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Photobiomodulation in Combination with Biomimetic Serums: An Innovative Protocol for Skin Restoration in Patients with Post-Acne

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Abstract

Post-acne, including atrophic scars, post-inflammatory erythema (PIE), and post-inflammatory hyperpigmentation (PIH), represents not only a dermatological but also a pronounced psychosocial problem, affecting, according to clinical observations, up to 95 % of patients who have experienced acne. Traditionally applied more aggressive correction methods (ablative laser resurfacing, medium–deep chemical peels, etc.) are associated with a high incidence of adverse events and a prolonged recovery period. This is particularly critical in individuals with sensitive skin and dark phototypes (Fitzpatrick IV–VI), who, being more predisposed to the development of PIH, paradoxically at the same time belong to a high-risk group for complications with aggressive interventions. Under these conditions, the aim of the study is the theoretical substantiation of a minimally invasive synergistic protocol based on the combination of photobiomodulation (PBM) and topical application of biomimetic serums. Methodologically, the work is structured as a systematic review of publications from 2019–2024 in the PubMed and Scopus databases, with an emphasis on the analysis of cellular and molecular mechanisms of PBM and regenerative cosmeceuticals. The conclusions formulated on the basis of this analysis indicate that the combined protocol (for example, the sequence superficial peel, serum application, PBM) has a theoretical advantage over any form of monotherapy. Such a combination makes it possible simultaneously to increase the energetic potential of cells and to set a regenerative program for them, which opens up prospects for a safer and more effective reconstruction of the skin in post-acne. The presented propositions have practical significance for dermatologists, clinical estheticians, and researchers focused on the development of non-aggressive, physiological protocols of regenerative cosmetology.

Keywords: *Post-Acne, Atrophic Scars, Photobiomodulation, LED Therapy, Biomimetic Peptides, Growth Factors, Post-Inflammatory Erythema, Post-Inflammatory Hyperpigmentation, Cytochrome C Oxidase, Synergistic Protocol.*

INTRODUCTION

Acne vulgaris is among the most common dermatoses and ranks eighth among global diseases in terms of prevalence, affecting an estimated 9.4% of the world's population [1]. In the United States, the annual number of patients with acne reaches approximately 50 million people [2]. At the same time, the clinical significance of acne is determined not so much by the duration of the active inflammatory phase as by the severity and persistence of its long-term consequences, or sequelae. Up to 95% of patients who have experienced acne develop one or another form of scarring [1]. The post-acne phenotype is extremely heterogeneous: it includes not only atrophic scars caused by collagen deficiency, but also dichromia, namely post-inflammatory erythema (PIE), associated predominantly with vascular remodeling [4], and post-inflammatory hyperpigmentation (PIH), related to excessive melanin accumulation [5]. In a cohort of 1034 patients who were admitted for the treatment of atrophic acne scars in the period from August 2013 to August 2019, factors associated with efficacy were analyzed, including age, sex, Fitzpatrick skin type, energy, treatment sessions, duration of follow-up, and pigmentation. A total of 82 patients met the inclusion criteria. The patients underwent from one to three treatment sessions with a CO2 laser. The setting parameters for individual patients were the same across different treatments. Mean ECCA scores decreased from 102.70 ± 24.95 to 87.28 ± 24.48 ($p \leq 0.001$). The number of treatment sessions and the duration of pigmentation persisting for less than 3 months positively correlated with better outcomes. Erythema was observed in all patients and persisted for more than 3 months in 16 patients (19.51%). Post-inflammatory hyperpigmentation (PIH) was observed in 60 patients (73.17%) and persisted for more than 3 months in 26 patients (31.71%). Hypopigmentation was observed in one patient (1.22%), and 8 patients (9.76%) experienced exacerbation of acne. Scars after laser therapy remained in 2 patients (2.44%). These data suggest that, in the treatment of atrophic acne scars in Asians using fractional CO2 laser, 3 treatment sessions and a duration of hyperpigmentation within 3 months provide better outcomes regardless of energy, sex, age, Fitzpatrick skin type, follow-up duration, and disease course [7]. A similar spectrum of complications is compounded by a pronounced psychosocial burden, including emotional distress, reduced self-esteem, and the development of depressive disorders [1].

