

Guardian of Oral Cavity- Mechanism of Gingival Defense

Dr Nanditha Chandran¹, Dr Jilu Jessy Abraham², Dr Arjun MR³, Madhuvandhi⁴, KrishnaPriya K S⁵

^{1,2,3}Associate Professor, Department of Periodontics and Implantology, Mahe Institute of Dental Sciences and Hospital, Mahe, Puducherry, India

^{4,5}Final year student, Department of Periodontics and Implantology, Mahe Institute of Dental Sciences and Hospital, Mahe, Puducherry, India

ABSTRACT

Defense is the ability to fend off an attack. The oral cavity has several defense systems that can fend off different types of injuries. A variety of mechanical, chemical, and antigenic stresses are continuously applied to gingiva; some of these challenges are temporary, while others are ongoing. This study will go over various defense mechanisms the gingiva uses to protect and prepare itself from the harsh environment. Defense is the ability to withstand the attack; the mouth cavity has many defense systems that protect it from injuries. A vast range of mechanical, chemical, and antigenic obstacles are continuously faced by gingiva. This study will go over the several defense mechanisms how gingiva will protect from the harsh environment.

ARTICLE DETAILS

Published On:
08 October 2024

Available on:
<https://ijmscr.org/>

INTRODUCTION

Gingival Crevicular Fluid is an exudate derived by inflammation by periodontal. The gingival tissue is subjected to bacterial and mechanical aggressions. The saliva, epithelial surface and initial stage of inflammatory response provide resistance to such action. Therefore, it has been proposed that a crucial role for the interaction between periodontopathogenic bacteria and epithelial cells in the early stages of periodontal disease. As a medication that prevents stomach ulcers, isogladine maleate operates on the gastric mucosal epithelium. Additionally, it has been demonstrated that amphotericin B modifies the host's immunological responses to fungus infections.²

GINGIVAL CERVICULAR FLUID:

- It is an exudate that is inflammatory and comes from the periodontal tissues.² It is made up of serum and locally produced substances such as inflammatory mediators, and antibodies against dental plaque bacteria.¹
- Its constituents are derived from a number of sources, including serum, connective tissue, and epithelium through which GCF plays a special part in maintaining the structure of junctional epithelium and the antimicrobial defense of periodontium.¹ It has been established by numerous researchers that GCF is a complex mixture of materials originating

from oral bacteria, leukocytes, and periodontium structural cells.³

FORMATION

There are 2 theories that suggest the formation of GCF.

- Theory-From the work of Alfano (1974) and from the hypothesis postulated by Pashley (1976) the initial fluid produced could simply represent interstitial fluid which appears in the crevice as a result of an osmotic gradient.³
- The macromolecular by products of the bacteria in dental plaque create this osmotic gradient by permeating the gingival crevicular epithelium and the basement membrane, so blocking additional penetration. The flow of fluid capillaries into the tissues (capillary filtrate) and the lymphatic system's evacuation of the fluid (lymphatic uptake) control GCF production, according to the Pashley (1976) model.²

FUNCTION:

- The gingival margin is accessible to GCF, inflammatory cells, and elements of the immune system's host defence because the junctional epithelium is firmly linked to the tooth, creating an epithelium barrier against plaque bacteria.²
- Additionally, its quick turnover helps maintain the balance between the host and the parasite and speeds up the healing of injured tissues.¹

Guardian of Oral Cavity- Mechanism of Gingival Defense

- GCF is an exudate varying composition found in the sulcus or periodontal pocket between the tooth and marginal gingiva. The amount of GCF in a healthy sulcus is extremely little. The GCF flow rises and takes on characteristics of an inflammatory exudates during inflammation.³

