Skin ageing

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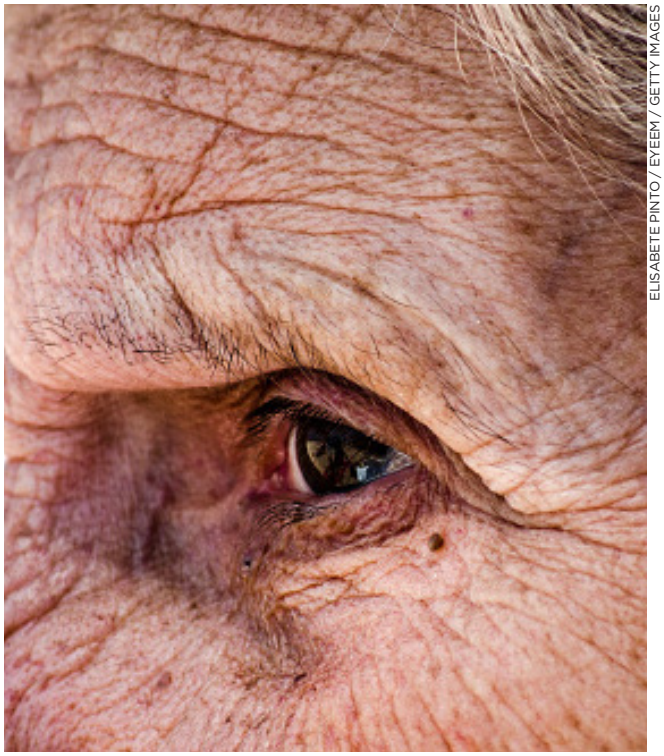
Dr Paul Charlson discusses the intrinsic and extrinsic factors involved in skin ageing and the treatments used in aesthetic medicine



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Sun exposure accounts for about 80% of extrinsic skin ageing

The skin ages due to intrinsic and extrinsic factors. Intrinsic ageing is an inevitable physiological process leading to dry, thin skin with fine wrinkles. Extrinsic ageing is caused by a variety of factors such as smoking, sun exposure and poor nutrition, leading to deep wrinkling, reduced elasticity, and thickened skin with a rough texture. Sun exposure is the main factor for extrinsic ageing, accounting for probably about 80% of the effect.

In intrinsic ageing, reduced proliferation of the basal cell layer results in a thinner epidermis and decreased contact area between dermis and epidermis. What is termed `cellular senescence' occurs and there is reduced proliferation of keratinocytes, melanocytes and dermal fibroblasts. Skin oligosaccharides also degenerate, reducing the ability of the skin to retain water.

In extrinsic ageing the stratum corneum thickens, due to corneocyte desmosome degradation failure. In the dermis, there is reduced production of type VII collagen, producing wrinkles because of a diminished epidermo-dermal connection. Type I collagen is also diminished. Abnormal elastic tissue accumulates in the deep dermis in a process known as solar elastosis. Many researchers consider most skin ageing is due to extrinsic factors, possibly as much as 97%.

THEORIES AND MODELS OF SKIN AGEING

At a molecular level there are a range of theories and models to describe skin ageing.

DNA damage

There is good evidence that persistent UV exposure leads to DNA damage and mutation. Although some repair can be effected by the nucleotide excision repair pathway, this does not compensate for the damage occurring. Sunscreens can prevent this damage and may even be more effective if DNA repair enzymes are added.

Chronic low grade skin inflammation

This is a major factor in skin ageing. It is thought that UV damage causes oxidative stress in the epidermal cells, resulting in cell damage and oxidised lipids. The oxidised lipids are recognised by the complement system, causing inflammation. Repeated UV exposure causes chronic inflammation and overburdened macrophages, leading to permanent damage to the epidermo-dermal junction.

Oxidative stress

This is important in both intrinsic and extrinsic skin ageing. It is an imbalance between reactive oxidative species (ROS) such as free radicals and peroxides which are produced in the cells, and the cells' ability to detoxify ROS and repair the damage caused by them.

Telomere shortening

Telomere are nucleotides that prevent the ends of chromosomes being damaged. These shorten over time and cell senescence occurs. Telomerase adds nucleotides to the ends of chromosomes preventing cell ageing. ROS leads to telomere damage.

Advanced glycate end (ACE) product accumulation

This is a non-enzymatic process in which proteins and lipids in the skin are bound to sugars. This results in a stiffening and reduced elasticity in tissues. It is quite common in the dermal matrix and accumulates with age. UV light-exposed skin exhibits greater glycation.

TREATMENTS

Treatments unsurprisingly are aimed first at preventing UV damage by the use of sun blocks, then neutralising ROS by using antioxidants such as vitamin C and E.

Other treatments include retinoids which prevent collagen degradation, activating dermal fibroblasts and thickening the epidermis whilst compacting the stratum corneum.

One of the issues is that skin ageing effects are frequently at dermal level and it is difficult to ensure that topical agents reach the target site.

Treatments such as microneedling, peels and heat-based technologies may allow for greater penetration to the target area. Agents such as synthetic peptide mixture matrixyl and human copper-binding peptide GHK-Cu are attracting particular interest as additional anti-ageing agents.

Other areas of interest are hormone replacement therapy, which thickens the epidermis, increases collagen and elasticity and improves hydration, and diets that recommend steamed or boiled low-sugar foods to reduce protein glycation.

Dr Paul Charlson is a GPwER in dermatology and president of the British College of Aesthetic Medicine