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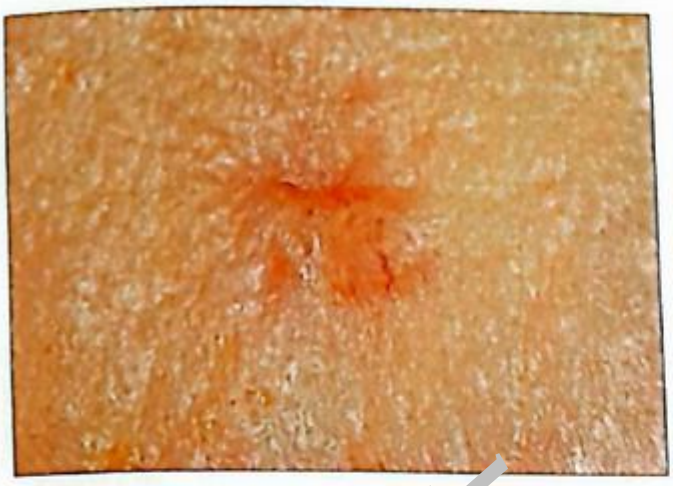


Fig. 1.44 Basal cell carcinoma is the most common form of non-melanoma skin cancer in humans. Among ardent sunbathers, this localized tumor with its destructive growth can sometimes be found even at a young age.

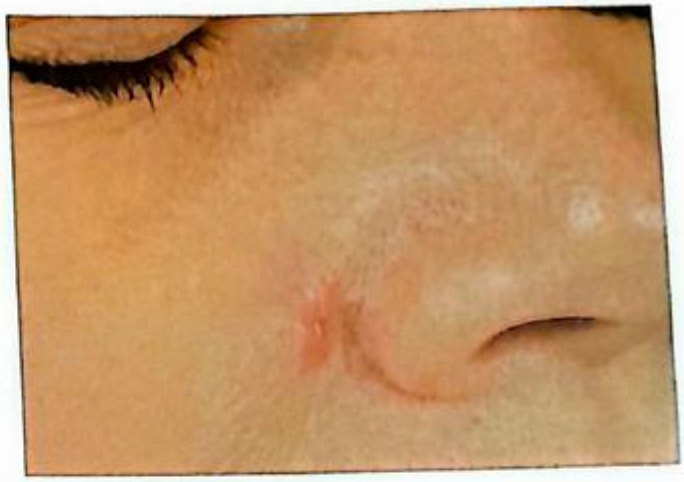


Fig. 1.45 Squamous cell carcinoma is the second most common non-melanoma skin cancer in humans. It often develops on skin showing prior actinic damage.

Effects on the dermis

The extracellular matrix, made up of collagenous and elastic fibers, assures the structural integrity of the dermis (Weinstein et al., 1990). The UV-triggered aging of the connective tissue leads to non-elastic, lax skin via the induction of MMP (Fisher et al., 1996). Elastic fibers decrease in number and density, with amorphous masses of elastolytic material found as the principal sign of exogenous, UV-triggered skin aging. The shortening, thinning and decrease in number of the elastic fibers is a significant determinant of wrinkle depth (Lee et al., 2008). In addition, water is increasingly found in unbound form (Pulido et al., 2003), since glycosaminoglycans are bound into elastotic material (Bernstein et al., 1996). In the histological section, this is characterized by the appearance of the typical blue color in the polygonal polymorphic fibers, which are no longer clearly identifiable as a network. Directly below the basal membrane is a junction zone consisting of a narrow band of dense collagen fibrils arranged parallel to the surface, as an expression of microscarring (Lober et al., 1990). The classical histological methods do not show up the changes in the elastic fibers, even though homogenization is clearly visible (see Fig. 1.46). Special stains reveal the thickened, coarse elastic fibers, which have aggregated into clumps (see Fig. 1.47 and Fig. 1.48). Perivascular infiltrates are also found around the frequently dilated vessels, as an expression of chronic inflammation (see Fig. 1.49). This impaired vascular function manifests in the form of red blood cell extravasation.

