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Infrared and skin: Friend or foe

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ABSTRACT

In the last decade, it has been proposed that the sun's IR-A wavelengths might be deleterious to human skin and that sunscreens, in addition to their desired effect to protect against UV-B and UV-A, should also protect against IR-A (and perhaps even visible light). Several studies showed that NIR may damage skin collagen content *via* an increase in MMP-1 activity in the same manner as is known for UVR. Unfortunately, the artificial NIR light sources used in such studies were not representative of the solar irradiance.

Yet, little has been said about the other side of the coin. This article will focus on key information suggesting that IR-A may be more beneficial than deleterious when the skin is exposed to the appropriate irradiance/dose of IR-A radiation similar to daily sun exposure received by people in real life.

IR-A might even precondition the skin – a process called photoprevention – from an evolutionary standpoint since exposure to early morning IR-A wavelengths in sunlight may ready the skin for the coming mid-day deleterious UVR.

Consequently IR-A appears to be the solution, not the problem. It does more good than bad for the skin. It is essentially a question of intensity and how we can learn from the sun.

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1. Introduction

The spectrum of solar radiation reaching the Earth ranges from 290 to more than 1,000,000 nm and is divided as follows: 6.8% UV, 38.9% visible, and 54.3% near infrared radiation (NIR) [1]. Infrared constitutes the waveband longer than 760 nm and up to 1 mm. It accounts for approximately 40% of the solar radiation reaching the ground at sea level. It has been divided into three bands: IR-A (760–1400 nm), IR-B (1400–3000 nm), and IR-C (3000 nm–1 mm) (Fig. 1). IR radiation can penetrate the epidermis, dermis, and subcutaneous tissue to differing extents depending on the exact wavelength range being studied. Exposure to IR is perceived as heat [2].

The strength of electromagnetic radiation depends on the energy of the individual particles or waves as well as the number of particles or waves present.

Electromagnetic radiation covers a spectrum with a wide range of photon energies that can also be expressed as a range of wavelengths. The spectrum has two major divisions:

- non-ionizing radiation
- ionizing radiation

Radiation that has insufficient energy to completely remove electrons from atoms and molecules is referred to as non-ionizing radiation. Examples of this kind of radiation are visible light, infrared, microwaves and radio waves. Radiation that falls within the ionizing radiation range has enough energy to remove tightly bound electrons from atoms, thus creating charged ions. This type of radiation includes X-rays and gamma rays.

Ultraviolet (UV) radiation is intermediate between these two broad ranges, and short-wavelength UV has enough energy to break chemical bonds and carry out photochemical reactions.

Although the consequences of sun exposure on the skin have been extensively studied over the years, the impact of IR radiation has received far less attention than its UV counterpart that is well known to cause skin cancer, photoaging, and immune suppression.

Moreover, the solar IR-A (also called NIR) irradiance level is critical to trigger beneficial effects in the skin beyond which it becomes deleterious.

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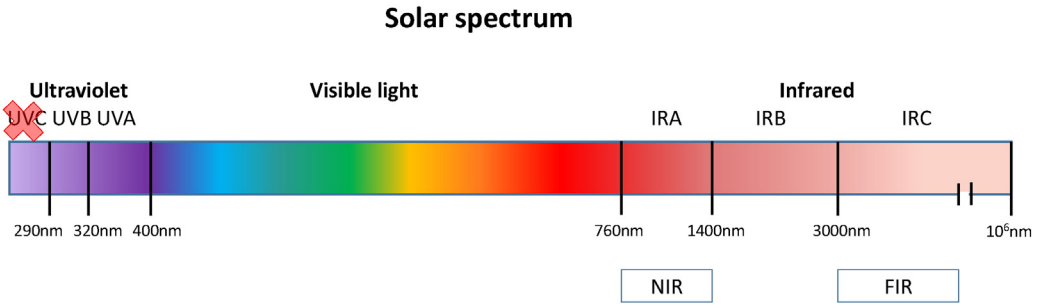

Fig. 1. Solar spectrum composition. Red X over UVC means that they are blocked by the ozone layer (NIR: near infrared, FIR: far infrared).

Most studies reporting the detrimental effects of IR-A (upregulation of matrix metalloproteinase 1 or MMP-1) used artificial light sources way above the solar IR-A irradiance threshold. This review article highlights the discrepancies in published data in order to bring a new perspective on this controversial topic.

2. NIR & Skin

2.1. NIR Detrimental Effects: Heat

It has been known for a long time in dermatology that chronic IR exposure can be deleterious to the skin. It was classically seen on the legs of those sitting too close to hearth fires named erythema *ab igne*. Such reticulated, erythematous or hyperpigmented dermatoses resulted from chronic and repeated exposure to relatively low levels of infrared radiation, and generally had a good prognosis. However, this was not necessarily a self-limiting diagnosis as patients were at long-term risk of developing subsequent cutaneous malignancies such as squamous cell and Merkel-cell carcinomas [3]. This diagnosis recently made a comeback with laptop-computer induced erythema *ab igne* [4] being described. Furthermore, severe skin aging may develop occasionally on bakers' arms because of exposure to hot ovens and on the faces of glass blowers [5]. In the above examples, the skin was exposed to massive heat *via* convection (hot air flow), conduction (direct contact) and/or radiation (IR). Although the proportion of heat transmitted by radiation is unknown, it can be estimated as far from negligible, at least for people sitting by fires or for bakers. Most importantly, the distinct effect of NIR was not measured independently from the heating effects by convection and/or conduction.

