

Potential protective effects of melatonin against UV-A irradiation on fibroblast cell line

Giovanni Damiani, Michele Peroni, Antonia Infranco and [Francesca Bonomini](#)

Human Anatomy Division, Department of Biomedical Science and Biotechnology, University of Brescia, Brescia, Italy

The sun's radiation that reaches Earth contains ultraviolet (UV) wavelengths made up of a combination of UV-A (95%) and UV-B (5%) radiations. Chronic sun exposure is responsible for long term clinical skin changes such as photoaging, photodamage and photocancers. Moreover, inflammation is mostly due to UV-A which stimulates the production of reactive oxygen species (ROS) inducing also photoaging (Mouchet et al., 2010; Marionnet et al., 2010). In order to protect themselves against oxidative stress, skin cells developed several defense systems, including ROS and metal ions scavengers and a battery of detoxifying and repair enzymes (Bickers and Athar, 2006). In addition, UV-A can also directly influence the structure of nucleic acids, breaking the chain or changing the nucleotide sequence. Altogether these perturbations of cells homeostasis advantages a significant up-regulation of oxidative and inflammatory responsive genes. In this study, we focused our attention on prevention of photodamage, choosing melatonin as antioxidant agent. Melatonin is a neuroendocrine mediator with pleiotropic bioactivities such as hormonal, neurotransmitter, immunomodulator and biological modifier actions. Its antioxidant activity is the result of two different but synergic actions: a direct, due to its ability to act as a free radical scavenger and an indirect due to the up-regulation of antioxidant enzymes (Fischer et al., 2008). The aim of the present study was to analyze the impact of pre-treatment of murine fibroblasts cells (NIH-3T3) with melatonin (10⁻³ M- kindly provided by Chronolife S.r.l., Roma, Italy) later irradiated by UV-A irradiation (15 J/cm²) evaluating the changes of fibroblast microenvironment conditions. We observed that UV-A irradiation caused matrix restructuring and alteration, oxidative stress and inflammation; while melatonin pre-treatment suppresses UV-A induced photodamage. Collectively, these results suggest that melatonin provides relevant protective effects against UV-A irradiation. A new chapter of melatonin in dermato-endocrine research could be open.

References

- Mouchet et al. (2010) In vivo identification of Solar radiation-responsive gene network: role of the p38 stress-dependent kinase. *PLoS ONE* 5: e10776.
- Marionnet et al. (2010) Different Oxidative Stress Response in Keratinocytes and Fibroblasts of Reconstructed Skin Exposed to Non Extreme Daily-Ultraviolet Radiation. *PLoS ONE* 5: e12059.
- Bickers and Athar (2006) Oxidative stress in the pathogenesis of skin disease. *J Invest Dermatol* 126: 2565-2575.
- Fischer et al. (2008). Melatonin as a major skin protectant: from free radical scavenging to DNA damage repair. *Exp Dermatol* 17: 713-730.

Keywords: Fibroblast, melatonin, oxidative stress, UV-A irradiation.