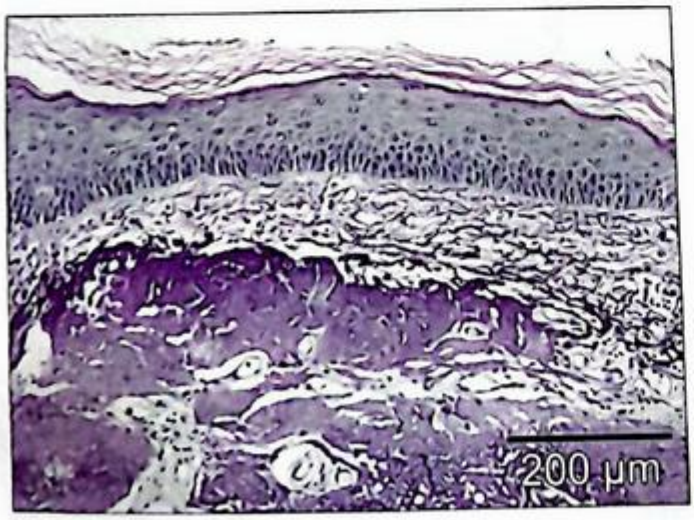


Fig. 1.46 The loss of the elastic fiber network becomes visible with elastic staining (resorcinol-fuchsin/Orcein: elastic fibers appear purple to black). Whereas fibers are still recognizable as such immediately below the epidermis, the connective tissue in the middle layers appears completely homogenized.

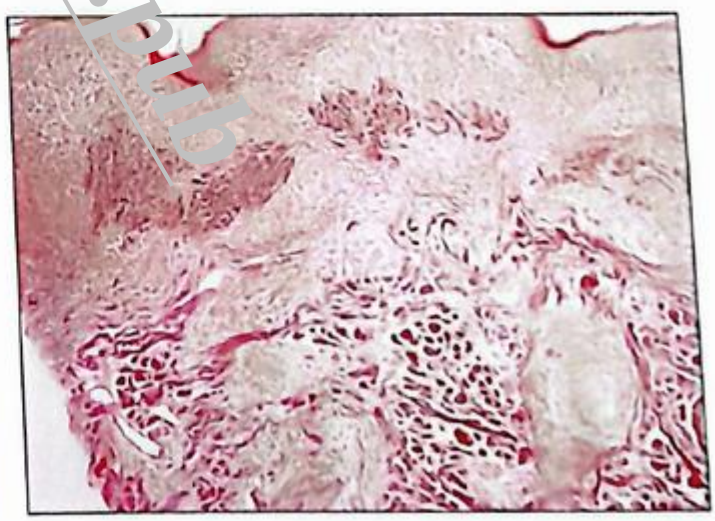


Fig. 1.47 Elastica-van Gieson staining shows up structures known as elastolytic granulomas as the extreme variant of elastic fiber destruction.

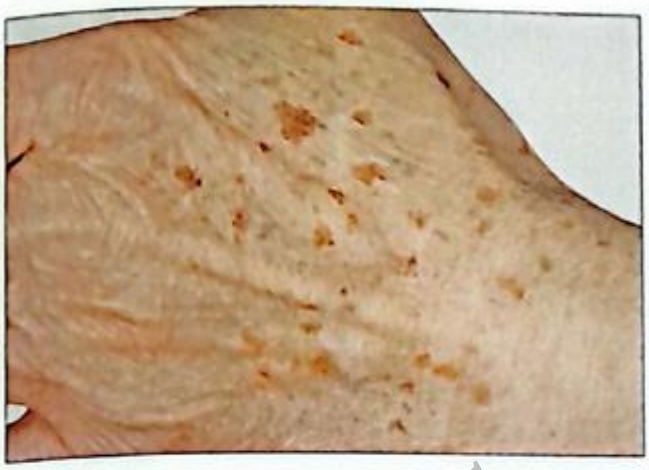


Fig. 1.30 Areas of unsightly pigmentation are found especially on the hands. Lentigo senilis needs to be clearly distinguished from malignant neoplastic growths.

