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NAIL PLATE MICROBIOLOGY AND THE EFFECT OF NAIL PROCEDURES ON THE SKIN BARRIER

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Abstract:
This paper summarizes current data on the microbiology of the nail apparatus from the perspective of molecular research and examines the main mechanisms of damage to the skin barrier during nail procedures, including cuticle microtrauma, lipid extraction with solvents, allergic sensitization to (meth) acrylates, occlusion, and the photobiological effects of UV/LED devices. It is shown that barrier deficiency of the periungual skin creates conditions for microbial dysbiosis, maintenance of chronic inflammation, and the formation of mixed infections of the nail apparatus. The integration of microbiological and dermatophysiological data emphasizes the need for a comprehensive approach to the prevention and treatment of nail diseases, including maintaining the integrity of the skin barrier, reducing trauma. nail procedures and taking into account the polymicrobial nature of nail lesions.

Keywords: Nail plate, microbiome, onychomycosis, paronychia, Pseudomonas, (meth) acrylates, TEWL, skin barrier, acetone, UV lamps.

Introduction

The scientific novelty of this article lies in its comprehensive examination of the nail apparatus as a barrier-associated polymicrobial ecosystem, in which disruptions to the skin barrier induced by nail procedures determine changes in the microbial community and the risk of developing nail diseases. For the first time, molecular data on the nail microbiota are summarized and compared with the mechanisms of barrier damage, allowing for the substantiation of a pathogenetically oriented approach to the prevention of complications in nail practice.

The nail apparatus (nail plate, nail bed, matrix, proximal and lateral nail folds, cuticle/ eponychium) is not only an aesthetically significant structure but also a functional component of the integumentary tissues, participating in the mechanical protection of the distal phalanges and in the formation of a barrier at the interface between the external environment and the matrix/nail bed. In clinical practice, the periungual skin and cuticle are key elements of the barrier system: they limit the penetration of water, chemicals, and microorganisms into the proximal sections of the nail apparatus.

Furthermore, the nail care industry (manicures/pedicures, gel coatings, acrylic systems, solvent removal, UV/LED curing) involves regular physical and chemical exposures that can modify the barrier and provoke diseases of the nails and periungual tissues. This is reflected in detail in a dermatological review of cosmetically induced nail lesions, which describes complications associated with both the technique of performing the procedures (cuticle trauma , micro-tears) and with materials (coatings, primers , adhesives) [1].

At the same time, the very paradigm of understanding nail infections has shifted in recent years. The nail plate is not sterile, and pathological nail conditions are often characterized by complex, mixed microbial communities. Metagenomic and amplicon sequencing studies of onychomycosis show that, in addition to fungi (dermatophytes, etc.), a bacterial component is often detected in affected nails, supporting the concept of polymicrobiality and the dependence of clinical outcomes on the microecology of the nail niche [2].

Furthermore, studies of fungal diversity in healthy and diseased nails demonstrate that healthy individuals may have a rich 'background' diversity of fungi, while in onychomycosis the community structure changes, sometimes with the dominance of one taxon, but not always in a 'linear' pattern [3] .

An important clinical example of the relationship between external factors, microecology and the condition of the nail plate is green nail syndrome (green discoloration of the nail), often associated with Pseudomonas aeruginosa and risk factors such as frequent exposure to humid environments, trauma, and onycholysis. Observational data highlight that this condition can be associated with other nail pathologies, including onychomycosis, making the topic of microbiota interactions and barrier damage particularly relevant to the nail field [4].

From a dermatophysiological perspective, the key measurable indicator of skin barrier impairment is transepidermal water loss (TEWL) and the associated restoration of the epidermal lipid organization. Classic experimental data demonstrate that water flow through the epidermis is considered a signal that triggers barrier repair processes, including stimulation of lipid synthesis [5].

for nail procedures , as aggressive solvents and frequent occlusive exposure of periungual skin can increase TEWL and alter permeability. In particular, a model of acetone-induced barrier damage demonstrated a significant increase in skin permeability for compounds of varying lipophilicity, illustrating the mechanism of "lipid extraction → barrier deficiency → facilitated penetration of irritants/allergens" [6].

