epithelium formed by basal cells from the oral gingival epithelium .This theory is supported by observations during the de novo formation of the junctional epithelium after gingivectomy.

IV. Structure of junctional epithelium

Anatomical Considerations:

The junctional epithelium is a part of the marginal 'free' gingiva. It encircles the cervical region of the tooth peripherally and is therefore not visible inside the oral cavity. In the interproximal region, the junctional epithelia near neighboring teeth merge to create the epithelial lining of the interdental col. The uppermost end of the junctional epithelium forms a free surface, which can be found either at the base of the sulcus, at the gingival margin, or in the interdental col area.

The Junctional Epithelial and Interstitial Cells:

The junctional epithelium gradually thins towards its apical end, consisting of 15 to 30 cell layers towards the crown and only 1 to 3 cell layers at its apical extremity. This epithelium is a stratified squamous non-keratinizing type composed of just two layers: a basal layer (stratum basale) and a suprabasal layer (stratum suprabasale). The basal cells are oriented towards the gingival connective tissue, with the basal cells and the adjacent 1 to 2 layers of suprabasal cells being cuboidal to slightly spindle-shaped. The remaining suprabasal cells are flat, aligned parallel to the tooth surface, and exhibit similar characteristics to each other. The innermost suprabasal cells, which face the tooth surface, are also known as DAT cells (Directly Attached to Tooth). These cells create and maintain the 'internal basal lamina' that interfaces with the tooth surface.

The Epithelial Attachment:

The junctional epithelium, positioned between the gingival connective tissue (lamina propria) and the tooth surface, presents a unique cellular arrangement. Basal cells of this epithelium face both the connective tissue and the tooth surface. Separating these basal cells from the gingival connective tissue is a basement membrane, also termed the external basal lamina. Furthermore, an internal basal lamina, forming part of the interfacial matrix between tooth-facing junctional epithelial cells (referred to as DAT cells) and the tooth surface, exists. This basal lamina contributes to securing the junctional epithelium to the tooth surface. At the lower end of the junctional epithelium, the basal lamina seamlessly connects with the basement membrane, ensuring structural coherence and stability for the junctional epithelium's interaction with both the tooth and surrounding gingival tissue.

V. Function and Dynamic Interactions:

Beyond its structural role, the junctional epithelium serves as a dynamic barrier, regulating the passage of molecules and cells between the periodontal tissues and the oral cavity. This selective permeability is crucial for immune surveillance, as the junctional epithelium orchestrates the influx of neutrophils and other immune cells in response to microbial challenges.

VI. Clinical Implications:

Understanding the junctional epithelium's pivotal role is imperative in the context of periodontal diseases. Disruptions in its integrity, such as epithelial attachment loss, facilitate bacterial infiltration and contribute to the pathogenesis of periodontitis. Moreover, its resilience to mechanical trauma and inflammation underscores its significance in maintaining periodontal health.

Regeneration and Therapeutic Perspectives:

In periodontal therapy, efforts to regenerate the junctional epithelium hold promise for enhancing clinical outcomes. Various regenerative techniques, including guided tissue regeneration and growth factor-based therapies, aim to promote the formation of a functional junctional epithelium, thereby restoring periodontal architecture and function.

Expression of various molecules and their functions

The maintenance of normal tissue architecture and function relies on numerous cellular and extracellular molecules. Understanding how tissue integrity is preserved is crucial, especially considering the various unknown factors that may contribute to the onset of periodontal diseases. Key areas of interest include mechanisms that uphold the attachment of epithelium to the tooth surface, the interface between epithelial and connective tissues, and the spatial and interactive relationships among cells within the junctional epithelium itself.

Understanding the structures and molecules responsible for maintaining cell-cell contacts is crucial, especially considering the changes the junctional epithelium undergoes during its transformation into a pocket lining. Cadherins play a vital role in ensuring tight cell contacts, with E-cadherin being particularly significant in maintaining structural integrity as an epithelium-specific cell adhesion molecule (CAM). Studies have shown varying levels of E-cadherin expression in different parts of the junctional epithelium, highlighting its importance in cell cohesion. Additionally, the carcino- embryonic Ag-related cell adhesion molecule 1 (CEACAM1) has been found to play a role in cell- surface adhesion within the junctional epithelium, possibly influencing cell migration and bacterial interactions. Other cell adhesion molecules such as Intercellular adhesion molecule-1 (ICAM-1 or CD54) and lymphocyte function antigen-3 (LFA-3) also contribute to cell interactions and leukocyte migration in inflammatory reactions, with their expression observed in junctional epithelial cells.These molecules, along with cytokines like interleukin-8 (IL-8) and others, play roles in host defense mechanisms against bacterial challenges in the gingival sulcus, highlighting the active role of the junctional epithelium in host-parasite interactions rather than just tooth surface attachment. Analyzing the expression of specific blood-group-specific carbohydrates also provides insights into cellular differentiation levels within the junctional epithelium, potentially indicating cellular proliferative potential in specific cell types.

Recent focus has highlighted the significant role of the epithelial attachment to the tooth surface. However, the dynamic nature of the junctional epithelium suggests a more crucial role for the cells themselves in maintaining tissue integrity.

