Volume 4, Issue 6, June – 2026 **ISSN (E):** 2938-379X

BIOPHYSICAL AND BIOCHEMICAL BASIS OF APHTHAE FORMATION IN THE ORAL MUCOSA

Nastinov Sadriddin Tojiddin o‘g‘li
Teacher, Namangan State University
Email: sadriddin_1995_08_29@mail.ru

Abstract
This article analyzes the biophysical and biochemical mechanisms of aphthae formation in the oral mucosa. The structural characteristics of epithelial cells, microbial colonization, immunological response reactions, and the role of inflammatory mediators are scientifically discussed. In addition, the possibilities of mathematical modeling of cellular dynamics and the pathogenesis of the aphthous process are considered.

Keywords: Aphthous stomatitis, epithelium, biophysics, biochemistry, inflammation, cytokines, Candida albicans, mathematical model, immune response.

Introduction

OG‘IZ BO‘SHLIG‘I SHILLIQ QAVATIDA AFT HOSIL BO‘LISHINING BIOFIZIK VA BIOKIMYOVIY ASOSLARI

Nastinov Sadriddin Tojiddin o‘g‘li
Namangan davlat universiteti o‘qituvchisi
Email: sadriddin_1995_08_29@mail.ru

Annotatsiya
Mazkur maqolada og‘iz bo‘shlig‘i shilliq qavatida AFT hosil bo‘lishining biofizik va biokimyoviy mexanizmlari tahlil qilingan. Epitelial hujayralarning strukturaviy xususiyatlari, mikroorganizmlar kolonizatsiyasi, immunologik javob reaksiyalari hamda yallig‘lanish mediatorlarining roli ilmiy jihatdan yoritilgan. Shuningdek, aftoz jarayonning hujayraviy dinamikasi va patogenezini matematik modellashtirish imkoniyatlari ko‘rib chiqilgan.

Kalit so‘zlar: aftoz stomatit, epiteliy, biofizika, biokimyo, yallig‘lanish, sitokinlar, Candida albicans, matematik model, immun javob.

БИОФИЗИЧЕСКИЕ И БИОХИМИЧЕСКИЕ ОСНОВЫ ОБРАЗОВАНИЯ АФТ В СЛИЗИСТОЙ ОБОЛОЧКЕ ПОЛОСТИ РТА

Настинов Садриддин Тоджиддин ўғли
Преподаватель, Наманганский государственный университет
Email: sadriddin_1995_08_29@mail.ru

Аннотация
В данной статье проанализированы биофизические и биохимические механизмы образования афт в слизистой оболочке полости рта. С научной точки зрения освещены структурные особенности эпителиальных клеток, колонизация микроорганизмов, иммунологические реакции и роль медиаторов воспаления. Кроме того, рассмотрены возможности математического моделирования клеточной динамики и патогенеза афтозного процесса.

Ключевые слова: афтозный стоматит, эпителий, биофизика, биохимия, воспаление, цитокины, Candida albicans, математическая модель, иммунный ответ.

Introduction

The oral mucosa represents one of the primary biological defense barriers of the human body that remains in continuous contact with the external environment. Due to constant exposure to mechanical irritation, chemical agents, and microbial factors, this tissue is highly susceptible to various pathological alterations. Among the inflammatory disorders affecting the oral cavity, aphthous lesions are considered one of the most frequently encountered conditions and are characterized by epithelial damage, immune dysregulation, and disturbances in microbial homeostasis.

The development of aphthous ulcers is associated with a complex multifactorial biological mechanism involving epithelial membrane biophysics, cytokine-mediated inflammatory responses, oxidative stress processes, and microbial activity. These interconnected mechanisms contribute to tissue destruction and impairment of the protective functions of the oral epithelium.

Biophysical Characteristics of the Epithelial System

The oral epithelial layer consists predominantly of stratified squamous epithelium whose major physiological role is to provide mechanical and immunological protection. Structural integrity of the epithelial tissue is maintained through intercellular junctional complexes, including desmosomes and tight junctions, which ensure cellular cohesion and barrier stability.

