# "IN VIVO" ESTIMATE OF ANTIOXIDANT ENZYMES AND LIPID PEROXIDATION AT DIFFERENT LEVELS OF HUMAN STRATUM CORNEUM

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The skin is an high metabolic refill tissue, constantly exposed to ROS sources as UV light, ozone and air pollution.
By using a non-invasive method like tape strippings (Corneofix®), we studied the effects of UV-A radiation acute dose (15 J/cm$^2$) on superoxide dismutase (SOD) and catalase (CAT) activities and lipid peroxidation (LPO) at different levels (1$^{st}$,5$^{th}$,10$^{th}$) of human stratum corneum (SC). Furthermore we valued the protective effects of vitamin E (RRR-α-tocopherol) administration (Sursum Abiogen Pharma, 400 UI/die for 3 days) on these enzymatic activities and its role against the photo-induced lipid damages.
SOD and CAT show an activity gradient across the SC, lower in the outer stratum and superior in the inner one.
After vitamin E administration the enzymes activities decrease in all the SC strata; after the second vitamin E dose the SOD and CAT activities are generally halved.
After UV-A irradiation, CAT activity decreases within SC strata. The activity decrease is more evident in the 10$^{th}$ stratum, where it is also time-dependent (until 4 hours after irradiation).
SOD activity doesn't seem to be affected by photo-induced stress in the inner strata, while it's very low in the 1$^{st}$ stratum after UV-A irradiation.
The effects of UV-A irradiation arrange with the effects of vitamin E supplementation. The result is a large decrease of enzymes activities in all SC strata; 4 hours after UV-A exposure, a partial recovery of CAT activity is in evidence in the 10$^{th}$ stratum, while no recovery is evident for SOD activity.
The decrease of the enzymes activities are probably due to a strong control of antioxidant systems by cells, that down-regulate the enzymatic defences for exploiting the non enzymatic ones, when these are in large bioavailability. Offering an important antioxidant cover, vitamin E supplementation leads the antioxidant enzymes to "stand-by", as a strategy adopted by cells to save resources and energy.
Vitamin E administration in the normal skin shows strongly reduced LPO in SC, while the LPO undergo a progressive increase after UV-A exposure, particularly in the outer strata. The vitamin E supplementation before UV-A exposure shows LPO levels that are more contained than LPO levels after the only irradiation. Vitamin E has, at last, an extremely protective role against the UV-A mediated lipid damage.
So, it is possible to contend that strategies of nutritional and/or dermocosmetic supplementations, as RRR-α-tocopherol, are a valuable new approach for preventing and containing the skin UV-A mediated damages.