The fact that it covers the entire body means the skin is the most obvious place to observe signs of ageing. The skin is the body’s largest organ – an average adult’s skin has a surface area of approximately 1.67m² and weighs around 4-5kg (Marieb and Hoehn, 2015). Skin is a malleable but tough structure, serving as a bag holding in all the body’s contents; without it, we would quickly succumb to water and heat loss, and pathogen invasion.

Skin structure

The skin consists of two main parts: the epidermis and the dermis. The epidermis is the outside layer and protects the underlying layers from the environment. It hosts cells that produce keratin, a substance that makes the skin waterproof and stronger, and cells that contain melanin, a photoprotective pigment that gives skin its colour. Although there is no blood supply in the epidermis, there are cells that afford the body immunity against bacteria and other invading organisms.

The second layer or dermis, the thickest layer of skin, contains cells that give skin its strength, support and flexibility. Sensory receptors in the dermis allow the body to experience pressure, pain and temperature, while small blood vessels provide the skin with nutrients and remove its waste products. Sebaceous glands – present all over the body except on the palms of the hands and the soles of the feet – produce oil that keeps the skin hydrated, softens hair and helps kill bacteria in the pores. The dermis also hosts the sweat glands and hair follicles.

Below these two main layers is an innermost layer of subcutaneous tissue, the hypodermis. This anchors the skin to the fascia (the underlying connective tissues that wrap around skeletal muscles) and is therefore known as the superficial fascia. The hypodermis consists mainly of fat cells that insulate the body and helps it to conserve heat.

Skin function

The skin and its derivatives (sweat glands, hair and nails) perform important func-


Thermoregulation

Blood flow in the dermis helps the body adjust to cold and heat. Sweat glands produce about 500ml of sweat a day, providing an important cooling mechanism. In hot weather, the nervous system stimulates the dermal blood vessels to dilate and sweat glands can increase their production to up to 1.2L a day (Marieb and Hoehn, 2015). The evaporation of sweat cools the body and prevents it from overheating.

In cold weather, the dermal blood vessels constrict, warm blood flow bypasses the skin, the skin cools down further and this slows down heat loss from the body.

Storage and synthesis

The skin acts as a storage facility for lipids and water. When it is hit by sunlight, modified cholesterol molecules are converted to a vitamin D precursor, which is transported to other body areas to be made into vitamin D (essential for calcium absorption from the gut). Skin cells also make collagenases; these are enzymes that support the natural turnover of collagen.

Sensation

The skin has a variety of nerve endings that feel touch, pressure, vibration, warmth, cold and pain, allowing the body to receive sensory stimulation from the surrounding environment.

Protection

With its stratum corneum (multiple layers of dead, flat cells and glycolipids) and hard keratinised cells, the skin forms a continuous physical barrier. It also provides chemical barriers:

- Acid secretions inhibiting bacterial growth;
- Dermcidin in sweat;
- Bactericidal substances in sebum;
- Melanin preventing ultraviolet damage (Marieb and Hoehn, 2015).

Intrinsic and extrinsic skin ageing

Skin ages intrinsically (chronologically from within) and extrinsically (due to external factors). Intrinsic skin ageing results from the passage of time and is mainly due to the action of reactive oxygen species (ROS) (Box 1) (Naidoo and Birch-Machin, 2017). It occurs within the skin itself due to reductions in dermal mast cells, fibroblasts and collagen production, and a flattening of the junction between the epidermis and dermis. Inextrinsically aged skin is unblemished, smooth, pale, dry and less elastic with fine wrinkles (Landau, 2007).

Extrinsic skin ageing is caused by environmental factors such as:

- Smoking;
- Diet;
- Exposure to chemicals;
- Trauma;
- Exposure to UV radiation (photoageing).

Such factors have been shown to stimulate the production of ROS and generate oxidative stress (Valacchi et al, 2012).

The greatest source of extrinsic ageing is accumulated, unprotected exposure to UV radiation; over 80% of facial skin ageing is due to low-grade chronic UV exposure (Flament et al, 2013). Acute exposure affects the epidermis, causing irreparable damage to cellular DNA, and induces the generation of ROS. Less than 30 minutes after UV irradiation, the amount of hydrogen peroxide – a potent ROS – has been shown to more than double in human skin (Rigel et al, 2004). UV exposure also disrupts collagen synthesis, leading to acute collagen loss (Rinnerthaler et al, 2015).