The biophysical condition of the mucosal membrane is determined by several important parameters:

- membrane permeability;
- ionic exchange processes;
- osmotic pressure regulation;
- cellular diffusion mechanisms;
- bioelectrical potential.

A salivary film covering the epithelial surface creates a hydrodynamic protective environment that limits microbial adhesion and colonization. In addition, saliva contains several protective biomolecules such as lysozyme, lactoferrin, and secretory immunoglobulin A (sIgA), all of which play an essential role in maintaining local immune defense and mucosal stability.

Literature Review and Methodology

This scientific study provides a detailed analysis of the etiology, pathogenesis, and clinical manifestations of recurrent aphthous stomatitis (RAS). The authors scientifically demonstrated that immune dysfunction, hereditary predisposition, psychological stress, and microbial activity play essential roles in the development of aphthous lesions. The investigation further revealed that inflammatory mediators and activated T-lymphocytes contribute to epithelial cell destruction and tissue damage. Therefore, this article represents an important scientific source for understanding the immunological and biochemical mechanisms underlying aphth formation [1].

In the work authored by Crispian Scully, diseases affecting the oral mucosa, including aphthous stomatitis and inflammatory oral disorders, are discussed from both clinical and pathogenetic perspectives. The author thoroughly examined epithelial cell injury, immune response reactions, and cytokine activation involved in the development of aphthous lesions. This book is considered a fundamental reference for the diagnosis of oral pathologies and for explaining their biological mechanisms [2].

This textbook is regarded as one of the principal scientific resources in oral microbiology and the biology of *Candida albicans*. The study highlighted the ability of *Candida* species to form biofilms, adhere to epithelial cells, and produce proteolytic enzymes, which are considered important factors in oral mucosal inflammation. This source is particularly valuable for explaining the microbiological and biochemical basis of aphth formation [3].

The present scientific article provides an extensive analysis of the pathogenic properties of *Candida albicans*, including its capacity for biofilm formation and epithelial invasion. The authors demonstrated that *Candida* species are capable of producing proteolytic enzymes and intensifying inflammatory responses in oral tissues. This investigation is highly important for clarifying the role of microbial colonization in the development of aphthous lesions [5].

This textbook analyzes opportunistic microorganisms, especially *Candida albicans*, and their biological interactions with the host organism. The author comprehensively described the toxic effects, invasive characteristics, and immune-related reactions associated with these microorganisms. The source is considered significant for studying oral inflammatory conditions and microbiological processes [7].

Microorganisms and Aphth Formation

Disruption of the oral microbiota is regarded as one of the major factors contributing to the development of aphthous lesions. In particular, microorganisms such as *Candida albicans*, *Streptococcus sanguinis*, and *Fusobacterium nucleatum* possess strong adhesion abilities to epithelial cells, facilitating colonization and inflammatory tissue damage.

Candida albicans contributes to aphth formation through several pathogenic mechanisms associated with biofilm development:

- adhesion to the epithelial surface;
- disruption of cellular membrane integrity;
- secretion of proteolytic enzymes;
- activation of inflammatory mediators.

Toxins produced by microorganisms intensify oxidative stress within epithelial cells and accelerate lipid peroxidation processes, leading to cellular damage and impairment of mucosal barrier function.

Immunological and Biochemical Mechanisms

T-lymphocytes, macrophages, and neutrophils play essential roles in the development of aphthous lesions. During the inflammatory process, several cytokines actively participate, including:

- Interleukin-1β (IL-1β);
- Interleukin-6 (IL-6);
- Tumor Necrosis Factor-α (TNF-α);
- Interferon-γ (IFN-γ).

These inflammatory mediators contribute to:

- increased capillary permeability;
- enhancement of cellular necrosis;
- delayed epithelial regeneration [6].