METHOD OF COLLECTION

GCF is obtained from the sulcus. The methods includes

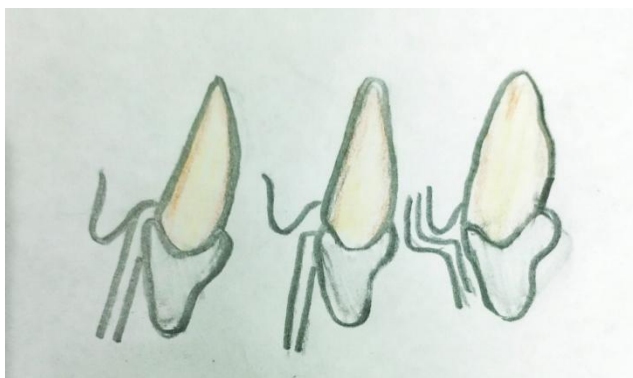
- Absorbing paper strips
- The placement of twisted threads

And the technique involves

- Micropipettes
- Crevicular washing

Absorbing paper strips:

- Placed in both Intrasulcular and extrasulcular method
- Place the filter paper strip in sulcus or in pocket



- Brill technique used to insert into the pocket until resistance is created.
- This method produces some degree of irritation to sulcular epithelium and itself trigger the flow of fluid
- To reduce the irritation, the filter paper is placed at the entrance over the pocket
- Fluid seeps out is picked up by the strip and the sulcular epithelium is not in contact with the paper

Pre-weighed twisted thread:

- Placed in gingival crevice around the tooth and amount is estimated by weighing the sample thread

Micropipettes:

- Used for collection of fluid by capillarity
- Capillary tubes of internal diameter is inserted into the gingival crevice
- Isolation and drying should be done
- GCF is collected by Capillary action since the internal diameter is known the volume can be determined by measuring the distance which GCF is migrated

- Provides undiluted sample of native GCF volume
- Accurate amount is provided in a short period
- Complication is difficult to remove the sample from the tube⁵

Crevicular washing:

- Used to study GCF from the normal gingiva clinically
- One method involves the use of appliance Consist of hard acrylic plate covers maxilla with soft border grooves follows the marginal gingiva and this is connected to four collection tube
- Washing obtained by rinsing the crevicular areas from one to other by the use of peristaltic pump
- Modifications of this method involves the two injection needles that has been fitted one within the other

- During sampling the ejection needle at the bottom and collecting needle at marginal gingiva
- Collection needle is drained into sample tube by continuous suction²

COMPOSITION:

- Microbial plaque:
 - Endotoxins (LPS)
 - Enzymes (acid phosphatase, alkaline phosphatase)
 - Metabolic end products (trypsin like enzyme)
- Host cells:
 - Lysozyme
 - Lactoferrin
 - Leucocytic enzyme
 - Myeloperoxidase
- Damaged host tissues:
 - Collagens
 - Proteoglycans
 - Materix proteins
- Host factors (Immune)
 - Immunoglobins (IgG, IgA, IgM)

Guardian of Oral Cavity- Mechanism of Gingival Defense

- Complement factors
- Cytokines
- Eicosanoids (IL-1)

SALIVA:

- Its presents in clinically normal sulci can be explained because gingiva that appears clinically normal invariably exhibits inflammation when examined microscopically.⁴
- When inflammation is present, the amount of GCF increases and is occasionally correlated with the degree of inflammation. Mastication of coarse food increases the generation of GCF rather than trauma from occlusion by eating, ovulation, smoking, brushing your teeth and massaging your gingival, and hormonal contraceptives. Periodontal treatment and circadian periodicity are two further factors that affect the quantity of GCF.⁷

COMPONENTS OF SALIVA:

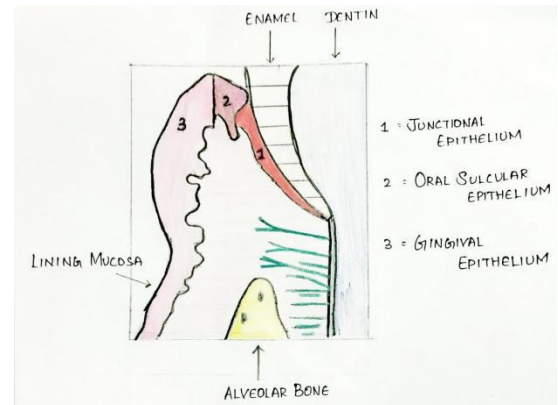
- Enzymes
- Antibodies
- Histamins
- Peroxides
- Cystatins
- Mucins
- Lysozyme
- Lactoferrin⁵

FUNCTION OF SALIVA:

- The major way that saliva affects plaque is by mechanically cleaning the oral surfaces that are exposed, preventing bacterial acid production, and managing microbiological activity.⁵
- Defense against heat, mechanical force, and chemical irritation.
- Buffering effect and stops harmful microorganisms from colonizing.
- Prevent sudden changes in pH.
- Supplies minerals, including calcium and phosphorus, to teeth
- Cleaning effect of washing away food debris.
- Makes swallowing food easier.
- Solvent effect: dissolves food and allows the tongue to taste of food.
- Lubricating effect that protects mucous membranes.⁶

EPITHELIAL TISSUE:

The epithelial layer that makes up gingiva is made up of stratified squamous epithelium, which acts as a physical barrier against infection and actively participates in the hosts natural defensive mechanisms.⁴



IN WHAT WAY DOES EPITHELIUM RESIST PATHOGEN ENTRY?

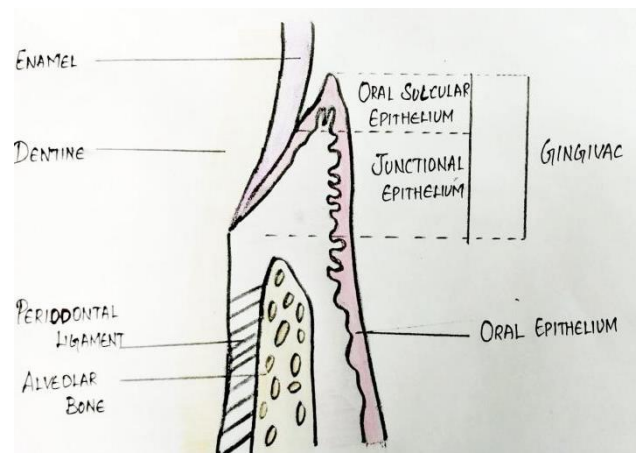
- It functions as a mechanical barrier to prevent entry.²

ORAL EPITHELIUM

- Turns to face the mouth.
- Parakeratinized and densely packed cells.
- Protecting the deeper structures while permitting a selected exchange with the oral environment is its function.⁶

SULCULAR EPITHELIUM:

- Turns to face the tooth without making contact with its surface.⁸
- Lack of keratinization and the existence of intercellular gaps²



Function:

- It creates an epithelial barrier against the plaque microorganisms by being firmly affixed to the tooth surface.⁵
- It permits gingival fluid, inflammatory cells, and immune host defense components to reach the gingival margin.⁷

Guardian of Oral Cavity- Mechanism of Gingival Defense

- Chemical barrier
- Biological barrier

MECHANICAL BARRIER:

- Gingival epithelium is classified according to its morphological and function traits.⁴
 - The oral epithelium
 - Sulcular epithelium
 - Junctional epithelium

DENTO-GINGIVAL JUNCTION:

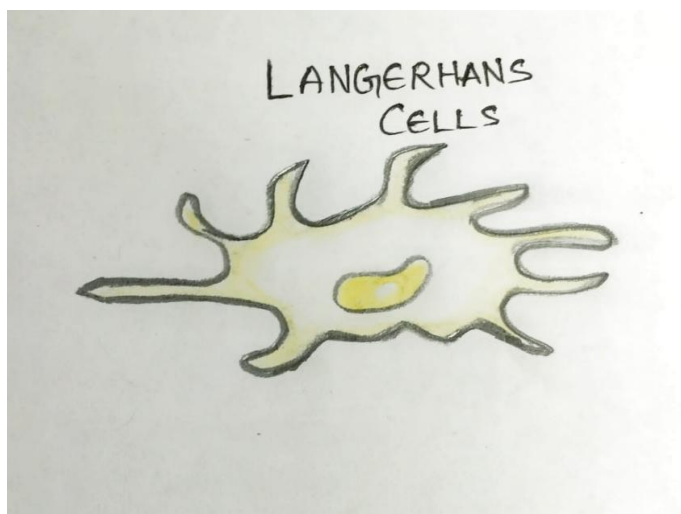
1. Junctional epithelium
 2. Sulcular epithelium
 3. Oral epithelium
1. Epithelial rests of Malassez²

JUNCTIONAL EPITHELIUM :

- Affixed to the tooth
- Obstacle
- Quick turnover
- Defense against microbes
- GCF movement

It display a quick rate of host parasite equilibrium turnover as well as quick tissue healing.¹⁰

The endocytic potential of junctional epithelium cells is comparable to that of neutrophils and macrophages³



LANGHERAN CELLS:

Dendritic cells, as described by Paul Langheran in 1868.³

- They are in charge of correspondence with the immunological responses.²
- They serve as cells that deliver antigens for T cells.²
- They identify, seize, and display foreign antigens them to T lymphocytes.⁵
- Inflamed gingival tissue contains five times more of them, in contrast to gingiva in good health.⁷

ROLE OF KERATINOCYTE:

These are the main cell type of various stratified squamous epithelium. The gingival epithelium primary role (defense and obstacle against oral environment) is accomplished by the growth and diversification of the keratinocytes. ⁵

PROLIFERATION:

Keratinocytes proliferate through the process of mitosis. Less commonly in the suprabasal layer and in the basal layer, in which a tiny percentage of cells persist as a proliferative section as more people start to rise to the surface.³

DIFFERENTIATION:

- It entails monitoring morphologic and biochemical processes. Increased frequency of tonofilaments and progressive flattening of the cell with its long axis parallel to the epithelial surface. ²
- Intercellular connections linked to the keratohyaline granule formation.
- Absence of the nucleus³
- Mitotic division causes cells to lose their capacity to divide.²
- The cytoplasmic organelles that are in charge of protein synthesis and energy generation are lost by cells.⁹
- Cells undergo intracellular keratinization, which causes them to finally degenerate into a cornified layer.⁶
- Desmosomes and gap junctions, two cell-cell attachment processes, eventually break down, causing cells to slough off the epithelial surface and into the oral cavity.⁷

CONNECTIVE TISSUE:

-The linked gingiva pliability and toughness allow it to tolerate pressures and friction brought on by mastication. The fiber apparatus regulates how teeth are positioned inside the dental arch. ⁵Gingival tissues biostability. Shields the cellular defences that are situated at the mouth to gingival contact.⁸

-Preservation of this complex, fibrous connective tissue rate of turnover

-As a result, the fiber apparatus post inflammatory repair is finished in 40 to 60 days.³

-The alveolar bone and root surface are shielded from the external oral environment by gingival connective tissue.⁶

CELLS OF GINGIVA INVOLVED IN DEFENSE MECHANISM:

- Immune cells: neutrophils, monocytes, macrocytes, lymphocytes, basophils, eosinophils, mast cells, plasma cells.
- Non immune cells: keratinocytes, Langherans cells, fibroblast

MACROPHAGES:

- Originating from monocytes
- The surface of cells are tangled in many folds and had projections that resemble fingers.⁴
- These folds actively engage in phagocytosis by engulfing targets for phagocytosis.⁵