Classical approaches to the treatment of atrophic scars, such as fractional laser resurfacing, deep chemical peels, and dermabrasion, are based on the concept of controlled intensive tissue damage aimed at inducing fibroplasia and dermal remodeling [9, 18]. However, the highly invasive nature of these techniques is associated with a prolonged recovery period and a substantial risk of complications, including secondary infection and iatrogenic pigmentary disorders [10].

Against this background, a fundamental therapeutic paradox becomes evident. Patients with dark skin phototypes (Fitzpatrick IV–VI) and individuals with increased skin sensitivity, on the one hand, demonstrate the highest tendency to develop PIH as a primary sequel of acne [11], while on the other hand, they belong to the highest-risk group for developing iatrogenic PIH and additional scarring in response to aggressive regenerative interventions [13]. Thus, a pronounced and essentially unmet clinical need is formed for therapeutic protocols capable of effectively stimulating dermal regeneration and modulating pigmentation without triggering an intense inflammatory cascade.

The current scientific discourse predominantly focuses on the isolated evaluation of individual modalities, either biophysical (for example, photobiomodulation) [14] or biochemical (for example, topical serums) [16]. The literature virtually lacks an integrated mechanistic analysis that would conceptually substantiate their synergistic interaction at the cellular level in the context of post-acne therapy.

The aim of the study is the systematization and in-depth analysis of the fundamental mechanistic foundations of photobiomodulation (PBM) and biomimetic serums for the theoretical construction and description of a combined, minimally invasive protocol for skin restoration in post-acne.

The author's hypothesis assumes that the combined use of PBM and topical biomimetic serums initiates a synergistic regenerative cascade whose effect exceeds the simple additivity of the individual modalities. In the proposed model, PBM acts as a biophysical catalyst that optimizes cellular metabolism and the inflammatory microenvironment, whereas the serums serve as a targeted biochemical substrate, providing highly specific signaling stimuli and structural components.

The scientific novelty of the work lies in the fact that, for the first time, a theoretically grounded mechanism of synergism is described, in which the non-thermal action of PBM optimizes mitochondrial metabolism (ATP level) and the cytokine profile of fibroblasts, increasing their metabolic activity and signaling sensitivity to regenerative instructions delivered by biomimetic peptides and growth factors.

MATERIALS AND METHODS

The study was conducted as a systematic literature review with elements of conceptual analysis, aimed at integrating and comparing the cellular mechanisms underlying the therapeutic approaches under consideration. The methodological design was modified on the basis of existing recommendations used in dermatological reviews, with a deliberate shift of emphasis toward qualitative mechanistic synthesis rather than quantitative meta-analysis of clinical outcomes.

To form the evidence base, a targeted and structured search of peer-reviewed publications was performed in the electronic databases PubMed (MEDLINE), Scopus, and Web of Science.

The selected publications were further allocated to three thematic clusters, which made it possible to perform a targeted analytical synthesis:

Group 1: Pathophysiology and epidemiology of post-acne. This group included sources describing the spectrum of acne complications (PIE, PIH, atrophic scars), their typology, prevalence, and psychosocial consequences for patients.

Group 2: Biophysical mechanisms (FBM). This category included fundamental reviews and in vitro experimental studies detailing the cellular and molecular targets of photobiomodulation: mitochondrial structures, cytochrome c oxidase, cytokine profile, fibroblast behavior, and others.

Group 3: Biochemical mechanisms (Serums). This group systematized studies and reviews in the field of cosmeceuticals and bioengineering devoted to the mechanisms of epidermal barrier restoration (ceramides, NMF) and regulation of cell signaling (peptides, growth factors).

Conceptual synthesis was used as the main analytical approach. The objective was not simple descriptive aggregation of disparate data, but identification of complementary and potentially synergistic relationships between the mechanisms related to Group 2 and Group 3 in the context of addressing the clinical problem formulated on the basis of materials from Group 1. In addition, an analysis of industry reports was carried out, which made it possible to document market trends and empirical practices confirming the practical relevance and demand for combined protocols that integrate biophysical and biochemical modalities.

