

BIOLOGICAL AND MORPHOLOGICAL CHANGES IN THE SKIN AFTER LASER REMOVAL OF TATTOOS AND PERMANENT MARKS

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Abstract

This article summarizes current data on biological and morphological changes in the skin after laser tattoo and permanent makeup removal. It examines the mechanisms of pigment destruction (photothermal and photoacoustic effects), the primary inflammatory response, dermal remodeling, and structural changes in the epidermis. Key cellular and molecular processes, including the activation of macrophages, cytokines, and matrix metalloproteinases, are analyzed.

Keywords: Laser tattoo removal, permanent makeup, biological changes, skin morphology, photothermolysis, photoacoustic effect, dermal remodeling, inflammatory response, macrophages, matrix metalloproteinases.

Introduction

The scientific novelty of the article lies in the systematization of modern data on morphological and biological changes in the skin after laser removal of tattoos and permanent makeup, with an emphasis on the correlation of these changes with clinical outcomes, as well as in identifying factors influencing the effectiveness and safety of the procedure.

With the development of aesthetic medicine and the growing popularity of decorative tattoos and permanent makeup procedures, the need for high-quality removal has also increased. Laser technologies, which allow direct action on the pigment while minimizing damage to surrounding tissue, have become the methods of choice. For example, in the scientific article «Lasers for tattoo removal: a review» it is noted that the use of lasers based on the principle of selective Photothermolysis allows for the removal of both black and colored tattoos with varying degrees of effectiveness [1].

The mechanism of removal is based on the selective absorption of the laser pulse by the pigment and the destruction of pigment granules through thermal and/or acoustic effects. In the study "Laser-Tissue Interaction in Tattoo Removal by Q-Switched Lasers" describes in detail how short pulses create a photoacoustic effect leading to the destruction of pigment particles [2].

Despite significant advances, tattoo removal remains a complex task due to tissue response, pigmentation characteristics (color, depth, density), the patient's skin phototype, and the wavelength used. As noted in the article «Laser Tattoo Removal : A Clinical Update», on

average 7-10 sessions are required for a satisfactory result, and patients should be informed about the options, risks and possibilities [3].

Modern research also emphasizes that the choice of wavelength, pulse duration, fluence, and interval between treatments are important for achieving optimal results. For example, the article "Laser Tattoo Removal" indicates that the key element is the selection of the wavelength taking into account the color of the tattoo and the skin phototype, as well as the suppression of thermal damage to adjacent tissues [4].

Thus, the present work aims to systematically analyze the biological and morphological changes in the skin that occur after laser removal of tattoos and permanent makeup, taking into account modern data on the mechanisms, features of laser action and the response of skin structures to this effect.

Laser tattoo and permanent makeup removal procedures are based on the principle of selective photothermolysis, where a laser pulse of a specific wavelength is absorbed by the pigment (chromophore) faster than the heat is dissipated into the surrounding tissue.

Modern lasers with short pulse durations (nanoseconds, picoseconds) additionally emphasize the photoacoustic or photomechanical effect: very rapid heating of the pigment particle causes the formation of an acoustic wave and cavitation, leading to its destruction. Phagocytosis is also involved: after pigment fragmentation, macrophages capture the particles and transport them through the lymphatic system.

After laser irradiation, the following primary changes are observed in the skin:

1. Formation of intracellular or extracellular vacuoles/bubbles (particularly at very short pulses) as a consequence of cavitation and acoustic stress.
2. Immediate fragmentation of pigment granules: reduction of their size, appearance of small particles, redistribution in the dermis.
3. Macrophage activation and uptake of pigment fragments; subsequent lymphatic removal.
4. Local damage to microvessels and capillaries: expansion, extravasation, microedema, which may appear immediately after the procedure [5].

Table 1 - Basic mechanisms of laser action and primary skin changes

Mechanism of action	Description	Primary changes
Selective photothermolysis	Pulse duration \leq Thermal Relaxation Pigment time \rightarrow targeted heating	Pigment fragmentation, minimal tissue damage
Photoacoustic /photomechanical effect	Very short pulse \rightarrow rapid expansion/cavitation around the pigment particle	Formation of vacuoles, acoustic explosion of a particle
Phagocytosis and lymphatic removal	Macrophages capture destroyed particles \rightarrow removal via lymph	Reduction of pigment mass, gradual lightening
Microvascular injury	High-peak impulse creates shock waves \rightarrow vascular changes	Erythema, microedema, possible pinpoint hemorrhage

Laser exposure of skin pigment leads to a complex of morphological changes that develop immediately after the procedure and over the following hours, weeks, and months. Key phenomena include the formation of vacuoles/ microcavities in the epidermis and dermis (laser-induced optical breakdown, LIOB), mechanical fragmentation of pigment particles, activation of an inflammatory response with leukocyte and macrophage infiltration, and subsequent remodeling of dermal matrix (collagenogenesis, changes in elastic fibers). These processes determine clinical outcomes: the rate of bleaching, the risk of hypo/hyperpigmentation, and the likelihood of scarring.

