

AGEING OF THE SKIN

Summary of a Stanford Study

Intrinsic (chronological) ageing (20%) and extrinsic (environmental) ageing (80%) in the skin consists of:



1. Epidermis:

Thickened stratum corneum dead cell layer, thinner living epidermal layer (higher cell turn-over rate causes cells to shed off before maturation)

2. Dermis:

Reduced collagen formation, increased collagen breakdown, loss of dermal hydration, loss of elasticity, uneven distribution of melanocyte cells, small broken veins due to chronic inflammation, reduced oil secretion.





Stratum corneum Granular cell layer

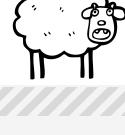
Spinous

Basal laver

Thin Stratum

Young Skin

- Corneum Thick epidermal layer
- Well defined
- epidermal-dermal junction Normal Collagen &
- Elastin



Thick Stratum

Old Skin

- Corneum Thin epidermal layer
- Poorly defined
- epidermal-dermal junction Broken down
- Collagen & Elastin

messenger proteins control the genes associated with inflammation, ageing and disease. Activation of the NFkB proteins happens through UV-radiation, pollution and chemical contact to the skin and old age. Previously there was no way of controlling the activation of the genes.

The NF-kB family of

are blocked Effects of genes associated with

- inflammation, ageing and disease are cancelled
- No inflammation No disease activation Reversal of ageing = old skin reverts to young skin (the effect is reversible)

• Smoother skin, more

tolerant skin, less wrinkles, better hydration, more even skin tone. No more DNA repairprotein present (thus no

more DNA-faults that

happen during cell-

division)

 All skin cells start to function normally (collagen-formation, melanin formation, elasticity)

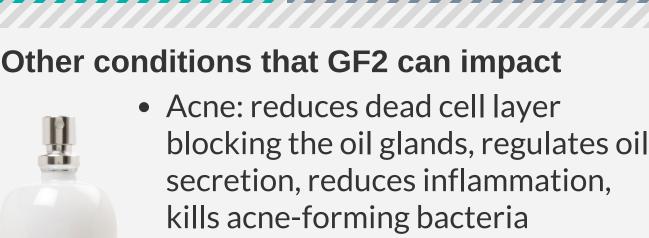
NF-kB not blocked

Effects of activated

no HOCI

- genes not cancelled Inflammation
- Disease present (sun damage keratosis, slow healing of wounds, skin
- cancer) Continuous ageing = rough dry skin, wrinkling, uneven tone, intolerant to irritation, chronic inflammation,
- sunspots Continuous presence of DNA repair-protein means that there are continuous faults developing in the
- No change in skin architecture, other than continued ageing

replication of DNA



- kills acne-forming bacteria Pigmentation disorders: normalises melanocyte function, allows for wash-out of excess pigment
- inflammation control Eczema: positive control of inflammation and itchiness in a large

Rosacea: strong infection and

Keratosis or sun spots

percentage of cases

Skin redness

Thoclor Labs GF2 HOCL Skin Rejuvenation NF-kB activated genes

THOCLOR **GF**² LABS Skin Series products

Investigation. Vol. 123, No.12 Dec 2013.

References:

1. Role of the NF-kB Pathway in the Pathogenesis of Human Disease States. Yamamoto, Y. Current Molecular Medicine, Volume 1, Number 3, 1 July 2001, pp. 287-296(10)

2. Research article: Leung H. et al. Topical hypochlorite ameliorates NF-kB mediated skin disease in mice. Department of Developmental Biology, Dept. of Dermatology and Radiation Oncology. Stanford University School of Medicine. Journal of Clinical