Discussion

Fat Liquefaction: Effect of Low-Level Laser Energy on Adipose Tissue

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Discussion by Spencer A. Brown, Ph.D., Rod J. Rohrich, M.D., and James J. Chao, M.D., Ph.D.

Neira et al. have explored the use of low-level laser energy on in vivo and in vitro models of adipose tissue biology. In a small number of patients, low-level laser irradiation was used before lipoplasty procedures. The authors further present interesting in vitro results examining the subsequent effects of low-level laser irradiation on adipocyte metabolism. From in vitro studies, scanning electron microscopy photomicrographs of freshly isolated human adipose tissue exposed to low-level laser irradiation (1 to 4 J/cm^2; 4 to 6 minutes) show cellular disruption compared with nonaffected, nonirradiated adipose tissue. Using scanning electron and transmitting electron microscopy, the authors postulate that an adipocyte-specific cell membrane pore is activated, allowing the passage of fat out of the cell. The authors hypothesize that low-level laser irradiation before laser-assisted lipoplasty will produce fat-depleted adipocytes and result in a more efficient and safer lipoplasty procedure. For the in vivo study, 12 healthy women underwent lipoplasty with low-level laser therapy and had excellent results. The authors conclude that laser-assisted lipoplasty should be considered when performing lipoplasty techniques.

Today low-level laser irradiation therapy is widely considered to have nonthermal, radiobiologically therapeutic effects by accelerating the wound-healing process by means of increased cellular proliferation, reduced inflammation, and promotion of vascular microcirculation.[1–3] The Food and Drug Administration recently approved low-level laser irradiation for pain therapy. With low-power (5 to 10 mW) and total energy (1.2 to 3.6 J/cm^2) at 635 nm, low-level laser irradiation is considered safe and a nonsignificant risk, according to the Food and Drug Administration. In in vitro studies on isolated whole blood, low-level laser irradiation was not associated with cellular damage or loss of cellular viability.[4]

The authors report that low-level laser irradiation influences the gross appearance of adipose cells embedded in freshly isolated tissue removed by lipoplasty. In vitro irradiation of dermal tissue for 2, 4, or 6 minutes, compared with controls, ultimately damages adipose cells. They further tested whether the presence of tumescent fluid improved the disruption of adipose cells in the isolated tissues. In the low-level laser studies, tumescent fluid seemed to accelerate adipocyte deformation. However, the authors did not directly test whether the adipocytes were still viable, and no data were provided with regard to the gross appearance of microvascular cells, nerves, muscle, or other cell types associated with dermal and subdermal tissues. The authors did state that the reculturing of low-level laser–treated adipocytes resulted in viable adipocytes (no data or references were provided). From an in vitro standpoint, the photoactivation of adipocytes toward fat mobilization and maintaining viability would be important to substantiate with well-controlled dose/time experiments. These data seem to be in contrast to the increased cell proliferation and viability studies associated with low-level laser irradiation and the nonsignificant risk status of low-level laser irradiation.

From transmitting electron microscopic

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analyses of low-level laser–treated adipose tissue, this research team postulates that fat was released from the adipocytes. The chemical form of fat—triglycerides versus glycerol and fatty acids—was not directly tested. The authors proposed that a specific cell-associated pore or channel was associated with loss of intracellular fat and cell formation. If intracellular triglycerides were hydrolyzed by hormone-sensitive lipase or lysosomal acid hydrolase, free fatty acids would be transported through reported fatty acid transporter proteins found in human adipose tissue.[5–7] Currently, only excess extracellular fatty acids and not intracellular fatty acids are reported to be transported through the plasma membrane, because specific fatty acid– binding proteins prevent movement of the fatty acids out of the cell.[5,8] The authors may have discovered a unique and adipocyte-specific channel or pore. However, more rigorous experiments are required to demonstrate specificity from other reported pores/channels or a new function of known proteins.

The in vitro results were remarkable for the dramatic effects provided by 4 to 6 minutes at a low-level laser energy signature. The authors did not comment on the depth of tissue that was isolated for scanning electron and transmitting electron microscopy analyses. The results of fat-depleted adipocytes may be limited to cells closely associated with subdermal locations proximal to the epidermis. Lipoplasty procedures remove adipose tissue from variable depths (1 to 6 cm) below the skin surface. It is difficult to understand from laser theory on energy transfer how the stimulatory effect of low laser irradiation at 635 nm with low fluence modulates or damages adipocytes deep below the skin surface. There is a well-known drop of the photon density during light penetration in soft tissues. Human skin can reflect, absorb, and/or transmit light rays. That portion of laser light that does penetrate will also be influenced by the percentage of melanocytes (light skin, 1.3 to 6 percent versus dark skin, 18 to 43 percent) and density of microvascular structures. With only 0.3 to 1.0 percent of the laser light penetrating skin, the cellular or tissue structures acting as acceptors to the 635-nm light that penetrates the skin to produce these nonthermal results are not reported. It is difficult to conceptualize that only one adipocyte surface protein would be photoactivated and other proteins would not. The authors did not present microscopic or histologic analyses on adipose tissue collected during lipoplasty from the 12 subjects.

Our laboratory had the opportunity to perform lipoplasty with the Erchonia laser (Majes-Tec Innovations, Inc., Mesa, Ariz.) on a porcine model. In two animals, suction-assisted lipoplasty, ultrasound-assisted lipoplasty plus suction-assisted lipoplasty, and laser-assisted lipoplasty were performed in the presence of superwetting solution. In the laser-assisted lipoplasty area, skin was exposed for 12 minutes with the protocol provided. From all conditions, tissue and lipoaspirate samples (middle of the top layer) were collected. Cell viability measurements and scanning electron microscopic results were determined. In the lipoaspirate specimens, the cell viability of suction-assisted lipoplasty equaled that of the laser-assisted lipoplasty specimens; i.e., there was no decrease in cell death with laser-assisted lipoplasty. This would be consistent with minimal damage but not cell death, as reported by the authors. Our preliminary results would seem to support previous reports that low-level laser irradiation does not damage cellular structures. It is possible that the porcine model may provide dramatically different results because of skin coarseness and other features that interfere with low-level laser irradiation penetration in comparison with human skin anatomy. We, along with other investigators, look forward to further examination of low-level laser irradiation in human lipoplasty procedures in a randomized clinical trial.

The authors have presented preliminary data that low-level laser irradiation may provide a powerful tool for both basic science research and clinical applications. They are to be congratulated on their efforts to explore the mechanism in adipocytes, a cell type known to be difficult to process and evaluate. It will be important to understand the mechanisms of action in isolated adipocytes and in intact tissues. Of clinical importance, plastic surgeons and others should wait for further reports from rigorous randomized clinical human trials before using low-level laser irradiation in lipoplasty procedures.

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