Anti-psoriatic action of lutein demonstrated by inhibition of rat photodermatitis

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Rat ultraviolet ray B (UV-B) photodermatitis has been used as an experimental model of *Psoriasis vulgaris*, to test for the anti-inflammatory activity of the xanthophyl, lutein, isolated from *Galium aparine* using the bioassay guided technique inhibition of chemical erythema. The aerial parts of *G.aparine* have been used in western herbalism to relieve the symptoms of psoriasis and as a hair rinse for scaling scalp problems.

UV-B induced dermatitis is characterised by demarcated brownish red lesions, intraepidermal accumulation of polymorphonuclear leukocytes with micro-abscess and hyperproliferation of the epidermal cells, Nakaguma,H., Kambasa,T., and Yamamoto,T., (1995). These are all characteristic of *Psoriasis vulgaris* in man, a common and complex disease distinguished by thickening of the skin and rapid proliferation of the keratinocytes.

Lutein was isolated from the chloroform extract of Gaparine by using bioassay-guided fractionation, using the technique of inhibition of chemically induced erythema on the mouse ear, Evans, F.J. & Schmidt, J. (1979). The chloroform extract was chromatographed on a silica gel column (eluting with chloroform and methanol). Lutein was isolated further using a sephadex gel column and purified using preparatory thin laver chromatography. Lutein was shown to have an ED50(dose required to produce a 50% inhibition to erythema) of 5µg after the application of the irritant, 12-O-tetradecanylphorbol-13-acetate.

Physically induced erythema, the induction of erythema by UV-B light, was carried out on two rectangular areas on the dorsal skin of male wistar rats (180g) The total UV-B exposure was 2.4J/cm. Lutein was applied to the test areas at a dose of 100μ g/day for five days. On the fifth day histological post-mortem sections were taken and stained using haematoxylin and eosin. The number

of epidermal cell layers and the epidermal thickness was measured at random. Figure 1.



The average number of cells was increased from 1.82 in normal skin to 3.54 in UV-B irradiated skin, a 97% increase (figure 1.) With the application of lutein a reduction by 52% could be found in the epidermal cell layers exposed to UV-B light, however this result, as with the UV-B exposure only, was still significant from the control (p=0.0017 UV-B and 0.0185 UV-B + lutein, figure 1). Although a slight increase in the number of epidermal cells could be found with the application of just lutein , it was not significant from the control.

It has been shown that UV-B and TPA both share a number of biological effects, stimulation of arachidonic acid release, prostoglandin E_2 production and cellular proliferation in cultured cells, although UV-B does not stimulate protein kinase C like TPA, Matsui,M. & DeLeo,V., (1990). It may be suggested that lutein might be acting on the accumulation of arachidonic acid, as it is known that abnormally high levels of arachidonic acid are found in psoriatic lesions, which is then metabolised into the generation of proinflammatory lipoxygenase products.

Evans,F.J.& Schmidt, J. (1979) Infl.,3:215-223 Nakaguma,H.,Kambasa,T.,&Yamamoto,T.(1995) Int.J.Exp.Path,76:65-73 Matsui,M. & DeLeo,V. (1990) Carcinogenesis, 11:229-230