A particular problem in modern nail practice is the increasing incidence of allergic contact dermatitis to (meth) acrylates used in gel and acrylic systems. A large series of observations indicates a significant number of cases of ACD associated with exposure to (meth) acrylates, with the most common sensitizers being monomers such as HEMA

and related compounds; clinically, this manifests as chronic hand eczema and periungual fold lesions in both nail technicians and consumers [7].

Finally, UV/LED polymerization is a factor under discussion. Experimental work in Nature Communications has shown that exposure typical of "UV nail " Dryer " can cause DNA damage and mutational changes in mammalian cell models (including human keratinocytes) , with dose-dependent effects. These data do not constitute direct evidence of clinical carcinogenic risk in users, but they do justify the need for exposure assessment and preventive measures when frequently performing procedures [1].

Thus, the relevance of this topic is determined by the intersection of three lines of evidence: the nail and periungual skin are a barrier system sensitive to trauma and chemical agents; microbial communities in the nail niche are complex and dependent on environmental conditions; and modern nail materials and technologies carry risks of irritation, sensitization, and potential photobiological effects. The aim of this study is to summarize data on nail plate microbiology and the mechanisms by which nail procedures influence the skin barrier, linking molecular and clinical observations into a unified model for complication prevention.

Application of molecular biological methods (amplicon 16 S sequencing The use of rRNA for bacteria and ITS regions for fungi, as well as metagenomic analysis, has significantly expanded our understanding of nail microbiology. Unlike traditional culture-based diagnostics, which focuses on identifying individual pathogens, molecular approaches allow us to view the nail as a complex microbial ecosystem whose composition changes depending on the clinical condition, microenvironment, and external influences.

Several studies have shown that nail microbial communities in onychomycosis are often polymicrobial. For example, molecular diagnostics analysis of clinical samples of affected nails revealed that a significant proportion of the samples contained both fungal and bacterial taxa, while the isolated presence of fungi is less common than previously thought. This suggests that bacteria may be involved in maintaining inflammation, altering local pH , and forming persistent microbial biofilms.

Metagenomic research onychomycosis confirm diversity mushroom component : along With dermatophytes (Trichophyton rubrum , T. interdigitale) are revealed yeast (Candida spp.) and non-dermatophyte moldy Fungi . Moreover, the mycobiome structure in diseased nails differs from that in clinically healthy nails, where a wide range of fungi may also be present, but without a pronounced dominance of a pathogenic species.

Molecular studies of inflammatory diseases of periungual tissues (eg, paronychia) using 16 S rRNA sequencing showed a shift in the bacterial community towards opportunistic skin bacteria, including Staphylococcus spp ., as well as the possible involvement of anaerobic microorganisms. These data highlight the role of local conditions (humidity, occlusion, microtrauma) in the formation of microbial dysbiosis .

Pseudomonas deserves special attention. aeruginosa associated with green nail syndrome. Molecular and clinical microbiological studies confirm that colonization with P. aeruginosa often develops in the context of onycholysis and prolonged exposure to moisture and can be associated with fungal infection, further supporting the concept of mixed microbial communities in the nail.

Overall, molecular data indicate that the microbiology of the nail apparatus is determined not only by the presence of individual pathogens, but also by the structure of the microbial community, which is sensitive to disruptions in the skin barrier and external influences, including nail procedures .

