SUN PROTECTION SHOULD PROTECT FROM IRA DAMAGE


Tan now…pay later. That sun-kissed smile comes at a price.

DEFENSE AGAINST SOLAR radiation is vital to human health but are current sunscreens up to the task? Sun care products that filter both UVA and UVB rays are insufficient since they do not block all the damaging rays from the sun. Recently, it became prudent to assess the effects of infrared radiation (IR) so that we can protect ourselves from it. Considering the facts brought to light about IR in the March column *(for a review of IR and key definitions, see p. 55,* HAPPI, *March 2012),* I will detail the biological effects and potential dangers of this one type of solar radiation. In future columns, I will review several proposed regimens for IR protection.

IRA rays (750-1,400nm) represent about one-third of total solar energy. They are capable of penetrating human skin and directly affecting cells located in the epidermis, dermis, and subcutis. This is in contrast to the IRC (3,000 nm–1mm) or the IRB (1,400–3,000nm), which are completely absorbed at the epidermis or only marginally affect the dermis. We are all familiar with the heat from these wavelengths; we can feel them. Radiation of these wavelengths is primarily responsible for increased skin temperature and, therefore, is experienced as ranging from pleasantly warm to burning hot sensations.[1]

IRA, similar to UVA or UVB, can cause skin damage and significantly contribute to the photoaging of human skin. Exposure of human skin fibroblasts in vitro[2] and human skin in vivo[3] to physiologically relevant doses of IRA causes an increase in MMP-1 (matrix metalloproteinase-1) without a change of its tissue inhibitor. Similarly, IRA exposure reduces type 1 collagen expression, possibly by reducing the production of procollagen-1–stimulating transforming growth factor-b1, -2, and -3 expression in human skin.[4] As a result, it was concluded that IRA causes the destruction of collagen fibers in human skin and, thereby, photoaging. It has therefore been proposed that efficient sun protection should include protection against IRA.[5]

Beside its effect on photoaging, IR is linked to photocarcinogenesis. Recent studies have shed light on the basic molecular processes, such as cellular signal transduction and gene expression triggered by exposure to IR radiation. This previously unrecognized molecular IR response shows that IR radiation is capable of interfering with cellular functions. This new information provides a molecular basis for biological effects of IR on human skin.[6]

The influence of IR radiation on biological processes in human skin has

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Dermatologists have harnessed the power of infrared light.

become obvious thanks to numerous studies during the past several years. Similar to UV, chronic exposure to IR appears to be involved in photoaging and photocarcinogenesis. IR may have beneficial effects on the skin, but there is no evidence showing if these benefits outweigh the potential damage. However, the factors that determine the specific biological outcome elicited by IR exposure remain mostly uncharacterized. The underlying molecular mechanisms activated by IR radiation have only begun to be identified.[6]

Premature Aging

Recent evidence indicates that IR and heat may induce premature skin aging, just like UV radiation including: (a) IR exposure of human skin stimulates the expression of MMP-1 and decreases type I procollagen expression in vivo. Acute IR irradiation also increases new, leaky vessel formation and induces inflammatory cellular infiltration and (b) Heat energy, which increases skin temperature, also increases MMP-1, -3, and -12, and modulates elastin and fibrillin synthesis, resulting in the development of solar elastosis. Acute heat shock in human skin stimulates new vessel formation, recruits inflammatory cells, and causes oxidative DNA damage.[7] Cho and associates stated that the number of mast cells in sun- exposed facial skin is always significantly higher than that of sun- protected buttock skin from the same individual. On the other hand, the effect of IR on dermal cell prevalence remains unclear. Tryptase expression (the most abundant granule-derived serine proteinase in mast cells used as a marker for mast cell activation) was also clearly up-regulated by IR treatment in human skin in vivo.[7] Based on these observations, it can be concluded that IR and heat are important physical stimuli that may result in aging in human skin. IR generates heat and increases skin temperature during sun exposure. Repeated and prolonged exposure to heat insufficient to produce a burn can cause a cutaneous lesion, described as *erythema ab igne,* which is characterized clinically by reticular hyperpigmentation and telangiectasia, and histologically by the basophilic degeneration of connective tissue and elastic fiber alterations, which are similar to those observed in photoaged skin.[7]

IR can produce wrinkles. Studies show that repetitive IRA irradiation produces significant wrinkle formation in hairless mice.[8] Chronic IR exposure can cause pronounced elastosis in mouse skin, which mimics UV damage. The effects of chronic IR exposure on skin aging were investigated, especially on the development of skin wrinkles, in a hairless mouse model. Kim and associates found chronic IR-induced skin wrinkles in hairless mice after 15 weeks of exposure and that IR-augmented, UV-induced wrinkle formation from 5 weeks post-UV/IR treatment. Although IR was less effective than UV at producing wrinkles, it was found in this study that chronic IR treatment can produce wrinkling in its own right, and augment wrinkle formation by UV.[7]