RESULTS AND DISCUSSION

Comparison of data from contemporary studies demonstrates that post-acne is not a single nosological entity, but a complex condition that is morphologically and pathophysiologically heterogeneous. An effective skin restoration protocol must inevitably be directed, at a minimum, toward three fundamentally different yet frequently coexisting damage mechanisms [1]:

– Atrophic scars (ice-pick, rolling, boxcar). They represent the outcome of a pronounced deficit and disorganization of the dermal extracellular matrix, primarily collagen fibers of types I and III, which leads to the formation of persistent depressions and disruption of dermal architecture [8].

– Postinflammatory erythema (PIE). Relates predominantly to vascular consequences of inflammation and is characterized by chronic dilation, microdamage, and persistent dysfunction of the capillary bed in the papillary dermis, clinically manifesting as long-standing erythematous or violaceous macules [4].

– Postinflammatory hyperpigmentation (PIH). Is a consequence of disordered pigmentation associated with excessive accumulation of melanin in epidermal and/or dermal structures against the background of a prior inflammatory cascade, which clinically results in persistent hyperchromic spots [5].

Clinical observations (see Fig. 1) confirm that the above components of post-acne in most cases do not exist in isolation. In a study including the largest group by number (36,2%) had a combination of scarring changes and PIH [7]. This fact highlights the limitations and conceptual inconsistency of monotherapeutic approaches that target only a single aspect of the pathology (for example, exclusively retexturization or only correction of pigmentation) and substantiates the need for multifactorially oriented protocols.

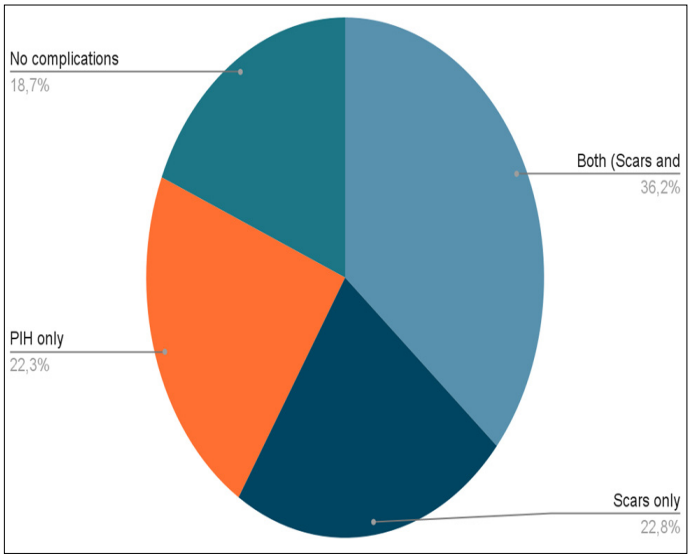

Fig. 1. Prevalence of complications in patients with post-acne (compiled by the author based on [7]).

Photobiomodulation (PBM), or low-level light therapy (LLLT), is a non-invasive and non-thermal modality that uses optical radiation in the red (approximately 600–700 nm) and near-infrared (NIR, about 700–1000+ nm) spectral ranges to induce targeted changes in cellular activity [14].

The key link in the mechanism of PBM action is associated with the absorption of light quanta by specific intracellular chromophores. In mammals, the primary photoreceptor for red and NIR irradiation is the enzyme cytochrome c oxidase (COX), corresponding to complex IV of the mitochondrial respiratory chain [15].

Absorption of photons by COX leads to optimization of electron transport, which is accompanied by two fundamentally important and rapidly occurring effects:

Enhancement of ATP synthesis. The production of adenosine triphosphate (ATP), the basic energy currency of the cell, increases; it has been shown that in certain cell populations PBM can induce an increase in ATP production of up to 70%.

Regulation of ROS levels. A short-lived, strictly dose-dependent increase in the concentration of reactive oxygen species (ROS) is formed. In this context, ROS predominantly has a signaling rather than a cytotoxic role, in contrast to the situation in oxidative stress.

Such a mitochondrial shift—a combination of an energy-directed effect (ATP increase) and a transient signaling peak of ROS—initiates activation of downstream intracellular signaling cascades. In particular, the central transcriptional regulators NF-κB (nuclear factor kappa-B) and AP-1 (activator protein-1) are activated. Their subsequent transcriptional activity leads to upregulation of genes responsible for fibroblast proliferation and migration, as well as for the biosynthesis of key components of the extracellular matrix (ECM), including collagen and elastin [14].

