ABSTRACT BOOK ABSTRACTS



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PIGMENTATION

PYCNOGENOL® INDUCTION OF MELANIN, TYROSINASE, ENDOTHELIN-1, AND PPAR PRODUCTION BY MELANOCYTES: AN IN-VITRO STUDY.

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Background: Botanical extracts, such as Pycnogenol®, have been studied for its bleaching action due to its antioxidant, anti-inflammatory, and supposedly-granted antimelanogenic activity. In addition, many efforts have been made to elucidate its possible etiopathogenic mechanisms involved in hyperpigmentation processes, such as the production of endothelin-1 and increased tyrosinase activity, which consequently lead to melanin production. Further, the genes involved in peroxisome proliferator-activating receptor signaling cascade (PPAR) seems to be downregulated.

Objective: Evaluating the Pycnogenol® activity on human melanocyte cultures by measuring synthesis of melanin, tyrosinase, endothelin-1, and PPAR, under the action of ultraviolet radiation A (UVA) and B (UVB), infrared-A radiation (IRA), and visible light (VL).

Materials and Methods: Human melanocytes were seeded and incubated with a dry extract solution of Pinus pinaster (Pycnogenol®), after determination of the non-cytotoxic concentrations, and exposed to UVA/UVB, IRA, and VL, as well as the association of the three radiations. Subsequently, quantification of melanin concentration, tyrosinase activity, and endothelin-1 and PPAR mediators were performed.

Results: Pycnogenol® promoted reduction in melanin synthesis up to 7.66, 5.14, and 4.05% when compared to UVA/UVB, IV-A and association radiation, respectively. In relation to the activity of the enzyme tyrosinase, Pycnogenol® achieved approximately 66.5% reduction in enzymatic activity and showed reductions in the synthesis of endothelin-1 by up to 56.47, 59.33, 58.00, and 73.03% when compared to UVA/UVB, IV-A, LV, and association radiation, respectively. The synthesis of PPAR was reduced up to 38.41, 26.39, 19.51, and 56.44% when compared to UVA/UVB, IV-A, LV, and association, respectively.

Conclusions: Pycnogenol® reduces production of melanic pigmentation by downregulating tyrosinase, endothelin-1, and PPAR synthesis.





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