IR is different from UV and must be treated as such. IRA exposure led to down regulation of collagen de novo synthesis. According to Krutmann, *et. al.*, "The IRA-induced up regulation of MMP-1 was different from that induced by UV at the mechanistic level, since it involves the formation of mitochondrial reactive oxygen species (ROS) and the subsequent initiation of a retrograde signaling response (i.e. from the mitochondria to the nucleus) in human skin."[9] The presence of IRA, its biophysical properties, and the fact that it acts differently from UV, points to the necessity of including specific IRA-directed strategies in modern sunscreens.[1,9]

IR is not all bad. Infrared radiation is used very successfully in cosmetic dermatological procedures. Equipment used by dermatologists (i.e., Fraxel, Nd:Yag, Fotofacial, Smoothbeam) involves IR laser damage to the skin in a controlled manner to induce a wound healing response and affects no more than 5-10% of the skin. These lasers emit a precise wavelength with a variance of +/- 20nm targeting the water molecules in the skin that denature collagen and cause cell death, thereby inducing a wound healing response. It is evident that Infrared radiation has both dangers and benefits, but its attributes will be useless if its dangers are ignored.

Skin Can Be Protected

Skin damage from oxidative stress triggered by UV radiation, IR radiation, smoking, pollution or other environmental conditions increases MMPs in fibroblasts and keratinocytes. This causes a degradation of dermal matrix components (i.e. collagen), inferior repair of matrix damage, DNA mutations leading to wrinkle formation, photodamage and ultimately skin cancer. The use of antioxidants induces a powerful protective barrier, preserves skin's genetic inheritance and neutralizes free radicals. Readily available today are protocols that deliver efficient antioxidants directly to the skin.

The hallmark of an efficient skin antioxidant product is the use of scientifically proven, efficacious ingredients with a known mode of action that are present in sufficiently high active concentration with superior tolerance and proven penetration into the skin. Antioxidants such as L-ascorbic acid (vitamin C), tocopherols (vitamin E), ubiquinone (coenzyme Q10), glutathione, alpha lipoic acid, beta carotein, ferulic acid, oleuropein and others have been used in our industry.

A lifetime of sunshine can lead to face full of wrinkles.

In many cases, their concentrations are woefully low and improperly formulated such that the antioxidants do not penetrate the skin. Ascorbic acid for example, the gold standard among antioxidants, must be un-ionized to get into the skin. Its pH must be acidic (below 3.5) and in concentrations that are significant (above 10%) to be effective against the UV and IR damage.[10] Most formulations on the market do not fit these criteria for effective antioxidant protection.

Armed with this clinical evidence about the damaging effects of solar radiation, the use of a broad-spectrum sunscreen product with efficient UVB and photostabilized UVA filters is a user's first line of defense. Moreover, a powerful antioxidant regimen that adequately penetrates the skin delivered in reasonable effective concentration is highly recommended.

In the future, additional ingredients that provide a line of defense from both the UV and IR spectrum will be formulated in all standard sunscreen products. Designing new sun safety products that can address these new revelations from research will improve significantly the protection we currently provide to the consumer. ●

References

1. Krutmann J., Morita A., and Chung J., "Sun Exposure: What Molecular Photo Dermatology Tells Us about Its Good and Bad Sides" J. Invest. Dermatol. (2012) 132: 976–984.

2. Schieke S, Stege H, Kurten V , Grether-Beck S, Sies H and Krutmann J. (2002) "Infrared-A radiation-induced matrix metalloproteinase 1 expression is mediated through extracellular signal regulated kinase 1/2 activation in human dermal fibroblasts." J. Invest. Dermatol. 119:1323–9.

3. Schroeder P., Lademann J., Darvin M., Bruhnke S. and Krutmann J. (2008)" Infrared radiation-induced matrix metalloproteinase in human skin: implications for protection." J Invest Dermatol 128:2491–7.

4. Kim MS, Kim YK, Cho KH and Chung JH. (2006) "Regulation of type I procollagen and MMP-1 expression after single or repeated exposure to infrared radiation in human skin." Mech. Ageing Dev. 127:875–82

5. Schroeder P, Calles C, Benesova T, Macaluso F, and Krutmann J. (2010) "Photoprotection beyond ultraviolet radiation—effective sun protection has to include protection against infrared A radiation-induced skin damage." Skin Pharmacol. Physiol. 23:15–7

6. Schiek, S., Schroeder,P. and Krutmann, J., "Cutaneous effects of Infrared radiation", Photodermatol. Photoimmunol. Photomed. (2003), 19: 228-34.

7. Cho S, Shin MH, Kim YK, Seo JE, Lee YM, Park CH, Chung JH. ,"Effects of Infrared radiation and heat on human skin aging in vivo", J. Invest. Dermatol. Symposium Proceedings (2009), 14: 15-19.

8. Kim HH, Lee MJ, Lee SR, Kim KH, Cho KH, Eun HC and Chung JH. (2005) "Augmentation of UV-induced skin wrinkling by infrared irradiation in hairless mice." Mech. Ageing Dev. 126:1170–7.

9. P. Schroeder, C. Calles, Dipl.-Biol, J. Krutmann, "Prevention of Infrared-A Radiation Mediated Detrimental Effects in Human Skin", Skin Therapy Letter (2009).

Pinnel, S. et al., (2001) Dermatol. Surg., 27:137-142