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Commentary

A Great Insight into Ultraviolet Phototherapy

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DESCRIPTION

Photobiology is a wide term that encompasses all biological processes that include non-ionizing radiation. Non-ionizing radiation causes chemical and/or physical changes in biological systems, which are known as photobiological reactions. UVR is one of the most serious environmental risks acting on the skin among the radiation effects. Chronic UVR exposure has been shown to speed up skin ageing, cause immunosuppression, and increase the risk of skin cancer. UVR, on the other hand, has been demonstrated to be useful in the treatment of a variety of skin illnesses, leading to the development of several phototherapy methods. Narrow Band Ultraviolet-B (NB-UVB), which fluoresces with a peak about 311 nm and is currently one of the most widely used phototherapy devices, was shown to be beneficial in the treatment of a variety of skin problems. Despite the fact that NB-UVB was developed over 30 years ago, the exact mechanism of its therapeutic activity is still unknown. To recently, most of NB-effects UVB's have been attributed to its action on immune cells; nevertheless, over 90% of NB-UVB irradiation is absorbed by the epidermis, and keratinocytes appear to play a key role in mediating NB-UVB biological activity. In this paper, we talk about the latest research on the effects of NB-UVB on epidermal cells, which are skin cells, with a focus on cell growth and death.

Phototherapy has primarily been an experiment. As a result, despite its long-term use, its methods of action remain a mystery. Researchers are now beginning to comprehend some of the pathways thanks to recent advances in photoimmunology and molecular photobiology. Many of the effects are almost definitely mediated by apoptotic cell death induction. Immunosuppression induction is another important method. UV radiation causes a temporary reduction in total DNA, RNA, and protein synthesis in both animals and humans. However, a rebound rise in synthesis and a proliferative state follow this occurrence. Furthermore, it is unclear whether psoriatic keratinocytes are more vulnerable to UVB radiation due to their metabolic activity and rapid replication, or whether UVB radiation reduces macromolecular production in all psoriatic cells. However, it is unknown whether UVB radiation selectively inhibits or kills highly proliferating cells. The fact that normal skin is hyper proliferative when exposed to erythemogenic amounts of UVB radiation, resulting in epidermal thickening, refutes the notion that UVB therapy helps psoriasis primarily by inhibiting cell proliferation.

When the skin is exposed to UVB light, DNA photo lesions formed, which can lead to cell death, mutations, and the initiation of carcinogenic processes. UVB activates specific proteins, which then start signal transduction pathways that result in biological reactions. The alteration of these signaling molecules is thought to be a key event in UVB-induced tumor promotion. DNA damage-inducible genes have been found, such as RhoB, which encodes a small GTPase. RhoB has a role in the trafficking of EGF receptors, cytoskeletal structure, cell transformation, and cell survival. When activated GTP-bound form of RhoB is increased rapidly within 5 minutes of UVB exposure, and then RhoB protein levels increased concomitantly with Epidermal Growth Factor Receptor (EGFR) activation, the regulation of RhoB was elucidated and its role in the cellular response of HaCaT keratinocytes to relevant environmental UVB irradiation was elucidated. RhoB protein expression and AKT phosphorylation are prevented by inhibiting UVB-induced EGFR activation, but not early RhoB activation. Through suppression of EGFR expression, blocking UVB-induced RhoB expression with particular small interfering RNAs lowers AKT and glycogen synthase kinase-3 phosphorylation. Furthermore, RhoB down regulation enhances UVB-induced cell death. RhoB overexpression, protects keratinocytes depending on the balance between pro and anti-apoptotic pathways. It was discovered that RhoB is regulated by a two-step process including an early EGFR-independent RhoB activation followed by an EGFR-dependent elevation of RhoB expression in response to UVB exposure. Furthermore, RhoB is required for keratinocyte cell survival after UVB exposure, implying a possible role in photocarcinogenesis.

CONCLUSION

The number of experimental investigations attempting to understand the cellular and molecular biological changes generated by UV radiation has increased exponentially during the last decade. The mechanisms underpinning UV-induced

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skin damage, such as sunburn, immunosuppression, photoaging, and photocarcinogenesis, are the common skin damages which are found from scientific investigations. Furthermore, it is unknown whether each UV responsive disease has an ideal or specific wavelength range. Empirical methods will be used to answer some of these problems. For a better knowledge of how UV radiation operates as a therapeutic agent, more experimental research explaining the processes underlying the biologic activities of UV radiation is required. As a result, these investigations will undoubtedly help to the creation of more effective regimens and, most likely, more targeted phototherapies.