The thermal nature of erythema *ab igne* means that the irradiance of exposure was elevated and that the cumulative dose (fluence) was very high.

Is heat really an issue in causing the deleterious effects of NIR? Some studies have shown that there is an increase in collagen degradation and ROS generation with a relatively small increase in temperature. Piazena et al. studied the effects of water-filtered infrared-A (wIRA) with convective cooling or heating on viability, inflammation, inducible free radicals and antioxidant enzyme content in natural and viable skin [6]. The water-filtered IR-A, applied over 30 min to the skin at an irradiance of 190 mW/cm^2, with the skin temperature maintained at 37 °C by convective cooling from air ventilation, did not significantly affect the cell viability, the inflammatory status, the free radical content, or the antioxidant defense systems of the skin. This is of clinical relevance since the irradiance exceeded the maximum solar IR-A irradiance at the Earth's surface more than 5 times. Conversely, after convective heating to about 45 °C, free radical formation was almost doubled and antioxidant power was reduced to about 50%. This may be also linked to temperature-dependent polymer photodegradation showing a linear increase with radiation dose.

Even a relatively low irradiance of IR may lead to an intradermal temperature rise (inside-out heating). Other studies by Tanaka et al. reported that NIR can non-thermally induce cytocidal effects in cancer cells as a result of activation of the DNA damage response pathway [8,9]. They used a broadband NIR source (Titan; Cutera, Brisbane, CA, USA) emitting 1100 to 1800 nm, with water filtering to simulate solar NIR radiation. Even though no irradiance is mentioned, they irradiated cells with one to ten rounds of NIR at 20 J/cm^2 *in vitro* and up to 40 J/cm^2 *in vivo* without temperature monitoring in tissues. The use of a broadband NIR source (intense pulsed light (IPL) with a contact cooling tip at 20 °C to protect epidermal damage) is totally irrelevant since it is essentially a thermal technology built to destroy chromophores by raising dermal temperature with very high peak power pulses. Consequently, it does not simulate NIR rays from the sun and explains the cytocidal effects of this artificial light source that occur as a consequence of the heat generated.

We reported this finding *via* intra-dermal thermocouple type-T temperature measurements. We observed temperatures up to 44 °C with as little as 80 mW/cm^2 delivered in 15 min (72 J/cm^2), using a NIR LED light source at 970 nm (Fig. 2) [10].

Even a simple non-IR heating pad may lead to collagen degradation at 43 °C for 15 min [11]. In this experiment, dorsal skin of hairless mice was exposed to heat three times per week for a period of 6 weeks. They showed that chronic exposure of the skin to heat can cause skin wrinkling by increasing matrix metalloproteinase 13 (MMP-13) expression and decreasing antioxidant enzyme activity with consequent oxidative damage. MMP-13 promotes closure of skin wounds [12]. Another study by Halper et al. using chicken embryonic gastrocnemius tendon explants at different temperatures (37 °C *vs.* 43 °C) reported increases in mRNAs representing several collagen regulators, transforming growth factor beta (TGF-β), heat shock protein 47 (Hsp47) and connective tissue growth factor (CTGF) at 43 °C [13].

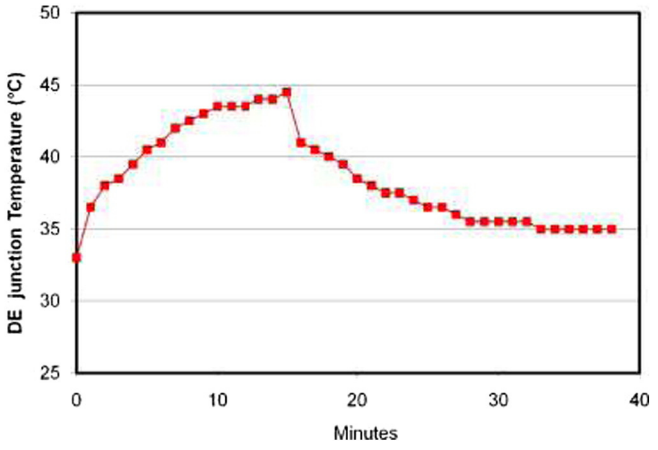

Fig. 2. Temperature increase with 970 nm light emitting diode at 80 mW/cm^2 was measured at the derma–epidermal (DE) junction as a function of time (minutes) for a patient. Data monitoring demonstrated that the temperature peaked at 45 °C after 15 min of irradiation and decreased slowly thereafter [10].

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