One of the most consistent and clinically significant effects of PBM is a pronounced anti-inflammatory and immunomodulatory action [3, 6]. This aspect is of fundamental importance for the therapeutic management of post-inflammatory erythema (PIE) and post-inflammatory hyperpigmentation (PIH), which by their nature represent predominantly inflammatory and vascular sequelae of injury.

The molecular basis of this effect involves remodeling of the cytokine profile. Numerous in vitro and in vivo studies demonstrate that under the influence of PBM:

– expression of pro-inflammatory cytokines such as interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-α (TNF-α) decreases;

– production of anti-inflammatory and regulatory cytokines, including interleukin-10 (IL-10) and transforming growth factor-β (TGF-β), increases.

This combination of effects—simultaneous suppression of the chronic inflammatory component (critical for PIE/PIH) and stimulation of the regenerative activity of fibroblasts (essential for scar tissue remodeling)—confers a unique therapeutic profile on PBM. Unlike fractional laser techniques, which induce acute inflammatory injury as a trigger mechanism for repair and are accompanied by a risk of developing PIH, PBM reduces inflammation, allowing regenerative processes to proceed under more physiological, non-fibrotic conditions.

A key parameter of the PBM protocol is the choice of wavelength, because different irradiation ranges exhibit distinct penetration depths in tissues and selectively affect different biological targets (see Table 1).

Table 1. Comparative analysis of therapeutic LED wavelengths in dermatology (compiled by the author based on [3, 6, 14]).

Wavelength	Color	Penetration depth	Primary target	Mechanism of action	Clinical application (Post-acne)
415 nm	Blue	Superficial (Epidermis)	Cutibacterium acnes	Activation of bacterial porphyrins, generation of reactive oxygen species (ROS) leading to bacterial cell death. Seboregulation.	Treatment of active inflammatory acne lesions, prevention of new eruptions.
633 nm	Red	Dermal (1–3 mm)	Fibroblasts, mitochondria (COX)	Stimulation of ATP synthesis, fibroblast proliferation, collagen synthesis, anti-inflammatory effect.	Stimulation of scar regeneration, rejuvenation, reduction of erythema (PIE).
830 nm	Near infrared (NIR)	Deep dermal / hypodermal	Mitochondria (COX), mast cells, endothelium	Maximum penetration depth, enhanced ATP stimulation, potent anti-inflammatory effect, cytokine modulation, improvement of microcirculation.	Accelerated healing, treatment of deep scars, reduction of inflammation (PIE/PIH), improved penetration of topical agents.

If FBM forms an energy and immune landscape for tissues, providing cells with resources for their functioning and creating an optimally controlled anti-inflammatory environment, then biomimetic serums complement this effect by supplying structural building blocks and guiding signaling molecules required for targeted tissue remodeling.

Initially, barrier restoration is performed (prerequisite). No regenerative protocol can be truly effective when the integrity of the epidermal barrier, that is, the stratum corneum (Stratum Corneum, SC), is compromised. The classical brick-and-mortar model of the SC considers corneocytes as bricks and the intercellular lipid matrix as mortar, which ensures their stable packing and barrier tightness. The functional integrity of this mortar is of key importance for the barrier and protective role of the skin and is determined by a strictly organized lipid composition: approximately 50% are ceramides, 25% are cholesterol, and about 15% are free fatty acids.

In patients with acne and post-acne, the barrier function of the skin is often impaired both as a consequence of the inflammatory process itself and under the influence of aggressive therapeutic interventions (for example, systemic or topical retinoids, acid peels). Therefore, the initial and mandatory stage of any regenerative protocol is the restoration of the epidermal barrier using biomimetic lipids (ceramides, cholesterol), as well as components of the natural moisturizing factor (NMF), which make it possible to reconstruct the physiological architecture and hydration status of the stratum corneum [17].