Guardian of Oral Cavity- Mechanism of Gingival Defense

- Upon coming into contact with foreign body, macrophages combine to produce giant cells are referred to as foreign body giant cell.⁶
- The fixed macrophages in response to antigen stimulation or inflammation.⁷
- Defences, cleanup and immune responses are examples of function.⁵

MAST CELL:

- Spherically shaped connective cells that are ovoid and engaged in inflammatory responses.
- The production of antibodies by allergies and hypersensitivity reaction is dependent on plasma cells, which in turn generate antibodies that attach to their surface.
- Mast cell release substances such as histamine, eosinophils chemotactic factor of anaphylaxis, heparin, and slow reacting substance of anaphylaxis.⁷

LYMPHOCYTES:

- Lymphocytes are the smallest free cells in connective tissue.⁷
- They move through blood and connective tissue, passing via their secretory product and their membrane, which has capacity to bind and recognize foreign molecules, quantity of tissues inflammation and restoration.⁸
- They resemble cells with a thin cytoplasmic ring encircling a highly pigmented heterochromic nucleus.⁹
- T lymphocytes are involved in the generation of antibodies and have varying lifespan.⁹

CONCLUSION

The oral cavity is well equipped to counterattack any adverse condition that may affect gingiva, from the superficial epithelial layer to innermost connective tissue, there is a line of defences that acts in harmony with oral structures to maintain homeostasis.¹⁰

REFERENCES

- I. Subbarao KC, Nattuthurai GS, Sundararajan SK, Sujith I, Joseph J, Syedshah YP. Gingival Crevicular Fluid: An Overview. *J Pharm Bioallied Sci.* 2019 May;11(Suppl 2):S135-S139. doi: 10.4103/JPBS.JPBS_56_19. PMID: 31198325; PMCID: PMC6555362.
- II. Fujita T, Yoshimoto T, Kajiya M, Ouhara K, Matsuda S, Takemura T, Akutagawa K, Takeda K, Mizuno N, Kurihara H. Regulation of defensive function on gingival epithelial cells can prevent periodontal disease. *Jpn Dent Sci Rev.* 2018 May;54(2):66-75. doi: 10.1016/j.jdsr.2017.11.003. Epub 2017 Dec 15. PMID: 29755617; PMCID: PMC5944110.
- III. Hatakeyama S, Yaegashi T, Oikawa Y, Fujiwara H, Mikami T, Takeda Y, Satoh M. Expression pattern of adhesion molecules in junctional epithelium differs from that in other gingival epithelia. *J Periodontol Res.* 2006 Aug;41(4):322-8. doi: 10.1111/j.1600-0765.2006.00875.x. PMID: 16827727.
- IV. Pöllänen MT, Salonen JI, Uitto VJ. Structure and function of the tooth-epithelial interface in health and disease. *Periodontol 2000.* 2003;31:12-31. doi: 10.1034/j.1600-0757.2003.03102.x. PMID: 12656993.
- V. 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.
- VI. Schroeder HE, Listgarten MA. The junctional epithelium: from strength to defense. *J Dent Res.* 2003 Mar;82(3):158-61. doi: 10.1177/154405910308200302. PMID: 12598541.
- VII. The pathogenesis of periodontal diseases. *J Periodontol.* 1999 Apr;70(4):457-70. doi: 10.1902/jop.1999.70.4.457. PMID: 10328661.
- VIII. Listgarten MA. Structure of the microbial flora associated with periodontal health and disease in man. A light and electron microscopic study. *J Periodontol.* 1976 Jan;47(1):1-18. doi: 10.1902/jop.1976.47.1.1. PMID: 1063849.
- IX. Cimasoni G. The crevicular fluid. *Monogr Oral Sci.* 1974;3(0):1-122. PMID: 4600951.
- X. Nicu EA, Loos BG. Polymorphonuclear neutrophils in periodontitis and their possible modulation as a therapeutic approach. *Periodontol 2000.* 2016 Jun;71(1):140-63. doi: 10.1111/prd.12113. PMID: 27045435.