See our full scientific file (101 publications).

SOD B® & UV Protection

Life-styles have altered in recent decades, leading to increased personal exposure to UV. More than 80% of the visible changes commonly attributed to skin aging are caused by UV. Between 2 and 14% of people aged between 35-44 years old have already used tanning devices and UV booths. A repeated and prolonged UV exposure leads to an overproduction of Reactive Oxygen Species (ROS), which result in clinical signs of photoaging. Even if the body endogenous antioxidant defenses help to prevent these alterations, these defenses are rapidly overwhelmed following a prolonged UV exposure. Superoxide Dismutase (SOD) efficiency in the prevention of UV skin damages is supported by more than 100 publications.

UV: the largest environmental source of free radicals

UV Radiations (UV-R) initiate molecular responses in human skin through the generation of Reactive Oxygen Species (ROS)[1]. Among exogenous sources that can induce ROS production, UV is the most important factor for the skin by contributing to 80% of this production[2]. When overproduced and not neutralized, ROS can target lipid-rich membranes as well as cellular DNA and proteins, resulting in an array of toxic effects. Skin is very susceptible to such reactions. Therefore, a repeated and prolonged UV exposure leads to skin aging and other cutaneous alterations (Figure 1)[1].

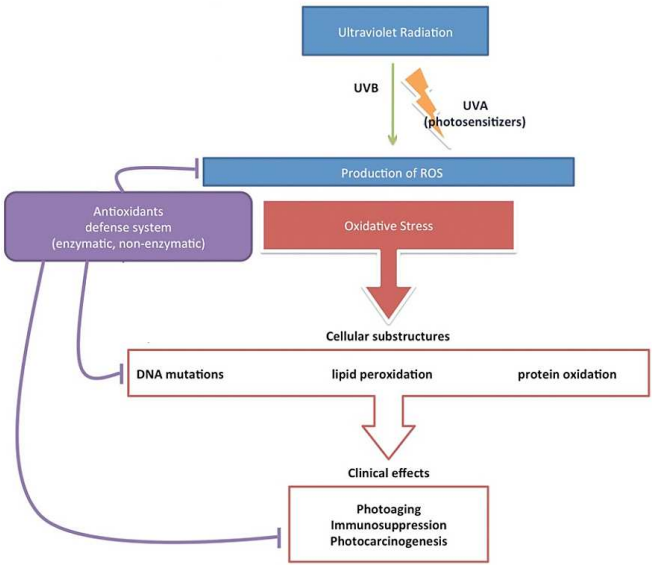

Figure 1: ROS induction by UV and skin alterations[1].

UV exposure alters antioxidant defenses

Cellular antioxidant defense mechanisms are crucial for the prevention of the UV-R oxidative damages. These homeostatic defenses, although highly effective, have limited capacity and can be overwhelmed. Under high sun exposure, UV-R lead to a massive development of ROS, and a decrease of the body antioxidant capacity (Figure 2)[3].

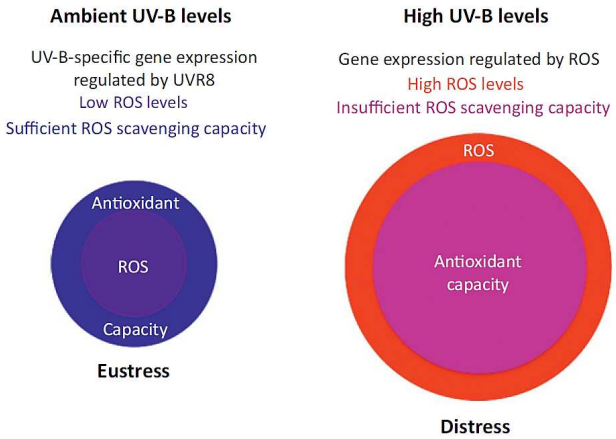

Figure 2: Antioxidant capacities under physiologically relevant UV levels and under high UV conditions[3].

Studies have shown that a single exposure to UV-R induces an impairment of the epidermal antioxidant defense system, including a decrease of the SOD, Catalase (CAT), and Glutathione Peroxidase (GPx) levels[4]. One approach to prevent the development of UV-induced cutaneous alterations is based on the restoration of endogenous antioxidant defenses[5].

