Cosmetic Treatment of Stretch Marks

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Abstract

Stretch marks (*striae distensae*) are cutaneous lesions that accompany the hormonal upheavals of the major stages of life: puberty and pregnancy are the main causes. Today, other additional triggering factors may be involved such as corticosteroid treatment and obesity but also cosmetic surgery and intensive body building for men. Stretch marks develop abruptly and progress slowly from a pink colour to an ivory white or sometimes nacreous hue. They never disappear.

Although the etiology of stretch marks has yet to be fully defined, there are 4 determinant factors: high circulating corticosteroid levels, an early inflammatory stage, marked extension of the skin and genetic and tissue susceptibility. Limiting the effect of mast cell degranulation releasing trypsin and chymotrypsin and offsetting leukocytic elastolysis is considered crucial. Helping fibroblasts to repair the matrix also remains indispensable.

This can be achieved by the combination of matrikines with plant extracts which exhibited a significant enzymatic inhibition associated to an *in vitro* stimulation of matrix protein synthesis by fibroblasts. These properties were objectively demonstrated *in vivo*. The results showed an increase in dermal thickness accompanied by a decrease in the depth of the stretch mark depression (72%), a significant improvement in colour (-22%) and a very significant improvement in stretch mark width (-27%).

The matrix regeneration and the wound healing activity can be reinforced by the terpenoids found in 2 well-known plants: *Siegesbeckia orientalis L.* and *Centella asiatica* of which its efficacy in reducing stretch marks length and width, demonstrated *in vivo*, can reach 2 mm.

Even though stretch marks never disappear, it is possible, with a suitable cosmetic treatment, to reduce their appearance.

Introduction

Stretch marks (*striae distensae*) are cutaneous lesions that accompany the hormonal upheavals of the major stages of life: puberty and pregnancy are the main causes.

Today, other additional triggering factors may be involved such as corticosteroid treatment (Rogalski, 2002) and obesity but also, more recently and more unexpectedly, aesthetic surgery (skin stretching due to breast and buttock augmentation, White *et al.*, 1995). Thus, over 60% of women are affected by stretch marks.

Men are not spared, although the prevalence is lower: puberty, obesity and intensive body building (particularly the shoulders, torso and arms) are the triggering factors. Stretch marks develop abruptly and progress slowly from a pink to violet colour, then to an ivory white or sometimes nacreous hue. They never disappear. They are thus experienced as an uncalled for calamity.

Although the precise etiology of stretch marks has yet to be fully elucidated, there are four determinant factors:

1. High circulating corticosteroid levels
2. An early inflammatory stage
3. Marked extension of the skin (growth or weight gain)
4. Genetic and tissue susceptibility

High corticosteroid levels

All the findings indicate a strong correlation between endogenous glucocorticoid secretion and the emergence of stretch marks. Under the action of corticosteroids, the growth of both keratinocytes and fibroblasts is blocked, as is their synthesis of collagens I and III: 90% less than normal skin. In addition, fibronectin synthesis is reduced: 93% less than normal skin (biopsy specimen results reported by Lee, 1994).

The early inflammatory stage

A study by SHEU *et al.*, 1991, including young subjects during puberty and presenting with incipient stretch marks showed that stretch mark biopsy specimens were the sites of inflammation displaying granulocyte-macrophage colony stimulating factor (CSF) and monocyte chemoattractant protein (MCP1). This is accompanied by visible elastolysis triggered by mast cell degranulation with release of trypsin and chymotrypsin. The picture of progressive destruction of the extracellular matrix in the