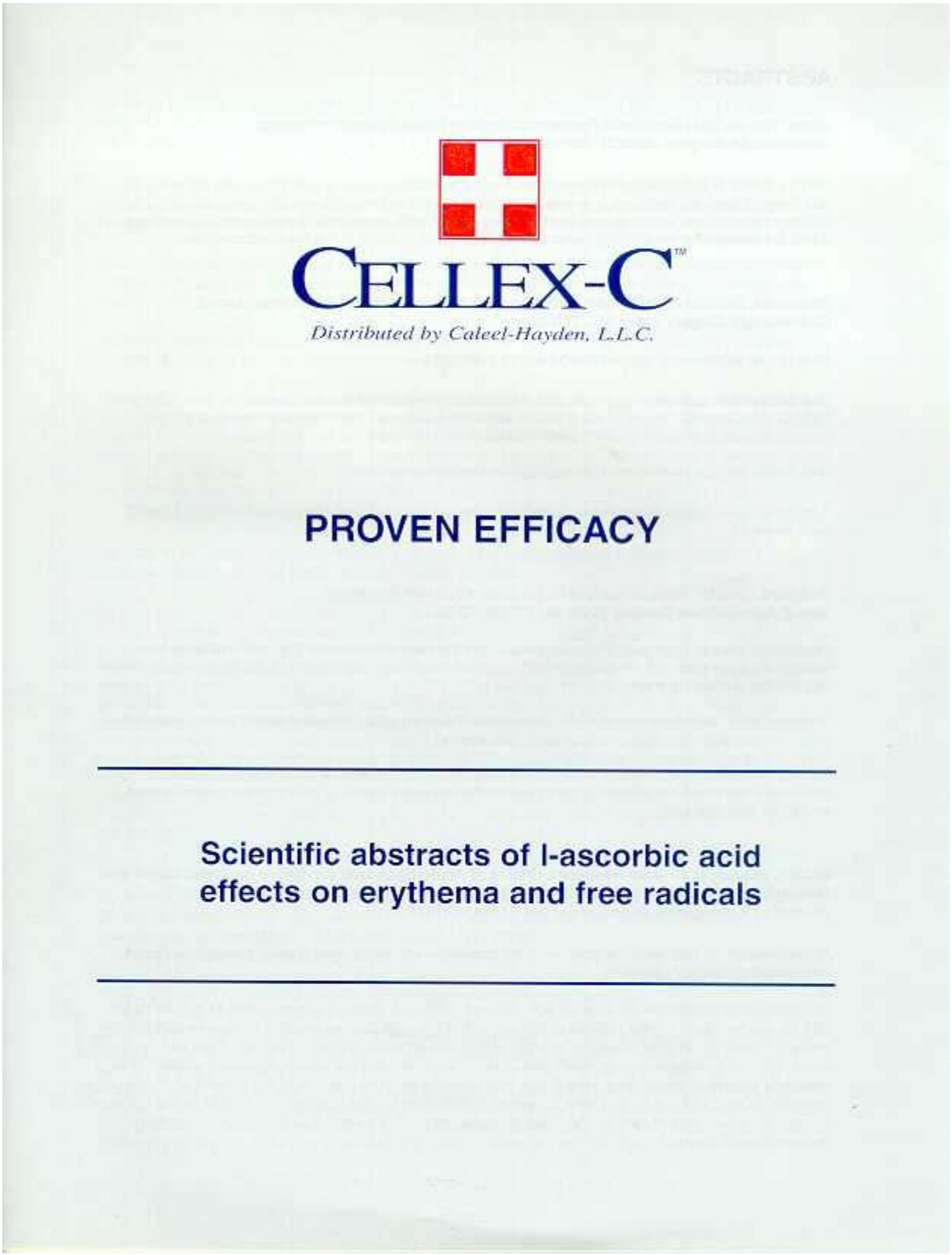
CELLEX-C™
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PROVEN EFFICACY
Scientific abstracts of l-ascorbic acid
effects on erythema and free radicals

ABSTRACTS

Alster, Tina S., MD. Table Talk: Common Questions about Laser Resurfacing. Dermatologic Surgery 1998; 24:121-130.

"The incidence of transient hyperpigmentation can be reduced by treating only those patients with pale skin tones. I have not observed a decrease in hyperpigmentation with the use of a preoperative regimen; however, the use of glycolic acid, retinoic acid or even ascorbic acid postoperatively (initiated within 3-4 weeks after resurfacing) reduces the severity and duration of the hyperpigmentation."