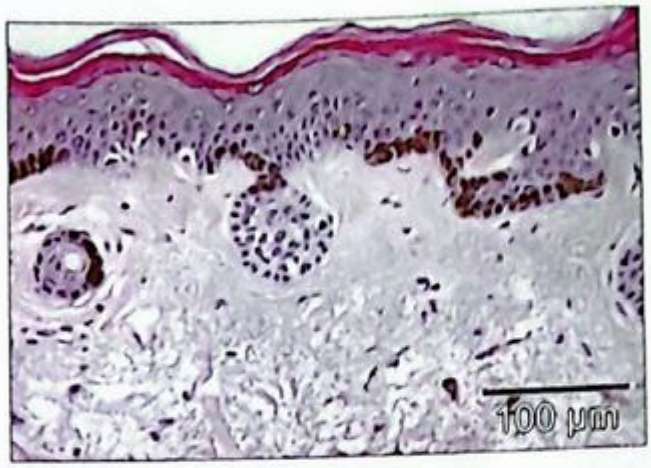


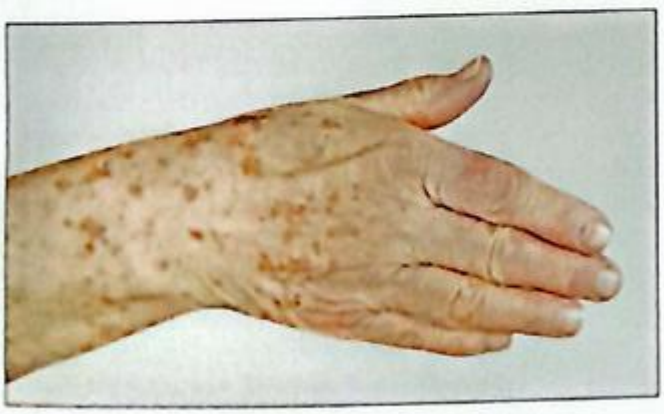
Fig. 1.31 In the histological section, pigment deposits become visible as hyperpigmented and elongated rete ridges. Note the clear appearance of the melanocytes.

and the symbiotic dendritic cells. Microscopically detectable sunburn cells appear 8–12 hours after the effects of UV light. Since aging leads not only to increased skin damage, but also to a decrease in the skin's ability to repair itself, mutations begin to accumulate.

UV-A light (320–400 nm) is regarded as being a thousand times less harmful, but still contributes appreciably towards skin wrinkling. It penetrates the epidermis and upper layers of the dermis with damaging effects. Even long-wave infrared light (720–1000 nm) still has an aging effect on the skin (Schroeder et al., 2008; Kligman, 1989). To summarize, there is general consensus that UV light is a key catalyst of skin aging, even if the exact molecular mechanisms of this have not yet been fully elucidated. The quantity and activity of the MMPs that break down collagen is increased, whereas the synthesis of new collagen is inhibited (Fisher et al., 1996; Fisher et al., 1997). Reactive oxygen species, which themselves directly lead to DNA strand fractures in the cells, are formed at the same time (Sams, 1986; Bacqueville et al., 2009). The chronic overload on the body's detoxification mechanisms caused by this cumulative oxidative stress leads to protracted aging (Passos et al., 2006). A key role in this process is played by the interaction of the fibroblasts and the tightly woven fiber network that holds them. With increasing fragmentation of this fiber network, the fibroblasts lose their function in direct

Fig. 1.32 Cutis rhomboidalis nuchae is a clearly visible, classical sign of extrinsic skin aging where the neck has been chronically exposed to the sun.

correlation to the decrease in the pull that is exerted on them (Fisher et al., 2008). This gives rise to interesting interventional approaches, some of which have already been introduced into the clinical setting and are being intensively researched.



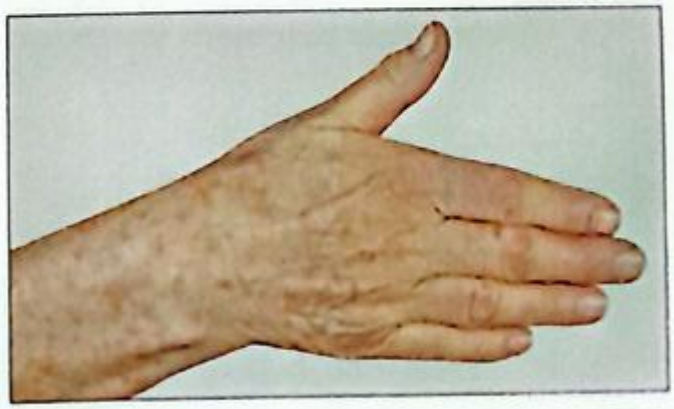


Fig. 1.33 Extrinsic skin aging on the hands. The unsightly pigmentation areas on the hands, caused by exposure, are common and successfully treated.