Exposure to UV radiation increases skin pigmentation and stimulates melanocyte proliferation. Melanin helps protect against the cumulative damaging effects of UV radiation, which explains why the rates of skin cancer are much higher in Caucasians than in black people and why basal cell carcinomas occur almost exclusively in the sun-exposed skin of light-skinned people (Tobin, 2017).

Extrinsically aged skin is characterised by coarse and deep wrinkling, rough texture, telangiectasia (spider veins), irregular or mottled pigmentation, a sallow or yellow complexion and a loss of elasticity (Tobin, 2017). The severity of extrinsic ageing depends on skin type – fairer skin is affected more than darker skin.

The ageing epidermis

The epidermis is formed of protective keratinised squamous epithelial tissue, with an outer barrier of dead cells (stratum corneum). Underneath are several layers of epidermal cells, ending in a basal layer of rapidly dividing cells located next to the basement membrane and dermis. Keratinocytes (cells producing keratin) and melanocytes (cells producing melanin) are found in the epidermis. Also present are Langerhans cells, dendritic cells that guard the ‘front door’ of the immune system, preventing unwanted foreign micro-organisms from entering the body via the skin. With age, there is a substantial loss of melanocytes and Langerhans cells (Yaar and Gilchrest, 2003).

Having no blood supply, the epidermis gains its nutrition through contact with the dermis. A major cutaneous change in intrinsic skin ageing is a reduction in the surface contact between the epidermis and dermis. There is a loss of rete ridges (epithelial extensions that project into the underlying connective tissue), which negatively affects the capillary-rich dermal papillae, resulting in a reduced supply of nutrients, metabolites and oxygen to the epidermis (Tobin, 2017). The reduced contact between dermis and epidermis also results in less resistance to shearing forces. In addition, with age, the epidermis atrophies because we produce fewer cells – cell production decreases by up to 50% between our 20s and our 70s (Cerimele et al, 1990).

The stratum corneum has a barrier function and keeps the skin from drying out; although it does not become thinner with age, it is not replaced as quickly, so skin is increasingly rough and dry. Extreme skin dryness (xerosis) can be seen in ageing skin, and this brings about an increased susceptibility to irritant dermatitis (Tobin, 2017). As mitosis in the basal layer of the epidermis is slowed down, healing takes more time.

With advancing age, there is a reduction in the hormones and chemical signals that are important for skin growth and repair, as well as a decline in the receptors that detect them; as an example, the number of vitamin D receptors in epidermal keratinocytes declines with age.

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**Box 1. Reactive oxygen species**

- Reactive oxygen species (ROS) are molecules generated by the skin during normal cellular metabolism that destroy skin cell membranes, DNA and enzymes.
- ROS cause the activation of collagenases and enzymes that degrade collagen as well as other proteins that make up the extracellular matrix, thereby impairing the structural integrity of the skin.
- With advancing age, the number of ROS increases and the ability of the body’s antioxidant defence system to get rid of them declines.

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Nursing Practice

**Systems of life**

- Thermoregulation;
- Storage and synthesis;
- Sensation;
- Protection.
The ageing dermis

The dermis contains blood capillaries that are important for providing oxygen and nourishment to all skin cells. It comprises an extracellular matrix containing a sturdy mesh of collagen and elastin fibres, which give the skin its strength, elasticity and resilience. Key cells in the dermis are:
- Fibroblasts – these synthesise collagen, elastin and other structural molecules of the matrix;
- Mast cells – these are immune cells that produce histamine.

With increasing age, there is a loss of dermal volume, and dermal thickness decreases by about 20%. There is a 50% decrease in the number of mast cells and a 60% decrease in blood flow (Farage et al, 2013); as a result, the skin’s response to injury or infection is compromised.

Collagen is the body’s most abundant protein, conferring strength and support to the skin. The collagen content of the dermis decreases by 1% per year throughout adult life (Rigel et al, 2004). In addition, collagen itself changes from well-organised bundles of fibres in young skin to fragmented and disorganised fibres in older skin; it also loses its interwoven extensions with elastin fibres, which in youth enable the skin to regain its shape after deformation (Graham-Brown, 2004).

Fibroblast activity decreases with age; these cells shrivel and have fewer epidermal growth factor receptors. There is an associated drop in collagen synthesis, an atrophy of collagen bundles and an increase in the levels of metalloproteinases and enzymes that degrade collagen. As a consequence, collagen is of poor quality and wound healing is impaired.