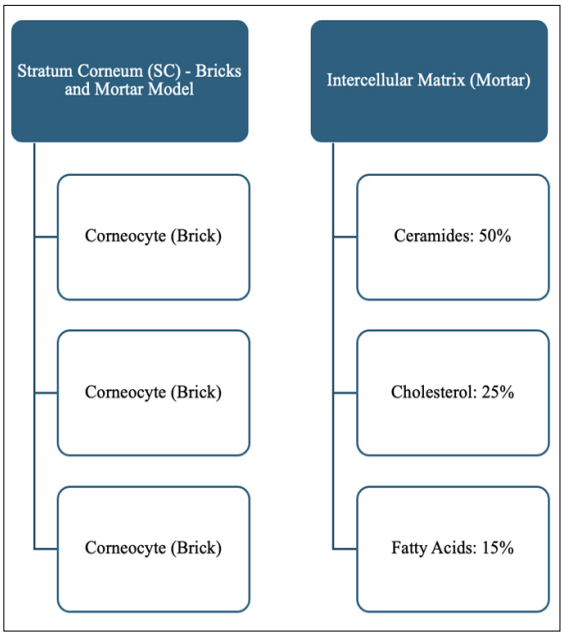

Fig. 2. The "Bricks and Mortar" model of the epidermal barrier (compiled by the author based on [16, 17]).

Subsequently, targeted signaling (peptides and growth factors) is initiated. Biomimetic peptides are short, strictly defined amino acid chains (typically comprising 2 to 10 residues) that functionally reproduce the action of endogenous signaling molecules of the skin [16]. Essentially, they act as highly specific instructions for cells: by interacting with specific receptors

on the surface of fibroblasts or selectively blocking individual enzymatic pathways, these peptides reprogram cellular behavior in the desired direction. In the context of clinical application, it is appropriate to classify them according to the predominant mechanism of action (see Table 2).

Table 2. Functional classification of biomimetic peptides for skin regeneration (compiled by the author based on [16]).

Peptide class	Mechanism of action	Examples (INCI / Trade name)	Target in post-acne
Signaling peptides	Mimic collagen degradation fragments (matrikines) or growth factors. Bind to fibroblast receptors, triggering the synthesis of new ECM.	Palmitoyl Pentapeptide-4 (Matrixyl); Palmitoyl Tripeptide-5 (Syn-Coll); Copper Tripeptide-1 (GHK-Cu).	Scar regeneration: Direct stimulation of the synthesis of new collagen I, III, elastin, and hyaluronic acid.
Neurotransmitter-inhibiting peptides	Block acetylcholine release at the neuromuscular synapse by competitively inhibiting SNARE complex proteins (e.g., SNAP25).	Acetyl Hexapeptide-8 (Argireline); Pentapeptide-18 (Leuphasyl).	Atrophic scars: Reduction of cutaneous microtension induced by facial musculature, which prevents scar contraction and promotes its leveling.
Carrier peptides	Stabilize and deliver trace elements (e.g., Copper, Manganese) required as cofactors for enzymatic processes (e.g., lysyl oxidase) in collagen synthesis.	Copper Tripeptide-1 (GHK-Cu) (also exhibits signaling properties).	Healing optimization: Providing fibroblasts with cofactors for proper cross-linking and maturation of collagen fibers.
Enzyme-inhibiting peptides	Inhibit enzymes that degrade collagen (MMPs, matrix metalloproteinases) or induce pigmentation (tyrosinase).	Oligopeptides derived from soy (MMP inhibitors); Acetyl Glycyl Beta-Alanine (tyrosinase inhibitor).	Collagen protection and PIH correction: Prevention of newly formed collagen degradation; inhibition of melanogenesis for the treatment of PIH.

Of particular interest in the context of scar correction are neurotransmitter-inhibiting peptides. Scar tissue is often fixed to the underlying anatomical structures, as a result of which facial microtension can enhance its retraction and severity. Peptides of this class, as illustrated in Fig. 3, induce myorelaxation, thereby reducing muscle tension and decreasing mechanical tension in the scar area.