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UV generates skin damages

By inducing ROS formation, UV-R activate several signaling pathways in the sub mucosa and epithelium. Nuclear Factor κB (NF-κB) is thought to be important as mediator of pro-inflammatory cytokines, implicated in various skin disorders such as erythema, edema and premature skin aging (Figure 3)[1].

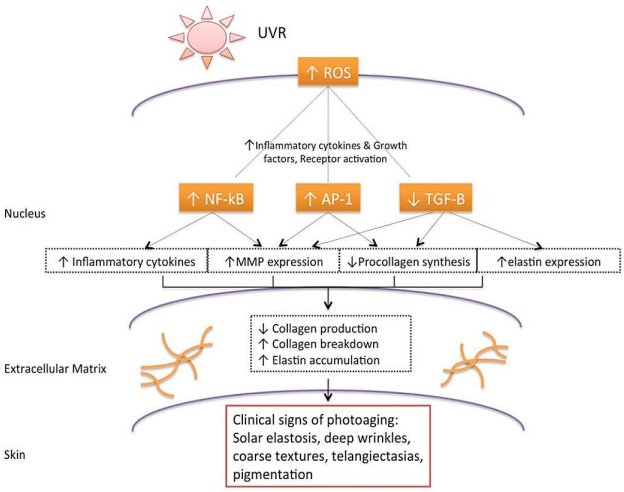

Figure 3: Role of ROS in UV skin damages[1].

SOD prevents UV-inflammatory reactions

Among ROS, superoxide anions ($O_2^{\bullet -}$) have pro-inflammatory properties by inducing the expression of NF-κB. Studies have showed that the anti-inflammatory effects of SOD are due to the inhibition of NF-κB expression[6]. Thanks to this specific mechanism of action, SOD prevents the expression of pro-inflammatory mediators (Figure 4).

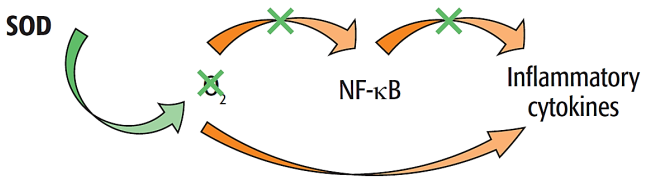

Figure 4: Anti-inflammatory action of SOD.

Clinical studies have highlighted the ability of exogenous SOD to stop the inflammatory response induced by UV-R on human skin[7]. Several reports have demonstrated that an SOD overexpression inhibits skin cells apoptosis induced by UV-B in an *in vitro* keratinocytes model[8, 9]. These results have been confirmed in an *ex vivo* model[9].

SOD corrects erythema

Erythema is the best known acute effect of excessive UV exposure. It is characterized by the familiar skin reddening appearance. Reports have showed that exogenous SOD is effective in inhibiting skin erythema[10]. The administration of SOD (1000 IU/ml) significantly suppressed erythema by 30 % *in vitro*[11]. SOD reduces the production of Prostaglandins-E2 (PGE2), which mediates the erythema response[12].

SOD reduces photodermatosis

An oral SOD supplementation has been clinically reported to be efficient against Polymorphic light eruption (PLE). PLE is the most common UV-induced dermatosis, accounting for 10-20% of cases in the U.S. and Europe. A significant reduction of PLE clinical manifestations has been observed in 70% of orally supplemented subjects.

SOD prevents immunosuppression

UV-R cause a dose-dependent inhibition of immune cells activity, such as Natural Killer (NK) cells[13]. The addition of SOD (100 and 1000 IU/ml) has been reported to reduce this suppressive effect. The efficiency of SOD in reducing immunosuppression has been confirmed *in vivo*[14].

Conclusion

Prolonged UV exposures are associated with skin oxidative damages and inflammation. As a powerful antioxidant and anti-inflammatory agent, SOD has been reported to:

- **inhibit skin cell apoptosis**
- **correct erythema**
- **reduce photodermatosis**
- **prevent immunosuppression**

Based on its unique way of action, SOD represents an efficient solution to prevent and correct UV-skin alterations.

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