Immediate (first minutes–hours) morphological manifestations:

1. Laser-induced vacuoles/ microspaces (LIOB): under fractional picosecond and some other modes, spherical voids that do not stain with hematoxylin and eosin appear in the epidermis and superficial dermis; they are associated with optical breakdown and cavitation. The presence of LIOB has been documented in human and ex vivo models [6].
2. Photomechanical fragmentation of pigment: short pulses (nanoseconds, picoseconds) cause rapid thermal/acoustic stress and mechanical destruction of ink granules - the formation of small fragments. This is observed in histological sections and in vivo / porcine models [2].
3. Vascular reactions and microedema: capillary dilation, pinpoint hemorrhages and plasma extravasation are frequent early phenomena, clinically manifested by erythema and edema [7].

Early healing (1-7 days):

1. Neutrophil and macrophage infiltration: inflammatory cells arrive at the site and begin phagocytosis of pigment fragments; macrophages contain intracellular pigment upon histological examination. This stage is critical for pigment removal via the lymphatic system [8].
2. Scab formation and epidermal regeneration: the damaged epidermis forms a crust, then reepithelialization occurs; under gentle conditions, the epidermis is restored without a scar.

Late changes (weeks - months) :

1. Dermal remodeling : activation of fibroblasts, increased collagen synthesis and reorganization of matrix fibers; a number of studies have shown increased collagenogenesis after Q- Switched and picosecond exposures, which can improve skin texture, but in some cases contribute to the formation of a consolidated scar [9].
2. Persistence of macrophages with pigment: some fragments are captured by macrophages and remain in the skin for a long time; clinically, this is manifested by “residual” (ghost) pigment.
3. Pigmentary disorders: long-term hyperpigmentation (especially in dark phototypes) or post-inflammatory hypopigmentation in deep thermal/ ablative injury [2].

Levels and localization of changes:

1. Epidermis: vacuolization, stratum bedsores Corneum, a temporary loss of pigment by melanocytes due to strong heating; rethelialization in most cases occurs without atrophy with proper care.
2. Dermis: fragmentation of pigment granules, macrophage infiltration, activation of MMPs and remodeling of collagen bundles; in the area of deep injury - risk of fibrosis and formation of hypertrophic scar.

Table 2 - Morphological changes in the skin after laser tattoo/permanent makeup removal (by time and tissue level)

Time interval	Epidermis	Dermis	Cellular-molecular	Clinical manifestations
Immediate (0–24 h)	Vacuolization (LIOB), microvoids; stratum disorders corneum .	Mechanical fragmentation of pigment; microhemorrhages.	Release of DAMPs , activation of HSPs and early proinflammatory cytokines.	Erythema, edema, “instant” darkening/“burning” of pigment.
Early (1–7 days)	Repitalization , crust formation	Infiltration of neutrophils and macrophages; onset of phagocytosis.	Increased IL-1 β , IL-6; early MMP activation.	Scab, itching, possible secondary infection if not properly cared for.
Later (weeks–months)	Epidermal restoration; possible hypo /hyperpigmentation	remodeling , increased dermal density, pigmented macrophages; risk of fibrosis in deep trauma.	Long-term activation of remodeling pathways (MMP/TIMP), collagen I/III remodeling.	Tattoo lightening, “ghost effect”, hypo /hyperpigmentation, possible scarring.

Practical analysis of biological and morphological changes in the skin after laser treatment has important clinical implications. Morphometric characteristics, such as the severity of the LIOB effect and vacuolization, can serve as a guide for assessing the intensity of photomechanical treatment and the need to reduce energy parameters in patients with a high risk of complications (e.g., darker skin types and facial areas).

Intervals between treatments should take into account the temporal characteristics of immune clearance: phagocytosis and lymphatic removal of pigment continue for several weeks, which justifies avoiding excessively frequent sessions. Remodeling Dermal matrix regeneration after laser treatment can improve skin quality, particularly in areas with scarring. However, excessive depth or density of treatment increases the risk of fibrosis and hypertrophic scarring. Therefore, individualized dosing, phototype-specific protection regimens, and rational postoperative patient management are key to achieving optimal aesthetic results while minimizing risks.

Morphological and biological changes in the skin after laser tattoo and permanent makeup removal directly determine the effectiveness and safety of the procedure. Fragmentation of pigment particles and their subsequent macrophage clearance ensure gradual tattoo lightening, while LIOB intensity and vacuole formation allow us to assess the degree of photomechanical action.

Dermal remodeling, including stimulation of collagenogenesis and reorganization of matrix fibers, can improve skin texture, especially in areas of scar tissue. At the same time, excessive epidermal damage or deep Traumatization of the dermis increases the risk of hypo- or hyperpigmentation, the formation of scars and residual (“ghost”) pigment.

Thus, understanding the dynamics of morphological changes allows us to predict clinical outcomes, adjust laser parameters, plan intervals between sessions, and tailor postoperative care. A personalized approach minimizes complications and improves aesthetic results, making the procedure safer and more predictable.

Therefore, laser removal of tattoos and permanent makeup initiates a complex of biological and morphological changes in the skin, including pigment fragmentation, an inflammatory response, dermal remodeling, and epidermal changes. Understanding these processes allows us not only to predict the outcome of the procedure but also to optimize treatment parameters and aftercare. In particular, the balance between effective pigment destruction and minimizing skin damage is key.

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