Junctional epithelium adjacent to oral implants

The junctional epithelium around dental implants originates from oral mucosa epithelial cells, unlike around teeth, which comes from the reduced enamel epithelium. Structural comparisons show similarities between peri-implant epithelium and tooth junctional epithelium, although some differences have been noted. Marker molecules related to defense mechanisms against bacterial challenges are also found in peri-implant epithelium, indicating functional adaptation despite different origins. This adaptability is also seen in the regenerating tooth junctional epithelium after gingivectomy.

Role of junctional epithelium in the initiation of pocket formation

Inflammation in the junctional epithelium typically involves a limited inflammatory infiltrate. The migration of leukocytes, especially polymorphonuclear leukocytes (PMNs), through the junctional epithelium plays a crucial role in the initial host defense at the periphery. These inflammatory cells in the subepithelial lamina propria and within the junctional epithelium are essential components of the defense mechanism against ongoing bacterial challenges, representing normal homeostasis and a critical part of the host's defense system.[6]

The junctional epithelium functions as an 'open system,' allowing cells and substances to migrate from the gingival connective tissue into the sulcus to combat continuous bacterial challenges. However, this also provides an opportunity for bacteria and their products to enter the junctional epithelium. Pocket formation has been linked to bacterial spread under compromised defense conditions. The adherence, invasion, and replication mechanisms of pathogens like Actinobacillus actinomycetemcomitans and Porphyromonas gingivalis (P. gingivalis) have been extensively studied, especially their interactions with epithelial cells and the role of virulence factors like gingipains. Gingipains from P. gingivalis have been found to degrade components of epithelial cell-to-cell junctional complexes, leading to proteolysis of focal contact and adherens junction proteins, altered cell adhesion, morphology changes, impaired motility, and cell apoptosis. They may also disrupt the ICAM-1-dependent adhesion of polymorphonuclear leukocytes (PMNs) to oral epithelial cells, aiding bacterial immune evasion and impacting junctional epithelium integrity. This disruption not only initiates pocket formation but also facilitates bacterial invasion into the subepithelial connective tissue in advanced lesion stages. Similar mechanisms of cell-to-cell contact destruction can affect the structural and functional integrity of the connective tissue, inducing cell death and fibroblast adhesion molecule degradation, particularly induced by the arginine-specific cysteine proteinase Arg-gingipain. Whether these mechanisms operate similarly in vivo remains an important question for future research.

Regeneration and Re-adaptation of junctional epithelium:

Accidental or intentional damage, brushing, flossing, or clinical probing can all cause injury to the junctional epithelium. It makes sense that the junctional epithelium would be highly adapted to withstand mechanical trauma given its location at a sensitive and strategically significant place.

Clinical probing causes the tooth's junctional epithelial cells to be mechanically interrupted. Several studies have sought to determine if and how quickly a new epithelial attachment rebuilds. Five days after the junctional epithelium completely separated from the tooth surface in an experimental research with marmosets, a new and full of attachment that was indistinguishable from that of controls was developed (Taylor and Campbell, 1972).

After clinical probing, it was demonstrated that the epithelial seal surrounding implants would regrow in roughly the same amount of time (Etter et al., 2002). There was no evidence of tissue damage or infection resulting from probing in either study. These two studies suggest that probing the area around teeth and implants does not appear to result in long-term damage to the soft tissue.

The formation of a new junctional epithelium must occur from basal cells of the oral gingival epithelium (Listgarten, 1967; Innes, 1970; Frank et al., 1972; Listgarten and Ellegaard, 1973; Braga and Squier, 1980).

In humans, a new junctional epithelium after gingivectomy may form within 20 days (Listgarten,

1972a,b; Schroeder and Listgarten, 1977). These investigations demonstrate that the oral gingival epithelium's basal cells are able to undergo de novo development or self-renewal, rendering the junctional epithelium a highly dynamic and adaptable tissue.

VII. Conclusion:

In summation, the junctional epithelium stands as a sentinel of periodontal health, intricately woven into the fabric of oral homeostasis. Its structural integrity, dynamic functions, and clinical implications underscore its significance in periodontics. As research continues to unveil its mysteries, the junctional epithelium remains a beacon of hope for advancing periodontal care and preserving oral well-being.

REFERENCES

[1.] NAKAMURA M. HISTOLOGICAL AND IMMUNOLOGICAL CHARACTERISTICS OF THE JUNCTIONAL EPITHELIUM. JPN DENT SCI REV. 2018 MAY;54(2):59-65. DOI: 10.1016/J.JDSR.2017.11.004. EPUB 2017 DEC 7. PMID: 29755616; PMCID: PMC5944073.
[2.] Schroeder H.E., Listgarten M.A. The gingival tissues: the architecture of periodontal protection. Periodontology. 2000;1997(13):91–120.
[3.] Schroeder H.E., Listgarten M.A. The junctional epithelium: from strength to defense. J Dent Res. 2003;82:158–161.
[4.] Ten Cate A.R. The role of epithelium in the development, structure and function of the tissues of tooth support. Oral Dis. 1996;2:55–62.
[5.] Bosshardt DD, Lang NP. The junctional epithelium: from health to disease. J Dent Res. 2005 Jan;84(1):9-20. doi: 10.1177/154405910508400102. PMID: 15615869.
[6.] Arjun Machingal Raveendran., et al. "Exploring the Dynamic Realm of Gingival Epithelium: A Comprehensive Review". EC Dental Science 23.3 (2024): 01-08