Table 1 - Data from molecular studies of the microbiology of the nail apparatus

Object of study	**Method**	**Main microorganisms identified**	**Key findings**
Affected nails with onychomycosis	Metagenomic, ITS/16S sequencing	Trichophyton spp., Candida spp., bacterial taxa	Frequent polymicrobiality, a combination of fungi and bacteria
Healthy and damaged nails	ITS sequencing	A variety of fungi, including non-pathogenic species	The nail is not sterile; the disease changes the structure of the mycobiome
Clinically suspicious nails	Molecular diagnostics	Fungi + bacteria	"Purely fungal" cases are less common than previously thought.
Paronychia	16S rRNA sequencing	Staphylococcus spp., anaerobes	Inflammation is associated with microbial dysbiosis
Green nail syndrome	Molecular/clinical identification	Pseudomonas aeruginosa	Colonization is associated with moisture and onycholysis , possible coinfections

The cutaneous barrier in the nail apparatus is formed primarily by structures of the periungual skin (proximal and lateral nail folds, cuticle/ eponychium), whereas the nail plate is a dense keratin matrix with a limited barrier function. The primary function of the cuticle is to create a "hermetic seal" between the nail plate and the proximal nail fold, preventing the penetration of water, chemicals, and microorganisms into the matrix. Disruption of this seal is considered a key pathogenetic factor in inflammatory and infectious nail lesions. This position is discussed in detail in dermatological reviews devoted to cosmetically induced nail diseases, where manicures and nail modeling are considered a significant exogenous risk factor [6].

Trimmed manicures, aggressive cuticle pushing, and excessive hardware treatment of the proximal nail fold lead to microtrauma that disrupts the integrity of the epidermal barrier. Even minimal ruptures in the eponychium facilitate the translocation of microorganisms and contribute to the development of acute and chronic paronychia. Clinical observations confirm a link between regular cuticle trauma and chronic inflammation of the periungual tissues [6].

Removing gel and acrylic coatings often involves prolonged skin contact with acetone under occlusion. Acetone is a powerful extractant. epidermal lipids and is widely used in experimental models of skin barrier damage. It has been shown that acetone-induced

destruction of the stratum corneum lipid matrix leads to a significant increase in transepidermal water loss (TEWL) and an increase in the permeability of the skin to chemical compounds of varying lipophilicity [1].

For periungual skin, these mechanisms are of particular importance, since repeated cycles of dehydration and restoration create a chronic barrier deficiency, accompanied by dryness, irritation, and increased sensitivity to allergens.

Gel and acrylic systems contain (meth) acrylate monomers, particularly 2-hydroxyethyl methacrylate (HEMA), which are well-documented contact allergens. Allergic contact dermatitis of the nail folds leads to chronic inflammation, impaired keratinocyte differentiation , and secondary barrier function impairment. Such reactions have been shown to occur in both clients and nail technicians and are often occupational in nature [8].

Long-term wear of artificial surfaces creates conditions of increased humidity and limited air exchange. Combined with microdamage, this contributes to maceration of the periungual skin and a weakening of the barrier. Clinically, this correlates with an increased incidence of bacterial complications, including colonization with Pseudomonas. aeruginosa in the presence of onycholysis.

Experimental studies have shown that the radiation used in UV lamps for gel polymerization is capable of causing DNA damage and mutational changes in keratinocytes and fibroblasts in in vitro. Although these data do not provide direct evidence of clinical carcinogenic risk, they highlight the potential impact of physical factors of nail procedures on skin cells and the need to minimize exposure [9].

The combined mechanical, chemical, allergic, and physical effects of nail procedures create conditions for chronic disruption of the skin barrier, which, in turn, contributes to microbial dysbiosis and the development of infectious and inflammatory diseases of the nail apparatus.

