

# Editorial

## Skin aging

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Aging is a gradual process which has to be faced by every individual. Although each and every body part ages with time, yet the aging of facial skin appears to be a matter of major concern, the reason is that the skin plays a major part in our social and sexual interaction. Skin aging has not much to do with physiological function, which may remain adequate in old age. Facial skin is particularly effective in continuing communication. So skin and hair has to look, feel and smell attractive.<sup>1</sup>

As the aging population of the world is increasing the dermatological problems of the old people are on the rise. Intrinsic structural changes are genetically determined and occur as the skin ages. It is considered to be a natural phenomenon. Aging process varies among different populations and also the rate is different at different anatomical sites in the same person. Environmental and personal factors also influence the intrinsic rate of skin aging especially the amount of exposure to ultraviolet light.

The risk of cutaneous malignancies also increases with photodamage. Therefore, it is important to understand the influence of both intrinsic and extrinsic factors on skin aging and also to differentiate the retractable aspects of cutaneous aging i.e. primarily hormonal and

lifestyle influences, from the irretractable aspect i.e. primarily intrinsic aging.<sup>2</sup>

The pH of the skin surface increases with age, increasing its susceptibility to infections. Neurosensory perception of superficial pain is diminished both in intensity and speed of perception, increasing the risk of thermal injury. Deep tissue pain, however, may be enhanced.

A decline in lipid content as the skin ages inhibits the permeability to nonlipophilic components, reducing the efficacy of topical medications. Allergic and irritant reactions are decreased, as is the inflammatory response, compromising the ability of the aged skin to affect wound repair.

The ultraviolet radiations have several direct and indirect effects on the skin. Most of the effects are caused by direct cellular injury i.e. DNA damage through free radical formation. The ultraviolet radiation activates the nuclear factor- $\kappa$ B (NF- $\kappa$ B) which leads to elevated levels of postinflammatory cytokines IL-1 and IL-6, vascular endothelial growth factor (VEGF) and tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ). These factors attract neutrophils which increase oxidative damage through formation of free radicals.<sup>3</sup>

Increased VEGF reduces thromboplastin-1 and increases platelet-derived endothelial cell growth factor. These changes in gene expression may contribute to telangiectasia and photocarcinogenesis. The ultraviolet radiations activate epidermal growth factor, IL-1 and TNF-

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$\alpha$  receptors within 15 minutes of sun exposure. These factors ultimately activate nuclear transcription factor activator protein-1 (AP-1). AP-1 regulates transcription of metalloproteinases (MMPs). This enzyme is responsible for degradation of the extracellular matrix and collagen.<sup>4</sup>

Gravitational force causes the activation of phospholipase A2, an enzyme involved in the generation of prostaglandins and leukotrienes. These can lead to cytokines release, neutrophil recruitment and free radical generation.<sup>5</sup>

Electromagnetic radiations from computers and mobile phones induce histamine, interleukin-1 and 6 release. In response to neurogenic inflammation the cutaneous neuropeptides regulate the expression of cell surface adhesion molecules on immune cells and endothelial cells, contributing to skin aging.<sup>6</sup>

Cigarette smoking causes elastic fiber thinning and fragmentation contributing to aging process.<sup>7</sup>

Aging occurs at all levels of skin. Cells become larger in stratum corneum and there is prolongation in corneum replacement time. This leads to dry, itchy and flaky skin. There is flattening and thinning of dermis and dermoepidermal junction. The melanocytes develop large vacuoles in them thus causing greying of hair. The activity of Langerhan's cells and immune response is reduced even in photoprotected areas of the skin. The delayed hypersensitivity response is reduced, but B and T cells remain normal. There is also slow repair and wound healing. Dermis becomes thin with age and it loses elastin and collagen. Vascular walls become thick and rigid. Fibroblasts and mast cells become fewer. Nerve endings become

abnormal causing decreased sensory perception but there is no effect on pain or itch perception.<sup>8</sup>

Permeability to chemicals increase so there is exaggerated response to chemicals, also there is more risk of malignant transformation.

There is alteration in activity of sebaceous, apocrine and eccrine glands thus causing reduction in sebum production. There is regression of axillary apocrine glands and reduction in sweating and odour. Nails also become brittle with age and this is due to reduction of lipophilic sterols and free fatty acids. The linear growth of nails is also reduced.<sup>9</sup>

Skin aging is a complex biological process that is a consequence of both intrinsic or genetically programmed aging that occur with time, and extrinsic aging caused by environmental factors .

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