Fitzpatrick, Richard E., MD. Table Talk: Common Questions about Laser Resurfacing. Dermatologic Surgery 1998; 24: 121-130.

What is your preoperative regimen prior to laser resurfacing?

"The preoperative regimen includes: the use of Retin-A or Renova in all patients; the use of Cellex-C in all patients; the use of full-spectrum sunscreens such as Ti-Silc or Shade UVA Guard in all patients; the use of Melanex in patients with skin types III or darker."

How do you manage postinflammatory hyperpigmentation postoperatively?

"I prescribe full-spectrum sunscreen such as Ti-Silc, Retin-A 0.25% cream, Melanex solution and Cellex-C each morning."

Boschert, Sherry. Topical Product Helps Clear Postlaser Erythema. Skin & Allergy News October 1996; Vol. 17, No. 10:29.

The topical Vitamin C product Cellex-C reduced the duration of erythema after full face laser skin resurfacing by at least 1-2 weeks in 16 of 20 patients, according to preliminary results from one of the first blinded, controlled trials.

"It may be more like 2-4 weeks faster," Dr. Alster said. "It has the added benefit of getting people started on a basic skin care regimen, which has photoprotective effects."

Patients who could not tolerate Cellex-C were taken off the product for a week and then restarted. Those who still could not tolerate it have been shifted from the Cellex-C serum to the cream version, which has less free acid.

Shindo, Yasuko, et al: Dose-Response Effects of Acute Ultraviolet Irradiation on Antioxidants and Molecular Markers of Oxidation in Murine Epidermis and Dermis. Journal of Investigative Dermatology, Inc.; 1994: 470-475.

At low doses of UV light many components of the cutaneous antioxidant system were damaged and some were almost completely destroyed.

The free radical hypothesis for solar UV light-induced cutaneous damage can be stated as follows: 1) UV light causes formation of free radicals in skin cells; 2) if the dose is large enough, these free radicals can overwhelm skin antioxidant defenses; 3) when antioxidant defenses are overwhelmed, free radicals can cause damage to cellular protein, lipids and DNA; 4) such damage can cause pathology. Aspect 1) is extremely difficult to test in vivo; aspect 4) is well established. The purpose of the present study was to examine, in vivo, using light that very closely attached solar UV light, aspects 2) and 3) of this hypothesis, which predict that as UV doses increase, antioxidants should be destroyed and cellular damage should appear.

Ascorbate is known to scavenge hydroxyl and superoxide radicals (21), both of which have been shown to be produced in skin homogenates irradiated with UV light (22). Ascorbate is also known to recycle tocopheroxyl radical back to x-tocopherol (7,8) and this may also explain some of its rapid decline at low doses.

Shindo, Yasuko, et al:, Enzymic and Non-Enzymic Antioxidants in Epidermis and Dermis of Human Skin. The Society of Investigative Dermatology, Inc.; 1994: 122-124.

Thus the antioxidant capacity of the human epidermis is far greater than that of dermis. As the epidermis composes the outermost 10% of the skin and acts as the initial barrier to oxidant assault, it is perhaps not surprising that it has higher levels of antioxidants.

Studying antioxidant defense mechanisms of the skin can be of benefit in discovering protective procedures against skin cancers, cutaneous aging and skin inflammatory disorders.

The function and capacity of antioxidants in the epidermis and dermis are probably different. The target of photoaging is mainly collagen in the dermis, whereas UV-induced cancers such as melanoma, basal cell carcinoma and squamous cell carcinoma are derived from the epidermis. We previously reported that the activities of many antioxidants were higher in the epidermis than in the dermis in hairless mouse skin (11). In this study, we found that the capacity of antioxidants in the human skin was also higher in the epidermis than the dermis, and the difference was much greater in human skin than in murine skin.

Darr, D., et al: Topical Vitamin C Protects Porcine Skin from Ultraviolet Radiation-Induced Damage. British Journal of Dermatology; 1992: 247-253.

Summary: Ultraviolet radiation damage to the skin is due, in part, to the generation of reactive oxygen species. Vitamin C (l-ascorbic acid) functions as a biological co-factor and antioxidant due to its reducing properties. Topical application of Vitamin C has been shown to elevate significantly cutaneous levels of this vitamin in pigs, and this correlates with protection of the skin from UVB damage as measured by erythema and sunburn cell formation. This protection is biological and due to the reducing properties of the molecule. Further, we provide evidence that the Vitamin C levels of the skin can be severely depleted after UV irradiation, which would lower this organ's innate protective mechanism as well as leaving it at risk of impaired healing after photoinduced damage. In addition, Vitamin C protects porcine skin from UVA-mediated phototoxic reactions (PUVA) and therefore shows promise as a broad-spectrum photoprotectant.

