DMK
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DMK BIOGEN C & AGEING

Ageing is driven by diverse molecular pathways and biochemical events that are promoted by both genetic and environmental factors. Specifically, ageing is defined as a time-dependent decline of stress resistance and functional capacity, these effects relate to (among others) age-related gradual accumulation of damaged biomolecules (including proteins), which eventually compromise cellular homodynamic as they result in the failure of most cellular maintenance pathways. Considering that most (if not all) of the critical cellular functions depend on the functionality of highly sophisticated protein machines, it is not surprising that proteostasis (proteome homodynamic) regulation is critical for cellular function.

To maintain proteostasis cells have developed a modular, yet integrated system which ensures general proteome quality control, and it is called the Proteostasis Network (PN) The PN curates the basal functionality of the proteome and it also responds to conditions of proteotoxic stress by addressing the triage decision of fold, hold, or degrade. PN is constituted of several complex protein machines that ensure normal proteome synthesis and recycling or respond to conditions of proteotoxic stress by launching the proteome damage responses (PDR), which firstly identify and then either rescue or degrade unfolded, misfolded or non-native polypeptides. Loss of proteostasis is a common feature of ageing and age-related diseases and is characterised by the appearance of non-native proteins or protein aggregates in various tissues. Likely, this relates to the fact that independently of the triggering event, senescent cells are characterised by accumulation of oxidative stress, reduced proteasome activity and high rates of genome/proteome instability.

Biogen C contains Hexapeptide-11 and the below research outlines how this peptide protects fibroblast cells, prevents cellular senescence, regulates MMP's creates proteostasis, which is the homeostasis of proteins.

Exposure of fibroblasts to Hexapeptide-11 promoted activation of proteasome, autophagy, chaperones, and antioxidant responses related genes. Moreover, it promoted increased nuclear accumulation of Nrf2; higher expression levels of proteasomal protein subunits and increased proteasome peptidase activities. In line with these findings, Hexapeptide-11 conferred significant protection in fibroblasts against oxidative-stress-mediated premature cellular senescence, while in vivo skin deformation assays in human subjects it improved skin elasticity. Finally, Hexapeptide-11 was found to induce the activity of extracellular MMPs, and it also suppressed cell migration. Hexapeptide-11 has been found to influence several genes that functionally relate to the Extracellular Matrix function. The presented findings indicate that Hexapeptide-11 is a promising anti-ageing agent.

Reference:
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4434199/