Microdermabrasion: A molecular analysis following a single treatment

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Background: Microdermabrasion is a popular method of superficial skin resurfacing. It is unclear if dermal remodeling actually occurs.

Objective: To rigorously investigate the molecular alterations observed following a single microdermabrasion treatment.

Methods: Forty-nine subjects received a single microdermabrasion treatment to buttock skin. Serial in vivo biochemical and immunohistological analyses were performed. Reverse transcriptase real-time polymerase chain reaction and immunohistochemistry assays were used to evaluate changes in transcription factors (AP-1, NF-κB), primary cytokines (interleukin-1β, tumor necrosis factor—α), matrix metalloproteinases (MMP-1, MMP-3, MMP-9), barrier repair enzymes (acetyl-coenzyme A carboxylase, 3-hydroxy-3-methylglutaryl coenzyme A reductase), and type I procollagen.

Results: Elevation of transcription factors, primary cytokines, and matrix metalloproteinases occurs rapidly after a single microdermabrasion treatment. Two of 11 subjects also demonstrated increased type I procollagen messenger RNA and protein levels 14 days after treatment. No alteration in stratum corneum thickness was detected.

Conclusion: Microdermabrasion activates a dermal remodeling/wound healing cascade with minimal epidermal disruption. Evidence now exists to further study manipulation of variables such as number and timing of microdermabrasion sessions. (J Am Acad Dermatol 2005;52:215-23.)

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Supported by the Babcock Endowment for Dermatological Research at the University of Michigan, Bellamed Products, and Dermatology Foundation Patient Directed Investigation Grant to D. J. K.

Bella Products donated the microdermabrasion machine, hand pieces, and crystals. Bella Products had no knowledge of the outcomes of this study. As a condition of acceptance of donations from Bella Products, the study was designed to be performed, analyzed, and reported solely by the faculty and staff of the Department of Dermatology at the University of Michigan.

Conflicts of interest: None identified.

A portion of the data herein was presented at the American Society for Dermatologic Surgery meeting in New Orleans, Louisiana, October 9, 2003.

Accepted for publication October 6, 2004.

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0190-9622/$30.00

doi:10.1016/j.jaad.2004.10.008

Abbreviations used:

AP:	activator protein
ATF:	activating transcription factor
ERK:	extracellular signal-regulated kinase
HMG CoA:	3-hydroxy-3-methylglutaryl coenzyme A
IL:	interleukin
JNK:	C-jun NH terminal kinase
MAB:	monoclonal antibody
MAP:	mitogen activated protein
MMP:	matrix metalloproteinase
mRNA:	messenger RNA
NF:	nuclear factor
NICHD:	National Institute of Child Health and Human Development
OCT:	ornithine carbamoyltransferase
RT-PCR:	reverse transcriptase real-time polymerase chain reaction
TNF-α:	tumor necrosis factor—α

Microdermabrasion has become an extremely popular method of superficial skin resurfacing. With approximately 860,000 cases of microdermabrasion performed in

2003, it follows only botulinum toxin injection and laser hair removal in terms of nonsurgical, cosmetic procedures performed in the United States.[1] The purported benefits of microdermabrasion include improvement in acne scars,[2] acne,[3] skin texture, mottled pigmentation,[4] and fine wrinkles.[5,6] It has been reported that microdermabrasion treatment results in histologic changes in collagen and elastin content of the dermis, with resulting improvement noted in acne scars and fine wrinkles in a subset of patients.[2,7,8] However, it is unproven if dermal remodeling effects actually occur at a biochemical and molecular level. Our purpose was to rigorously investigate the potential dermal remodeling effects at a molecular level following a single microdermabrasion treatment. Microdermabrasion has been reported to increase transepidermal water loss, which is evidence of epidermal barrier disruption.[9] We sought to determine if microdermabrasion: (1) induces epidermal signal transduction pathways associated with remodeling of the dermal matrix, and (2) causes epidermal barrier disruption that is sufficient enough to result in histologic alteration of the stratum corneum and increased gene expression of enzymes involved in stratum corneum repair.

