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DENTAL CARIES OF HARD TISSUES AS AN INFLAMMATORY PROCESS WITHIN THE SYSTEM OF THE LAWS OF THERMODYNAMICS

Introduction. Dental caries is the most prevalent pathological process affecting the teeth. Its development is associated with the complex interaction of microorganisms, dental tissues, ionic exchange, and local physicochemical processes. Considering caries as an inflammatory process makes it possible to complement traditional biological concepts with the principles of thermodynamics, particularly the regularities of energy transformation and exchange, and changes in entropy.

Aim. To describe the etiology of dental caries of the hard tissues as an inflammatory process within the system of the laws of thermodynamics and to determine the relationship between inflammation, phagocytosis, anisotropy, ion exchange, and energetic processes.

Materials and methods. A literature analysis was conducted concerning the pathogenesis of dental caries, the properties of dental hard tissues, microbial activity, demineralization and remineralization processes, as well as the physicochemical and thermodynamic regularities accompanying the pathological process. The principles of the zeroth, first, second, and third laws of thermodynamics were theoretically analyzed in relation to processes occurring in dental tissues.

Results and discussion. It was established that the development of the carious process is accompanied by a complex interaction of inflammatory reactions, microbial activity, changes in ionic composition, and disturbances in the energy balance of dental hard tissues. The roles of phagocytosis, anisotropy of enamel and dentin, ion transport, membrane potential, and entropy changes in the development of the pathological process were analyzed. The thermodynamic approach may help explain the direction and nature of physicochemical transformations associated with the demineralization of dental hard tissues.

Conclusions. The application of the laws of thermodynamics broadens understanding of the mechanisms underlying caries development and of the relationships among structural changes in dental tissues, ion exchange, and energetic processes. Further research may focus on developing the thermodynamic basis of caries pathogenesis and applying it to improve approaches to prevention and treatment.

Key words: dental caries, hard dental tissues, inflammation, phagocytosis, anisotropy, ion exchange, thermodynamics, entropy.

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КАРІЄС ТВЕРДИХ ТКАНИН ЗУБА ЯК ЗАПАЛЬНИЙ ПРОЦЕС У СИСТЕМІ ЗАКОНІВ ТЕРМОДИНАМІКИ

Вступ. Карієс зубів – найбільш поширений патологічний процес, розвиток якого пов'язаний із взаємодією мікроорганізмів, тканин зуба, іонного обміну та локальних фізико-хімічних процесів. Розгляд карієсу як запального процесу дає змогу доповнити традиційні біологічні уявлення положеннями термодинаміки, зокрема закономірностями перетворення та обміну енергії й зміни ентропії.

Мета дослідження. Описати етіологію карієсу твердих тканин зуба як запального процесу в межах системи законів термодинаміки та визначити взаємозв'язок між запаленням, фагоцитозом, анізотропією, іонним обміном і енергетичними процесами.

Матеріали та методи. Проведено аналіз наукової літератури щодо патогенезу карієсу, властивостей твердих тканин зуба, мікробної активності, процесів демінералізації та ремінералізації, а також фізико-хімічних і термодинамічних закономірностей, що супроводжують патологічний процес. Застосовано теоретичний аналіз положень нульового, першого, другого та третього законів термодинаміки щодо процесів, що відбуваються в тканинах зуба.

Результати та їх обговорення. Встановлено, що розвиток каріозного процесу супроводжується комплексною взаємодією запальних реакцій, мікробного впливу, зміни іонного складу та порушення енергетичної рівноваги твердих тканин зуба. Проаналізовано роль фагоцитозу, анізотропії емалі та дентину, іонного транспорту, мембранного потенціалу й ентропійних змін у формуванні патологічного процесу. Показано, що термодинамічний підхід може бути використаний для пояснення напрямку та характеру фізико-хімічних перетворень, пов'язаних із демінералізацією твердих тканин зуба.

Висновки. Застосування законів термодинаміки дозволяє розширити розуміння механізмів розвитку карієсу та взаємозв'язку між змінами структури зубних тканин, іонним обміном і енергетичними процесами.

Ключові слова: карієс, тверді тканини зуба, запалення, фагоцитоз, анізотропія, іонний обмін, термодинаміка, ентропія.

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Introduction. The word “caries” comes from the Latin caries – “rot, decay.” Historically, this term was initially used for inflammatory diseases of the bone marrow (osteomyelitis). Today, the term is used to refer to dental diseases. Dental caries (K02 according to ICD-10) is an infectious pathological process that occurs after tooth eruption and is characterized by demineralization and softening of the tooth’s hard tissues, followed by the formation of a cavity [1–2].

The aim of the study is to describe the etiology of caries in the hard tissues of the tooth as an inflammation within the laws of thermodynamics [3–4].

Methodology and research methods. The study uses a narrative conceptual approach rather than a clinical or laboratory experiment. The source material was critically organized around four interacting domains: cariogenic biofilm activity, mineral exchange in hard dental tissues, inflammatory responses of the dentin–pulp complex, and thermodynamic interpretation.