Histology of the epidermis

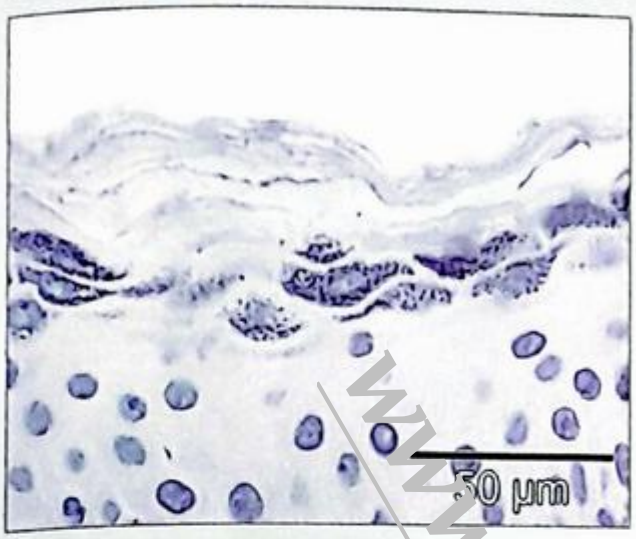


Fig. 1.11 Skin thickness is subject to natural fluctuations. The granular cell layer, in particular (Haemalaun 100×) can vary in thickness, the granules being precursors of the keratin filaments, which form the *stratum corneum*.

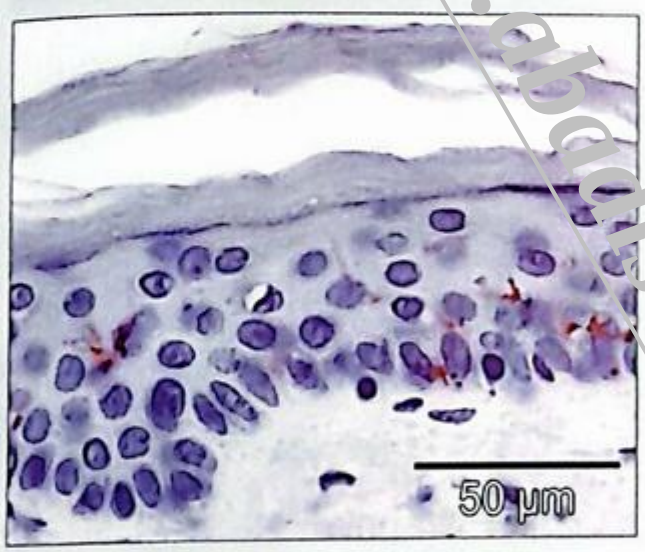


Fig. 1.12 Dendritic Langerhans cells can be found scattered in the suprabasal region of the epidermis (S100/Hemalaun, 100×). They occur in approximately the same numbers as the melanocytes and act as antigen-presenting cells. In addition, Merkel cells, which function as mechanoreceptors, can be found in considerably smaller numbers in hair follicles and the oral mucosa.

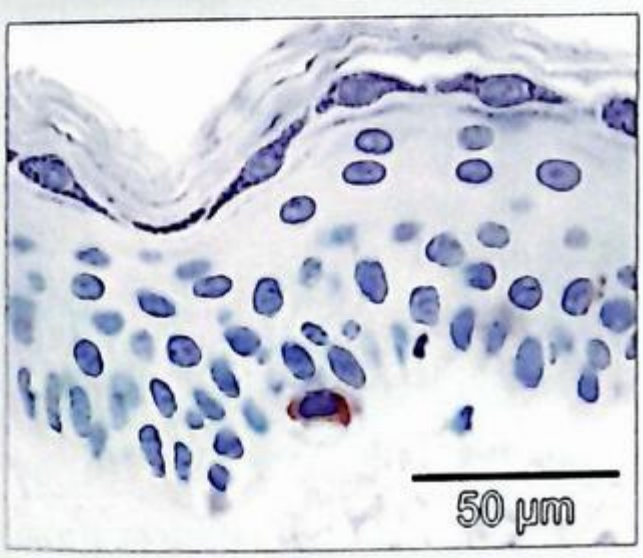


Fig. 1.13 The melanocytes are located between the basal keratinocytes in the epidermis (S100, 100×, melanocyte marked in red) and take on an important protective function by producing melanin and supplying it to the neighboring keratinocytes. A melanocyte is linked to about 30 keratinocytes.