From a biochemical perspective, the aphthous process is associated with impaired ATP synthesis, mitochondrial dysfunction, and elevated concentrations of free radicals. Such alterations disrupt normal cellular metabolism and intensify tissue injury within the oral mucosa.

Mathematical Interpretation of Aphth Formation

Mathematical modeling of biological processes makes it possible to predict the dynamics of aphth development and progression. Interactions between epithelial cells and pathogenic agents can be described using differential equations that characterize changes within the biological system over time.

Dynamics of *Candida* Colony Growth

The dynamics of a *Candida* colony represent a mathematical model describing the temporal growth, reduction, and interaction of fungal colonies with epithelial tissues in biological systems.

The principal differential equation can be expressed as follows:

$$\frac{dC}{dt} = rC\left(1 - \frac{C}{K}\right) - \beta CE$$

Where:

- C(t) - quantity (population size) of the Candida albicans colony over time;
- t - time variable.;
- r - growth rate of the fungal colony.;
- K - maximum carrying capacity of the environment;
- E(t) - number of epithelial cells over time.;
- β - interaction coefficient between the fungal colony and epithelial cells.

Components of the Mathematical Model:

Natural Logistic Growth of Candida:

$$rC\left(1 - \frac{C}{K}\right)$$

This component describes the proliferation of the colony under conditions of limited resources.

Adhesion to and Damage of the Epithelium:

−βCE

This component represents the adhesion of Candida to the epithelium and its biological effects.

If the immune response is also incorporated into the model:

$$\frac{dC}{dt}=rC\left(1-\frac{C}{K}\right)-\beta CE-\mu IC$$

Where:

- I(t) - concentration of immune cells over time;
- μ - suppression coefficient of the immune system.
- This model is applied for the mathematical analysis of:
- aphth formation;
- candidamycosis;
- epithelial degradation;
- inflammatory dynamics.

Results and Discussion

The analysis demonstrates that immunological imbalance, microbial colonization, and biophysical disturbances collectively contribute to the development of aphthous lesions. An increase in the *Candida albicans* colony accelerates epithelial destruction and enhances the secretion of inflammatory mediators.

Mathematical modeling enables:

- evaluation of inflammatory intensity;
- prediction of epithelial regeneration rates;
- assessment of therapeutic effectiveness.

Conclusion

The formation of aphthous lesions in the oral cavity is a complex biophysical and biochemical process based on multifactorial interactions among epithelial cells, microorganisms, and the immune system. Mathematical modeling of this process provides an opportunity for a deeper analysis of pathological dynamics and contributes to the optimization of diagnostic and therapeutic strategies.

References

1. Akintoye SO, Greenberg MS. Recurrent aphthous stomatitis. Dent Clin North Am. 2014 Apr;58(2):281-97. doi: 10.1016/j.cden.2013.12.002. Epub 2014 Jan 21. PMID: 24655523; PMCID: PMC3964366.
2. Scully C. Oral and Maxillofacial Medicine. — London: Elsevier, 2020.
3. Samaranayake L. Essential Microbiology for Dentistry. — Churchill Livingstone, 2018.
4. Greenberg M., Glick M. Burket's Oral Medicine. — PMPH-USA, 2021.
5. Williams D., Lewis M. Pathogenesis and treatment of oral candidosis // Journal of Oral Microbiology. — 2019.
6. Abbas A., Lichtman A. Cellular and Molecular Immunology. — Elsevier, 2021.
7. Murray P. Medical Microbiology. — Philadelphia: Mosby, 2020.

8. Guyton A., Hall J. Textbook of Medical Physiology. — Elsevier, 2021.
9. Lodish H. Molecular Cell Biology. — W.H. Freeman, 2021.
10. Alimova D.A. Og‘iz bo‘shlig‘i kasalliklari patogenezi. — Toshkent, 2022.
11. Xamidov X.X. Biologik tizimlarni matematik modellashtirish asoslari. — Toshkent, 2023.

Web of Teachers: Inderscience Research
webofjournals.com/index.php/1