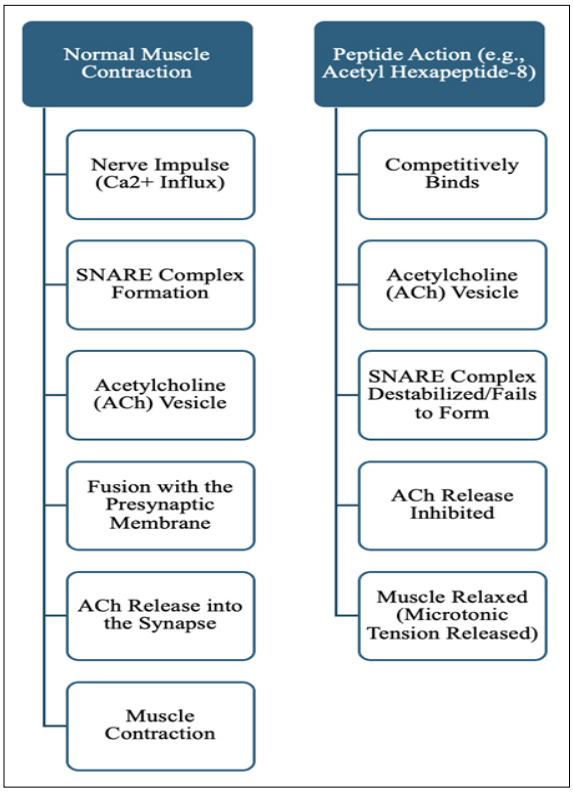

Fig. 3. Mechanism of action of a neuroinhibitory peptide (using Argireline as an example) (compiled by the author based on [16, 17]).

The rationale for the protocol's synergism is that photobiomodulation (PBM) and biomimetic serums do not merely act in parallel and sum their effects (additive action), but mutually potentiate each other's biological response, forming a qualitatively new level of influence (a truly synergistic effect).

Energy priming: PBM functionally energizes fibroblasts, creating an excess of intracellular ATP. The biosynthesis of high-molecular-weight structures, in particular collagen induced by signaling peptides, belongs to the most energy-intensive processes of cellular metabolism. In the case of insufficient energy resources, the cell is capable of perceiving a peptide signal but proves unable to implement it due to metabolic exhaustion. The additional pool of ATP generated under the influence of PBM eliminates this bottleneck, ensuring execution of the specified matrix remodeling program.

PBM modulates the tissue layer, clearing it of the chronic proinflammatory background by reducing the expression of key inflammatory mediators (IL-1β, TNF-α). As a result, fibroblasts and other effector cells shift from a mode of nonspecific defense to increased sensitivity to regenerative stimuli (TGF-β induced by PBM, as well as peptides of the biomimetic serum), which transfers the tissue from a state of chronic standby inflammation to a state of predominantly reparative activity and regeneration.

PBM, especially when using a wavelength of 830 nm, improves local microcirculation. This is accompanied by an increased delivery of oxygen and metabolic substrates and theoretically promotes more uniform distribution and enhanced absorption of topically applied active components of the serum. Industry data and practical reports confirm this trend, describing devices (for example, Water Light Injector) in which LED therapy and serum infusion are purposefully combined in order to enhance the depth and efficiency of penetration.

The proposed protocol was developed taking into account the author's clinical specialization in sensitive skin, acne, and LED therapy and is aimed at maximizing efficacy with the lowest possible aggressiveness of intervention. It is based on the principle of reducing risks associated with traditional invasive and traumatic techniques [13].

Phase 1: Preparation (controlled exfoliation).

Action: Application of a gentle, superficial chemical peel with low irritant potential, for example based on alpha-hydroxy acids (AHA) with a large molecular weight (mandelic acid) or polyhydroxy acids (PHA).

Mechanism: The agents mentioned induce keratolysis, that is, controlled weakening of intercellular junctions of corneocytes in the stratum corneum. In contrast to more aggressive peels (TCA, phenol), they do not induce pronounced coagulative necrosis and a significant inflammatory response in the dermis.

Purpose: Short-term and predictable increase in the permeability of the stratum corneum in order to optimize subsequent penetration of the biomimetic serum in Phase 2.

Phase 2: Biochemical signaling.

Action: Application of a sterile biomimetic serum including a multi-complex: 1) signaling peptides (for example, GHK-Cu), 2) neuroinhibitory components (for example, Acetyl Hexapeptide-8), 3) elements of the skin barrier (ceramides, hyaluronic acid) [17].