Table 2 - The main mechanisms of damage to the skin barrier during nail procedures

The Nail Treatment Factor	**Mechanism of action**	**Barrier consequences**	**Clinical manifestations**
Trimmed/hardware manicure	Microtrauma of the eponychium	Breaking the physical barrier	Paronychia, inflammation of the folds
Acetone and solvents	Lipid extraction	↑ TEWL, increased permeability	Dryness, irritation, dermatitis
(Meth) acrylates (HEMA)	Allergic sensitization	Chronic inflammation of the barrier	ACD, hand eczema
Artificial surfaces	Occlusion, maceration	Functional barrier weakness	Infections, green nail syndrome
UV/LED lamps	Photodamage to DNA	Cellular stress	Potential photobiological risk

The results of molecular studies of the nail microbiota, combined with data on the impact of nail procedures on the skin barrier, allow us to consider nail diseases as a multifactorial process based on the interaction of exogenous factors and microecological changes. Disruption of the integrity of the periungual skin and cuticle due to mechanical trauma, chemical dehydration, or allergic inflammation creates conditions for changes in local physicochemical parameters (humidity, pH, occlusion), which directly affects the structure of microbial communities.

Molecular data indicate that in nail pathology, the microbiota is often polymicrobial, including a combination of fungi and bacteria. Under conditions of barrier deficiency, such communities can sustain chronic inflammation, form biofilms, and reduce the effectiveness of monotherapy aimed solely at the fungal component. This is particularly relevant for conditions accompanied by onycholysis and increased moisture, where bacterial taxa, particularly Pseudomonas, are present. aeruginosa, gain competitive advantages.

Allergic contact dermatitis to (meth) acrylates and repeated exposure to solvents exacerbate barrier dysfunction, creating a vicious cycle: inflammation → increased permeability → increased penetration of microbial and chemical agents → continued inflammation. Experimental data on the photobiological effects of UV lamps further highlight the role of physical factors in skin cell damage, which may indirectly influence the local immune response.

Thus, the integration of microbiological and barrier aspects emphasizes the need for a comprehensive approach to the prevention and treatment of nail diseases. Assessing the skin barrier, reducing trauma during nail procedures, and considering the polymicrobial nature of nail lesions are key areas for optimizing clinical management and reducing the incidence of complications.

Taking into account the data on the microbiology of the nail apparatus and the mechanisms of damage to the skin barrier during nail procedures, the prevention of complications should be aimed at maintaining the integrity of the periungual skin and minimizing the factors that contribute to microbial dysbiosis.

First and foremost, minimizing mechanical trauma is recommended: avoiding aggressive trimming, using gentle hardware treatments, and preserving the cuticle as a functional barrier. This reduces the risk of paronychia and secondary infection.

To prevent chemically induced barrier damage, it is advisable to limit contact time with acetone-containing solvents, avoid prolonged occlusion when removing coatings, and use products that help restore the barrier function of the skin around the nail. Reducing dehydration reduces transepidermal water loss and sensitivity to irritants and allergens.

Given the high incidence of allergic contact dermatitis to (meth) acrylates, important measures include minimizing skin contact with monomers, ensuring adequate polymerization of materials, using personal protective equipment by technicians, and early diagnosis of sensitization. If signs of contact dermatitis appear, discontinuing exposure and undergoing a dermatological evaluation are recommended.

To prevent microbial complications, it is necessary to control occlusion and humidity under artificial surfaces: do not allow hermetically sealed delaminations, promptly remove damaged surfaces, and inform clients about the risks of prolonged contact with water.

Finally, given experimental data on the photobiological effects of UV/LED lamps, it is advisable to reduce cumulative UV exposure, especially with frequent procedures, by using physical skin barriers and rationalizing the frequency of polymerization. The combined use of these measures simultaneously maintains the skin barrier and reduces the likelihood of developing an unfavorable microbial environment, which is key to preventing nail diseases in modern nail practice.

Consequently, the nail apparatus is a microbial ecosystem, where mixed communities of fungi and bacteria are often present during diseases and injuries. Nail procedures can disrupt the skin barrier around the nail through microtrauma, chemical lipid extraction with solvents, allergic sensitization with (meth) acrylates, and UV-induced DNA damage in cellular models. Understanding these mechanisms allows for targeted reduction of the risk of complications through gentle techniques, controlled exposure to chemical and physical factors, and early diagnosis of dermatitis/infections.

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