Therefore, replenishment of skin Vitamin C would be an important pharmacological intervention against sun damage.

Nakamura, Toshiaki, et al. Vitamin C Abrogates the Deleterious Effects of UVB Radiation on Cutaneous Immunity by a Mechanism That Does Not Depend on TNF-x. The Society for Investigative Dermatology, Inc.; 1997:20-24.

Epicutaneous application of Vitamin C (10% l-ascorbic acid solution) abrogated the deleterious effects of acute low-dose UVR on induction of CH and prevented the induction of tolerance.

We have previously shown that human beings resemble inbred strains of laboratory mice in that some individuals are UVB-S, whereas others are UVB-resistant, and that UVB susceptibility may be a risk factor for the development of skin cancers (Yoshikawa et al, 1990). Because ingestion of Vitamin C has been shown to reduce the incidence of skin cancer induced in mice by UVR (Dunham et al, 1982) and our experiments show that epicutaneous treatment with Vitamin C prevents the deleterious effects of UVR on the cutaneous immune system of UVB-S mice, we propose that ROI's are involved in the pathogenesis of UVR-induced impairment of cutaneous immunity and UVR-induced skin cancers.

Ames, Bruce N., et al: Oxidants, Antioxidants and the Degenerative Diseases of Aging. Proceedings National Academy of Science, USA Vol. 90; Sept. 1993: 7915-7922.

Metabolism, like other aspects of life, involves tradeoffs. Oxidant by-products of normal metabolism cause extensive damage to DNA, protein and lipid. We argue that this damage (the same as that produced by radiation) is a major contributor to aging and to degenerative diseases of aging such as cancer, cardiovascular disease, immune-system decline, brain dysfunction and cataracts. Antioxidant defenses against this damage include ascorbate, tocopherol and carotenoids.

The amount recommended (Vitamin C) (e.g. 60mg/day for ascorbate) is primarily for avoiding an observable deficiency syndrome (e.g. scurvy) and is not necessarily the amount for optimum lifetime health, which is usually not known. (ii) A recommended blood level of each antioxidant (e.g. 80u l-ascorbate) would be a more desirable standard. People vary considerably in the intake required to keep their blood level adequate. A smoker, for example, needs to take in several times as much ascorbate as a nonsmoker to keep the blood level the same. Infections may also cause an oxidative stress that leads to antioxidant depletion by activating phagocytic cells. The observation that antioxidant inadequacy is associated with oxidative damage to DNA of the germ line, as well as somatic cells emphasizes the urgency of defining adequate blood levels (198).

Since only 9% of Americans, and fewer in most other countries, are eating five fruits and vegetables per day, there is a great opportunity to improve health by increasing consumption.

Rose, Richard and Bode, Ann. Biology of Free Radical Scavengers: An Evaluation of Ascorbate. The FASEB Journal, Sept. 1993; Vol. 7: 1135-1142.

At the heart of the body's nonenzymatic protective mechanisms is a scavenging reaction in which some endogenous compound with the inherent trait of entering the redox reactions contributes an electron to fill the outer shell of R' and thereby neutralize it to a nonreactive species. In principle, many chemicals could serve this purpose because the high reactivity of R' results in it extracting an electron from almost any available molecule. A few of the compounds shown to have this property are: mannitol (3) enkephalins (4) and tyrosine (9).

Pinnell, Sheldon R. Topical Vitamin C. Advanced Technology Conference Proceedings; 1997: 34-35.

A topical preparation containing 10% stabilized aqueous formulation of l-ascorbic acid, in addition to zinc, tyrosine and bioflavinoids has been developed for reversal of photoaging. Volunteers who used this preparation topically on one side of their face showed dramatic reversal of wrinkling on that side. Moreover, ultraviolet photography revealed dramatic reduction of aging pigments in skin as revealed by this technology.

Alster, T.S. et al: Effects of Cellex-C on post-CO2 Laser Resurfacing Erythema. Laser Surgery Medicine 1997; 9 (Suppl): 33.

Conclusions: The addition of Cellex-C to the postoperative regimen of patients undergoing CO2 laser resurfacing may decrease the degree and duration of post-treatment erythema.

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