SYNERGIE SKIN
CLEAN SCIENCE

CLINICAL DATA – ACETYL HEXPEPTIDE-51 AMIDE

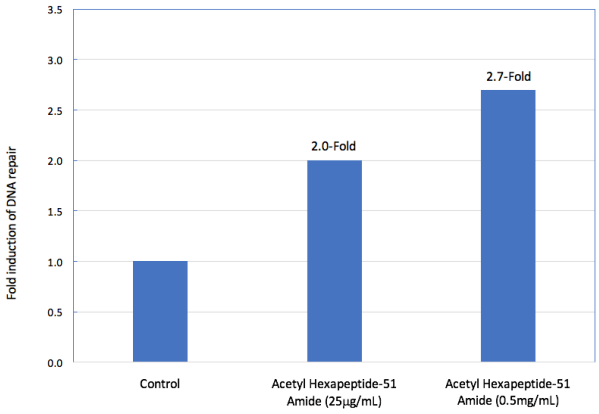
Description
Hexapeptide that mimics the activity of FOXO3a (member of the Forkhead box transcription factors), which is involved in cell repair, renewal and longevity. Acetyl Hexapeptide-51 Amide protects DNA from damage, stimulates its natural repair pathways and reverts senescence in fibroblasts.

Properties
Acetyl Hexapeptide-51 Amide maintains genomic integrity by protecting and repairing DNA damage induced by several agents, and delays cellular senescence to ensure longer and healthier ageing.

Science
The maintenance of DNA integrity is essential for the proper functioning and survival of organisms. DNA strands suffer from the constant challenge of endogenous and exogenous genotoxic agents as well as replication errors. UV-induced lesions may result in the formation of cyclobutane pyrimidine dimers (CPDs), a well-known form of pyrimidine dimers. Such damage can distort the structure of the DNA, altering its transcription and replication. Checkpoint pathways monitor DNA structure and control cell-cycle arrest allowing for repair and continue progression if everything is correct, before a mistake can be propagated to daughter cells.

FOXO3a acts as a master regulator that determines cell fate once damage is detected. Depending on the severity of the damage, this key transcription factor forces a state of quiescence in cells signalling for repair of errors or apoptosis. The active form of FOXO3a increases the level of well-known antioxidants, induces the expression of genes involved in DNA-damage repair and triggers cell death when necessary. Acetyl Hexapeptide-51 Amide imitates the activity of FOXO3a, increasing the expression of several genes involved in DNA repair pathways. It also provides protection to cells and rejuvenates fibroblasts, retarding their senescence.

***In vitro* efficacy**
1. Activation of DNA-Repair Pathways
The efficacy of Acetyl Hexapeptide-51 Amide in inducing DNA-repair pathway through the FOXO3a repair pathways was analysed through a host cell reactivation assay in primary human epidermal keratinocytes transfected with a UVC-damaged plasmid.

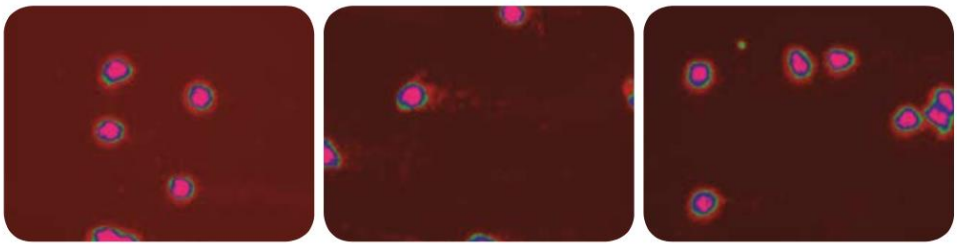


Acetyl Hexapeptide-51 Amide activates natural DNA repair pathways involving FOXO3a.

2. DNA-Protective effect against BaP

The protective effect that Acetyl Hexapeptide-51 Amide provides against photo activated benzo[a] pyrene (BaP) with UVA/visible light was evaluated by the comet assay in human normal fibroblasts, keratinocytes, and melanocytes.