Recent literature was used to anchor established biological mechanisms, while the thermodynamic component was treated as a theoretical framework. If the proposed model is subsequently evaluated with quantitative time-series or nonlinear data, surrogate-data testing may be considered as a methodological option [5].

The primary cause of dental caries is the activity of bacteria in the oral cavity, primarily *Streptococcus mutans* and *Streptococcus sanguis*, which decompose carbohydrates into acids and destroy tooth enamel. These bacteria often accumulate in dental plaque, forming a biofilm that can be difficult to remove during cleaning [1, 6].

If caries is considered as an inflammatory reaction, a corresponding response of odontoblasts to the endothermic reaction of carbohydrate breakdown in acid can be assumed. The entropy factor is also considered because the tooth is treated as part of a thermodynamic system [7].

Dentin inflammation is deep caries, in which bacteria destroy dentin and penetrate toward the dental pulp, causing pain and sensitivity. It may also lead to pulpitis (inflammation of the nerve) and subsequently to periodontitis (inflammation of tissues around the tooth root). Symptoms include acute pain in response to cold, sweet or sour stimuli, as well as pain when food enters the carious cavity. Treatment involves removal of infected tissues, filling, and, when necessary, pulp therapy [4, 8].

Inflammation of the dental pulp is called pulpitis. This is a condition in which the internal tissues of the tooth containing nerves and blood vessels become infected and inflamed. The primary cause of pulpitis is untreated caries, but it may also be caused by dental trauma, improper dental treatment or exposure to extreme temperatures [4, 8].

Materials and methods. In the proposed model, enamel damage is associated with phagocytosis as a response, the action of toxins and changes in the anisotropy index, as well as the ability to withstand load, including pressure in Pa [4, 8].

A disruption of harmony between S-shaped and spiral-shaped structures extending radially toward the enamel

surface may lead to changes in enamel anisotropy. Such anisotropic changes may create conditions for bacterial attack and targeted localization of bacteria in tooth fissures [2, 9].

Enamel prisms have an S-shaped course, similar to the spring function of the periodontium. Demineralization and remineralization are in dynamic equilibrium (ions). Within the proposed concept, inflammation disrupts the harmony between the periodontium and S-shaped enamel prisms, which may lead to changes in Poisson’s ratio and the anisotropy of enamel and dentin. Fluorapatite is the most caries-resistant apatite of enamel. The properties of tooth enamel depend on the Ca/P ratio [3, 10].

The causes of phagocytosis during inflammation are related to the fact that inflammation is the body’s response to injury or infection, while phagocytosis is a key mechanism for cleansing damaged tissue and fighting microorganisms. Phagocytosis is activated during inflammation due to the release of chemoattractants that attract phagocytes to the site of inflammation. Inflammation occurs only in a formed organ; it cannot occur at the genesis stage. Phagocytosis in the pulp and, consequently, at the dentin–enamel interface is considered as a process “from within.” Plaque in fissures may stimulate phagocytosis of the pulp and adjacent areas of dental tissue [4, 8].

Phagocytosis is the process by which cells capture and digest solid particles. Along with pinocytosis, it is a type of endocytosis. In some cells it is used to obtain useful substances, while in unicellular organisms it is homologous to nutrition [4].

Phagocytosis as a sign of inflammation occurs in response to plaque in fissures, disruption of surface temperature and ion exchange when the thermodynamic equilibrium of ions is disturbed. Ca-related anisotropy arises at the stage of dentin formation during dentinogenesis. The energy of these processes is proposed to be considered from the perspective of the second law of thermodynamics [3, 9].

Nervous tissue may become infected as one of the characteristics of inflammation. Load may be a basis because caries occurs only after eruption. Load may be primary, followed by infection and inflammation.

Anisotropy, homogeneity, self-healing ability and the presence of a constant melting point are determined by their internal structure.

Furthermore, enamel sections, especially after acid treatment, show oblique lines reaching the enamel surface, the so-called Retzius lines. Their formation is associated with the cyclicity of enamel mineralization during development. It is also possible to assume coherence between the spring function of the periodontium and S-shaped enamel prisms, i.e. harmony of their interaction. Disruption of such harmony leads to changes in the anisotropy of enamel and dentin and, consequently, to damage [3].

It is appropriate to add a correlation in the development of the odontogenic component underlying thermodynamic processes. In tooth development, the characteristics of the bony exoskeleton in fish with an active internal cartilaginous skeleton should be considered. In armored

fish, teeth appeared as part of the exoskeleton and were present on the body of vertebrates. Dental structures protruded externally as sensory organs, after which dense enamel appeared. Bony jaws developed in the oral cavity and teeth became located on them. Jaws appeared at the level of armored fish and formed in the skin where dentin formations already existed; the next step was teeth, followed by replacement with permanent teeth.