Purpose: Simultaneous delivery to the cells of the epidermis and dermis of instructions (signaling peptides that set the directions of remodeling and neocollagenesis) and building material (ceramides and other structural components) required for restoration of barrier function and matrix.

Phase 3: Biophysical catalysis.

Action: Immediate initiation of PBM (LED therapy) using a combined spectrum: red light 633 nm (predominant stimulation of fibroblasts) and near infrared range 830 nm (deep anti-inflammatory action and enhancement of ATP synthesis).

Purpose: Initiation of the protocol into its active phase: providing fibroblasts with an energy resource (ATP) sufficient for implementing the instructions embedded in the serum, while simultaneously suppressing potential microinflammation, a key trigger for the formation of PIE/PIH.

Key barriers to implementation include:

1. Standardization of PBM: The absence of unified dosimetric approaches (power density, total dose, number and frequency of procedures) with respect to PBM remains a significant problem of modern dermatological practice.
2. Quality of cosmeceuticals: The effectiveness of Phase 2 is entirely determined by the quality of the cosmeceutical product: stability of the formula, validity of concentrations, and efficiency of the delivery system of active components (peptides, ceramides) to the target skin layers.
3. From the safety standpoint, the overall risk directly associated with this protocol is minimal and substantially lower than when using aggressive techniques. PBM (LED) is a nonthermal technology with a favorable safety profile that is not associated with exposure to UV radiation. Adverse events are generally limited to mild, rapidly resolving erythema. The main array of potential risks is associated with Phase 1 (peeling) and Phase 2 (serum) and includes the possibility of developing allergic or irritant contact dermatitis to individual components of the formulas used.

CONCLUSION

The conducted analysis convincingly demonstrates that photobiomodulation (PBM) and biomimetic serums possess not merely complementary but highly complementary and genuinely synergistic mechanisms of action in the treatment of the complex postacne phenotype, which includes atrophic scars, postinflammatory erythema (PIE) and postinflammatory hyperpigmentation (PIH).

PBM in the wavelength range of 633–830 nm functions predominantly as a biophysical catalyst. Its key molecular target is mitochondrial cytochrome c oxidase, the activation of which leads to optimization of cellular respiration processes, an increase in ATP production [15] and, critically, to a shift of the cytokine profile toward an anti-inflammatory and pro-regenerative state (a decrease in IL-1β and TNF-α with a simultaneous increase in IL-10 and TGF-β).

Biomimetic serums, in contrast, act as a biochemical substrate. They provide cells with a highly specific set of instructions (signaling peptides, neuroinhibitors) and building blocks (ceramides, amino acids) required, on the one hand, by fibroblasts for neocollagenesis and remodeling of the extracellular matrix and, on the other hand, by epidermal cells for the restoration and stabilization of the epidermal barrier.

The stated aim, namely the theoretical substantiation of the protocol, has been achieved. The authorial hypothesis of a synergistic interaction, in which PBM (as a catalyst) and biomimetic serums (as a substrate) generate not an additive but a multiplicative therapeutic effect, appears mechanistically well substantiated. PBM creates an energetic basis (ATP) and establishes a favorable anti-inflammatory microenvironment, thereby enabling cells to fully implement the program defined by the active components of the serum. The proposed three-stage protocol (Peeling → Serum → PBM) represents a logical and internally consistent clinical implementation of this synergism.

The conclusions obtained have high practical value for practicing dermatologists and clinical aestheticians who focus on the use of safe and scientifically substantiated protocols in patients with postacne. The approach is of particular importance for the growing group of patients with contraindications to aggressive interventions, including individuals with sensitive skin and dark phototypes (Fitzpatrick IV–VI), in whom minimization of inflammation is a key prerequisite for the prevention of PIH [12].

The formulated concept underscores a pronounced need for randomized controlled trials to achieve quantitative validation of this synergistic protocol. Prospective studies should focus on the refinement and optimization of intervention parameters (dosimetric characteristics of PBM, concentrations and composition of peptide complexes) and on direct comparison of the combined protocol with monotherapy (isolated PBM or isolated use of serums) in different clinical patient cohorts.

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