

Skin Changes in the Elderly

Geriatric Essentials

- The overall result of age-related structural changes is an increase in skin dryness, roughness, wrinkling, and laxity, and a decrease in skin elasticity.
 - The overall result of age-related functional changes is a decline in skin barrier function, mechanical protection, sensory perception, wound healing, immunologic responsiveness, thermoregulation, and vitamin D production.
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Aging leads to many changes in the skin, hair, and nails. These changes can be broadly categorized as either age-related or photoaging. Age-related changes are presumed to be due to age alone, whereas photoaging is due to chronic exposure to ultraviolet (UV) radiation superimposed on aging itself. Popular notions of “old skin” often correspond more closely to photoaging than to aging itself, and dramatic differences between aged skin protected from UV light and younger unprotected skin are evident to patients and clinicians alike. Other factors that affect the skin include smoking, which accelerates wrinkle development, and disease, most notably connective tissue disorders.

The overall result of structural changes is an increase in skin dryness, roughness, wrinkling, and laxity, and a decrease in skin elasticity. The overall result of functional changes is a decline in skin barrier function, mechanical protection, sensory perception, wound healing, immunologic responsiveness, thermoregulation, and vitamin D production. Aging may also affect the absorption of some topical drugs, although clinically important differences have not been identified.

Epidermis: The epidermis gives rise to the outer barrier layer of dead cells, the stratum corneum, through terminal differentiation of keratinocytes, the predominant cell type. The epidermis recognizes invading pathogens and other foreign substances and generates abundant cytokines. Melanocytes reside in the epidermal basal layer, producing and distributing photoprotective melanin to the keratinocytes.

With aging, the dermal-epidermal junction flattens—the number of interdigitations dramatically decreases—resulting in a smaller contact surface area between the dermis and epidermis. As a result, dermal-epidermal separation occurs more readily in elderly skin, and elderly skin is more likely to tear or blister. The change probably also compromises communication and nutrient transfer between epidermis and dermis, affecting the mechanical, barrier, and immunologic functions of the epidermis.

Elderly skin often appears dry and flaky, especially over the lower extremities, at least partly due to a dramatic age-associated decrease in epidermal filaggrin, a protein required for the binding of keratin filaments into microfibrils.

Epidermal turnover rates decrease by about 30 to 50% between a person’s 20s and 70s. This decrease slows the replacement rate of the stratum corneum, likely resulting in a rougher skin surface and a less adequate barrier. Slow replacement of the surface layer is also thought to be responsible for the prolonged healing times for epidermal wounds as well as the decreased barrier function that results from slow replacement of neutral lipids. The number of active melanocytes decreases by about 10 to 20% per decade, probably explaining in part the increased vulnerability to ultraviolet (UV) radiation in old age. An accompanying age-associated decline in DNA repair capacity compounds the loss of melanin protection and increases the risk for developing skin cancers. The prevalence of melanocytic nevi also declines, from a peak between ages 20 and 40 to near zero after age 70.

Vitamin D production, which depends on sun exposure, declines with aging, possibly because of a 75% decrease between early and late adulthood in the amount of epidermal 7-dehydrocholesterol, the immediate biosynthetic precursor of vitamin D. Decreased vitamin D production is often compounded by reduced outdoor activity, leading to insufficient sun exposure.

Dermis: The dermis contains the blood vessels, lymphatics, nerves, and deeper portions of the hair follicles and glands that arise from the epidermis. It is composed largely of extracellular matrix and gives skin its strength and elasticity.

Dermal thickness decreases by about 20% in the elderly and often even more in photodamaged areas. UV damage produces hyperplastic changes initially, followed by atrophic changes, particularly in fair-skinned people. These opposing changes probably explain observed variations in the effects of photodamage.

Even when elderly skin has been consistently protected against the sun, within the dermis there is about a 50% decrease in mast cells and a 30% decrease in venular cross-sectional area. Basal and peak levels of cutaneous blood flow are reduced by about 60%. As a result of these decreases, there is a decrease in release of histamine (a mast cell product) and other measures of inflammatory response after exposure to UV radiation or immune challenge. Vascular responsiveness during injury or infection is also compromised. The striking involution of vertical capillary loops in dermal papillae is thought to account for the pallor, decreased temperature, and impaired thermoregulation found in elderly skin. As well, the decline in vascular supply to hair bulbs and to the eccrine, apocrine, and sebaceous glands may contribute to their senescence.