FIBROBLASTS

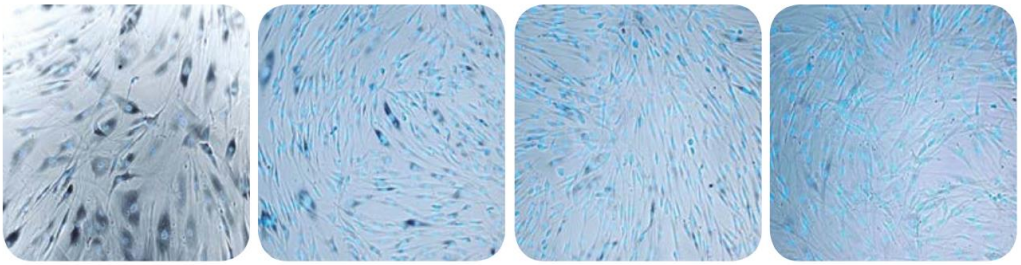


Control | BaP (40µM) | Acetyl Hexapeptide-51 Amide (10µg/mL) + BaP (40µM)

All three types of human skin cells were protected by Acetyl Hexapeptide-51 Amide against BaP. A statistically significant DNA protective effect of 84.3%, 99.1% and 90.8% was obtained for fibroblasts keratinocytes and melanocytes respectively.

3. Reverting Cellular Senescence

The senescence-associated β-galactosidase activity is one of the most commonly used biomarkers of cell ageing. The efficacy of Acetyl Hexapeptide-51 Amide was evaluated by the decrease in the number of senescent cells using a histochemical staining kit in primary human dermal fibroblasts (HDFa) from a 55-year-old donor.



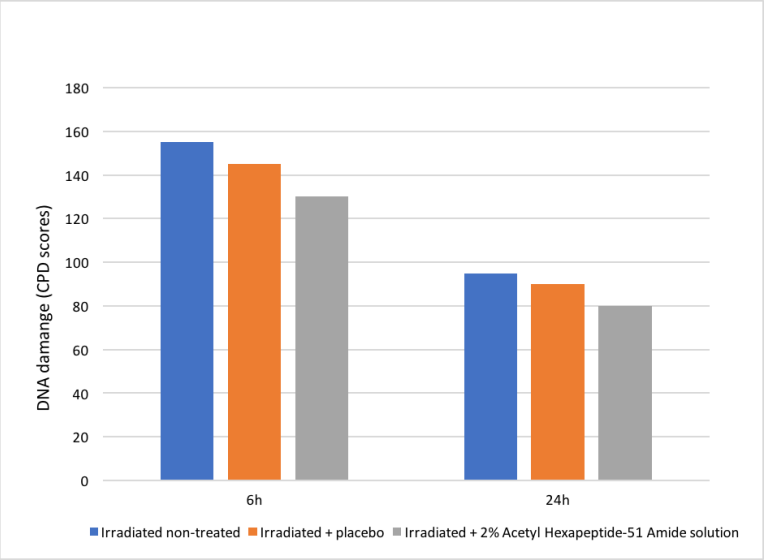
67-year-old HDFa | 55-year-old HDFa | 55-year-old HDFa + Acetyl Hexapeptide-51 Amide (0.01mg/mL) | 37-year-old HDFa

Cellular senescence is reverted with Acetyl Hexapeptide-51 Amide. The morphology of fibroblasts was recovered to that of 10 years prior.

***In vivo* efficacy**

Repair of UV-Induced DNA Damage

Four test areas, two on the inner site of each forearm, were designated on 21 volunteers. Three of the test areas were irradiated with 2 MED UV-light. Then, two of these irradiated zones were treated with either a cream containing 2% Acetyl Hexapeptide-51 Amide solution or a placebo cream. The third irradiated area and the fourth non-irradiated area were used as controls. A suction blister biopsy of each of the four areas was collected to analyse CPD presence at 6 hours after irradiation, and only three suction blisters for the irradiated zones were collected after 24 hours.



Acetyl Hexapeptide-51 Amide diminishes UV-mediated DNA damage. A statistically significant reduction of pyrimidine dimers was induced by Acetyl Hexapeptide-51 Amide, while placebo did not.