METHODS

This study was approved by the University of Michigan Medical School Institutional Review Board for Human Subjects Research. All subjects provided written informed consent. Forty-nine subjects, aged 18 to 73 (average age, 35.7 years) received a single aluminum oxide microdermabrasion treatment to buttock skin using the Bellamed Microdermabrasion unit. Three 2 cm × 2 cm square areas of buttock skin were treated with microdermabrasion in the following manner. The microdermabrasion device was set to its most aggressive setting (15 mm Hg) and one pass with the microdermabrasion hand piece was performed in the horizontal, vertical, and oblique directions for a total of 3 passes. This method of treatment resulted in a mild pink erythema similar to what is seen in our day to day clinical practice of microdermabrasion. Four additional subjects underwent tape stripping of buttock skin in order to compare microdermabrasion barrier disruption to that of tape stripping. Cellophane tape (3M Office Supply Division, St. Paul, Minn) was repeatedly applied to an area of buttock skin that measured approximately 2 cm × 2 cm until the skin became red, shiny, and tacky to touch. Punch biopsies (4 mm) were obtained from treated and untreated (control) skin at different sets of time intervals ranging from 15 minutes to 16 days after treatment. Each subject served as its own control with the baseline (no treatment) biopsy. Skin specimens were either snap frozen in liquid nitrogen or embedded in optimal cutting temperature (OCT) medium and frozen.

The OCT embedded tissue was sectioned into 7 μm thick sections and immunohistochemically stained for matrix metalloproteinase (MMP) -1 (interstitial collagenase) with monoclonal antibody MA 1346; MMP-3 (stromelysin-1) with monoclonal antibody (MAB) 13412; MMP-9 (gelatinase B) with monoclonal antibody MAB 13415 (MMP antibodies were obtained from Chemicon International, Inc., Temecula, Calif); c-jun component of AP-1 (Transduction Laboratories, Lexington, Ky); p50 component of NF-κB (Santa Cruz Biotechnology, Santa Cruz, Calif); type I procollagen (SP1.D8 developed by Dr Heinz Furthmayer, and obtained from the Developmental Studies Hybridoma Bank developed under the auspices of the NICHD and maintained by the University of Iowa, Department of Biological Sciences, Iowa City, Iowa); type III procollagen (Chemicon International, Inc. Temecula, Calif); and tropoelastin (Elastin Products Co, Owensville, Mo). Presence of tissue-bound primary antibody was visualized with a secondary antibody-peroxidase complex.[10] Substitution of isotype γ-globulin for primary antibody was used to assess specificity of staining. No staining was observed with any isotype γ-globulin.

Snap frozen tissue was used to quantify messenger RNA (mRNA) levels using reverse transcriptase/real time quantitative polymerase chain reaction (RT-PCR).[11] Transcript levels for the key enzymes involved in barrier lipid synthesis (3-hydroxy-3-methylglutaryl coenzyme A [HMG−CoA] reductase and acetyl-coenzyme A [CoA] carboxylase), primary cytokines (interleukin [IL] -1β, tumor necrosis factor [TNF]-α), extracellular matrix-degrading metalloproteinases (MMP-1, MMP-3, and MMP-9), c-jun component of AP-1, type I and type III procollagen, and tropoelastin were quantified. Nile Red staining of the stratum corneum was performed as described[12] using 1:80 dilution of the Sorensen-Walbum buffer for 8 minutes to expand the stratum corneum.

Statistical methods

Changes in biochemical endpoints over the time course of the study were statistically evaluated using the repeated measures analysis of variance. Individual pairwise comparisons of values at each subsequent time point with baseline levels were made with the Dunnett test. The Type I error rate was set at 0.05. When necessary, logarithmic transformations of the data were made to achieve normalcy before analysis; however, the figures depict the data on the