Reduced synthesis and increased degradation of collagen, the major component of the dermal matrix, probably contribute to impaired wound healing in the elderly. Elastic fibers decrease in number and diameter with aging, accounting for decreased elasticity in elderly skin. Fragmentation, progressive cross-linkage, and calcification of elastic fibers also occur. Alterations of mucopolysaccharides that normally bind water in the dermal matrix may affect skin turgor.

Subcutaneous fat: Subcutaneous fat acts as a shock absorber, protecting the body from trauma, and plays a role in thermoregulation by limiting conductive heat loss. The overall volume of subcutaneous fat usually diminishes with aging. Distribution changes as well; eg, there is a relative decrease in subcutaneous fat on the face and hands but a relative increase on the thighs and abdomen. These changes can alter the appearance of the face and hands and reduce the pressure diffusion over bony areas that prevents some pressure ulcers and fractures.

Hair: Hair substantially grays in about 50% of people by age 50, apparently due to loss of melanocytes. Although the degree of hair graying often runs in families, the responsible genes are unknown.

Linear growth rate decreases with aging because the follicular keratinocytes that normally differentiate to form the hair shaft proliferate more slowly. Hair loss (more correctly, conversion from terminal to vellus hairs) in the vertex and frontotemporal regions (androgenetic alopecia) in men begins between the late teens and the late 20s; by the time they reach their 60s, 80% of men are substantially bald. In women, the same pattern of hair loss may occur after menopause, although it is rarely pronounced. Hair thinning, or diffuse hair loss sometimes termed female alopecia, is more correctly termed miniaturization of hairs. The cause is a shortened anagen (growth) phase and decreased proliferation of follicular keratinocytes. Diffuse hair loss normally occurs in both sexes with aging and should be distinguished from diffuse hair loss caused by iron deficiency, hypothyroidism, chronic renal failure, under-nutrition, and use of certain drugs (especially anabolic steroids and anti-metabolites).

Excessive or unwanted hair growth becomes common after menopause in women as a result of altered estrogen-androgen balance in hormonally sensitive hair follicles. The most distressing symptom may be the appearance of scattered terminal hairs in the beard area. Men may notice excessive hair growth in the eyebrows, nares, or ears.

Nails: Linear growth rate and thickness ("strength") of nails decreases with aging because of a decrease in the proliferative rate of nail matrix keratinocytes, which differentiate to form the nail plate. Nails become dry and brittle and flat or concave instead of convex, often with longitudinal ridging. Longitudinal pigment banding, common among blacks, often becomes more pronounced with aging. Nail color may vary from yellow to gray, reflecting changes in the nail bed. The lunulae can become poorly defined. Occasionally, the nails become grossly thickened and distorted.

Lamellar dystrophy manifests as brittle nails with split ends or layering and commonly occurs in elderly people, though it may also occur in middle-aged women.

Nerves and glands: The density of cutaneous sensory end organs decreases progressively between the ages of 10 and 90 by about 1/3. The result is an age-related reduction in sensations of light touch, vibration, corneal sensitivity, 2-point discrimination, and spatial acuity. The cutaneous pain threshold increases by about 20%.

Eccrine glands decline in number by an average of 15% during adulthood. Decreased gland secretion results in marked decreases in spontaneous sweating in response to dry heat. These changes, compounded by decreased cutaneous vascularity, make the elderly more vulnerable to heat. Apocrine glands also decrease in size and function with aging, but these changes do not appear to have any clinically significant effect (except possibly a decline in body odor).

The size and number of sebaceous glands do not appear to decrease with aging. However, sebum production decreases by about 23% per decade, beginning in early adulthood, probably due to the concomitant decrease in production of gonadal or adrenal androgens, to which sebaceous glands are exquisitely sensitive.

Immunologic function: The number of epidermal Langerhans' cells (immune cells in skin responsible for antigen presentation) decreases by 20 to 50% during adulthood. Alterations in the production of ILs and cytokines by other cells such as keratinocytes may also contribute to overall immunologic decline observed in the elderly. The result is presumed to be increased susceptibility to infections and increased incidence of neoplasms.