The odontogenic component is what appears on the back in the form of teeth. In vertebrates, during formation of the spinal cord and neural tube, some cells disperse throughout the organism, where some of them form dentin. There are fish that have no bones but have retained an odontogenic component. Sharks are covered with sharp scales called placoid scales ("shagreen skin"), which are small tooth-like structures. The spiny dogfish has a sharp external tooth. At the level of cartilaginous fish, tooth replacement appeared, i.e. biphodonts appeared. Descendants of fish became tetrapods – those in which fingers appeared.

Inflammation and thermodynamic regularities. Inflammation (Latin: inflammatio) is a complex local and general protective-adaptive process arising in response to injury (alteratio) or a pathogenic irritant. It is manifested by reactions aimed at eliminating products and, if possible, agents of injury (exudatio and others), leading to maximum restoration in the damaged area (proliferatio) [4, 8].

Clinical symptoms of inflammation include: 1. Redness (hyperemia). 2. Local increase in temperature (hyperthermia). 3. Swelling. 4. Pain. 5. Dysfunction. Acidosis promotes dissociation of salts and protein breakdown, increasing osmotic and oncotic pressure in damaged tissues and resulting in edema [4, 11].

The inflammatory process is divided into three main stages: alteration – damage to cells and tissues; exudation – release of fluid and blood cells from vessels into tissues and organs; proliferation (or the productive stage) – cell proliferation and tissue growth, resulting in restoration of tissue integrity (repair) [4, 11].

Reversible and irreversible processes and reactions, the kallikrein-kinin system, and the possibility of microvibrations from microorganisms are also considered. In enamel, inflammation and destruction begin and may be considered primary or secondary alteration. At the same time, certain processes may be attributed to proliferation, i.e. irreversible changes. Odontodiagnostics is also considered [4, 8].

The membrane is an osmotic barrier. Water and gases dissolved in it have the greatest penetrating ability. Active transport occurs against the concentration gradient and requires expenditure of ATP energy. Active ion transport systems (ion pumps) ensure a nonequilibrium distribution of ions between the cell and extracellular environment, as well as between the cytoplasm and organelles [8, 11].

Ion entropy is a characteristic of a system describing the degree of disorder associated with their movement and distribution. Ion entropy increases with increasing disorder and spatial dispersion. Ions are components of all body fluids and biologically important molecules, regulating metabolic efficiency and energy conversion.

Cellular metabolism is sensitive to changes in K^+ , Na^+ and Ca^{2+} concentrations.

The membrane potential at which a sharp influx of Na^+ ions into the cell begins and membrane depolarization develops is called the critical depolarization level (CDL), while the potential shift sufficient to reach the CDL is called the threshold value, or threshold. An action potential is a wave of excitation moving along the membrane of a living cell during nerve signal transmission. Essentially, it is a rapid short-term change in potential across a small region of an excitable cell membrane (neuron, muscle fiber or glandular cell). The action potential is the physical basis of a nerve or muscle impulse and has a signaling and regulatory role.

This membrane model is thermodynamically stable and explains its properties: plasticity, self-closing ability, fluidity, and selective permeability.

Laws of thermodynamics. The zeroth law is the norm between the dentoalveolar system and the organism. It states that if two systems are in thermal equilibrium with a third system, they are also in thermal equilibrium with each other. In this case, the tooth system, the oral cavity system, and the system of the whole organism are considered [7].

The first law states that heat is a form of energy and energy is conserved: it cannot be created or destroyed, but only transformed from one form into another, for example, into work or heat. The proposed model accounts for a localized increase in temperature (hyperthermia) [7].

The second law postulates the impossibility of spontaneous heat transfer from a colder body to a hotter one and also indicates an increase in entropy as a measure of disorder in closed systems. Changes in the anisotropy of enamel and dentin, the presence of inflammation in these areas and disruption of the spring-like properties of harmony between the periodontium and tissues are considered factors in the formation of a new state of the system [7].

The third law states that the entropy of a system tends toward a constant value as temperature approaches absolute zero, while absolute zero cannot be reached in a finite process [7].

Conclusions. Within the presented concept, the etiology of dental caries of hard tissues is considered as an inflammatory process in relation to the laws of thermodynamics. Caries is associated with microbial activity, acid formation, altered ion exchange, demineralization, and subsequent damage to the tooth's hard tissues [1–4, 7–12].

The interaction of inflammation, phagocytosis, changes in enamel and dentin anisotropy, mechanical load, ion exchange, and energetic processes is proposed to be considered as a single system. This approach combines biological, chemical, biochemical, and physical regularities [7].

Prospects for further research include experimental assessment of ion fluxes, local pH, mineral loss, mechanical properties, and inflammatory biomarkers in coordinated models of enamel, dentin, and pulp. Quantitative validation is needed before the thermodynamic interpretation can be regarded as more than a conceptual model.

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Дата першого надходження статті до видання: 30.07.2026
Дата прийняття статті до друку після рецензування: 25.08.2026
Дата публікації (оприлюднення) статті: 09.10.2026