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Phosphorylase kinase inhibition in skin disease

The central molecule in the injury pathway is the transcription activator nuclear factor-kappa B (NF-kB) activated 30 min after injury. NF-kB is responsible for the transcription of over 200 genes related to multiple processes, including inflammation and scarring. Phosphorylase kinase released 5 mins after injury activates downstream NF-kB-dependent processes, such as TGF-β1-dependent fibroblastic and myofibroblastic proliferation responsible for scarring after injury. Curcumin, a phosphorylase kinase inhibitor, blocks downstream NF-kB-dependent inflammation and scarring, with minimal scarring following burns, trauma and surgical wounds. Because of ultraviolet light, damaged skin will make injury to the DNA, particularly double stranded DNA breaks (DSBs), leads to increased risk of photo-carcinogenesis. Phosphorylase kinase also phosphorylates a family of phosphatidylinositol-3 kinases (ATR, ATM and DNA-PK), which control the entry to the DNA Damage Repair Pathway i.e., Cell Cycle Arrest, Nuclear Excision and DNA replication. The repair processes are slow and often incomplete, resulting in photo-aging and photo-carcinogenesis. By blocking phosphorylase kinase, Curcumin induces Curcumin-induced apoptosis, allowing not only for the rapid removal of the severely damaged cells, but also creates the space for replacement by new, healthy undamaged cells. This results in rapid healing of burns and sun-burns. In addition, the removal of premalignant cells leads to healing of damaged skin with a decreased tendency for malignant transformation. Psoriasis, a genetic skin disease precipitated by injury (trauma, contact allergy and infection), is associated with elevated levels of phosphorylase kinase, believed to result from a defective genetic-based switch-off mechanism. The elevated phosphorylase kinase is associated with increased PCNA+ (proliferating cell nuclear antigen) resulting in psoriasiform proliferation. Curcumin, by inhibiting phosphorylase kinase, causes apoptosis of the PCNA+ cells, returning the skin to normal. We present the results of a protocol based on suppression of phosphorylase kinase activity with topical Curcumin, topical steroids, avoidance of precipitating factors (contact allergens), treatment of bacterial, fungal and viral infections, and maintenance of a strict lactose free diet

Speaker Biography

Madalene C Y Heng is a Professor of Medicine/Dermatology, David Geffen UCLA School of Medicine. After 25 years in full-time academia, she is currently in private practice as a dermatologist in Camarillo, California. She is the author of over 85 publications, in peer-reviewed journals. She is a reviewer of multiple journals with Editorial positions in others. Her expertise includes an interest in the biochemistry and pathophysiology of disease including acne, wound healing and psoriasis. She is the Inventor of Curcumin gel.

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