As collagen fibres decrease in number, rupture, cross-link and stiffen, their capacity to bind water diminishes and the skin loses elasticity and becomes wrinkled. Wrinkles appear to be due not so much to the degeneration of elastin fibres, as previously thought, but to a decreased water-holding power of collagen and mucopolysaccharides (Richards and Edwards, 2014).

Skin ageing is associated with a decrease in cutaneous perfusion and reduced vascularity, mainly in the superficial (papillary) part of the dermis. There is a drastic reduction in the number of dermal blood vessels accompanied by a shortening of capillary loops in the dermal papillae. This results in skin pallor, depleted nutrient exchange and impaired thermoregulation (Baumann, 2007; Waller and Maibach, 2005).

Less tolerant to trauma, the reduced volume of subcutaneous fat means the role of the hypodermis in limiting conductive heat loss is impaired. The distribution of subcutaneous fat changes too: it decreases in parts of the face and hands but increases in the thighs and abdomen. The reduction of fat over bony areas increases the risk of pressure ulcers (Box 2) and fractures.

Loss of protective function

The skin protects the body against mechanical injuries, mainly due to its ability to undergo reversible deformation when influenced by external forces – for example, weight. Human skin can be stretched to several times its original size. If, on termination of the external force, the skin returns to its initial shape, it is considered perfectly elastic; failure to do this is known as residual deformation. The skin of children is better able to withstand load
and tension, and is more elastic than that of older people (Vogel, 1987). With age, skin becomes stiffer, thinner, less tense and less flexible (Pawlaczek et al, 2013).

In addition, there is a loss of sensory receptors that enable the body to feel pressure, pain and temperature (Pacinian and Meissner’s corpuscles), as well as a loss of sensory nerve endings in the epidermis and dermis. This makes older people less able to detect changes in the environment and, therefore, more prone to injury.

Sebaceous glands produce less sebum, so the protection against infection afforded by the acidity of sebum is reduced. This makes the skin less able to resist disease and skin infections, which is why older people have an increased susceptibility to skin infections. The types of organisms that cause primary skin and soft tissue infections are diverse, and include bacterial (cellulitis), viral (shingles) and fungal (thrush) pathogens.

Features of aged skin

Wrinkles and sagging

Several factors contribute to the formation of wrinkles: gravitational force, loss of subcutaneous fat and repeated traction exerted by facial muscles over expression lines, which results in deep creases over the forehead, between the eyebrows, periorbitally and in the nasolabial folds. Clinical manifestations include dryness, laxity and slackness; these are most easily observed on the face, which is the most exposed area of skin. In addition, facial muscles show an accumulation of the ‘age pigment’ lipofuscin, a marker of muscle cell damage. This damage, along with diminished neuromuscular control, contributes to wrinkle formation.

Depletion and redistribution of facial fat (which tends to accumulate in pockets such as the nasolabial folds and submandibular region) as well as gravity contribute to the loosening and sagging of the skin.

Changes in hair and nails

Chest, axillary and pubic hair all decrease in density with age, but men may experience increased hair growth in other body sites like the eyebrows, ears and nostrils (Tobin, 2017). Hair becomes drier because sebaceous glands produce less sebum.

Hair greying, which is genetically controlled, appears to be a consequence of a depletion of hair melanocytes, which can no longer provide colour to the developing hair in the follicle. This depletion may occur as a result of signal failing by melanocyte stem cells, which can no longer maintain the production of melanocytes (Nishimura et al, 2005). Wood et al (2009) have shown that hydrogen peroxide, which is produced by hair follicles, builds up over time and leads to a gradual loss of hair colour. Greying occurs at different rates in different hair follicles, either rapidly or slowly over decades. White hair is thicker and grows faster than pigmented hair (Trueb and Tobin, 2010).

As we age, nail growth starts to slow down. Nails become more brittle and develop beaded ridges due to a reduction in lipophilic sterols and fatty acids (Heldmacher et al, 2000) (Box 3).

Skin lesions

The number of melanocytes decreases with age and those remaining increase in size; this explains why blotchy pigmented ‘liver spots’ may appear on the back of the hands.

The formation of benign skin lesions such as seborrhoeic keratoses or solar lentigos (‘age spots’) typically starts between the third and fifth decade of life, and speeds up thereafter. Due to the cumulative effects of chronic sun exposure, the risk of skin cancers also increases with age.

Cherry angiomas are cutaneous vascular proliferations commonly seen in older people, and typically appear as round-to-oval, bright red, dome-shaped papules and pinpoint macules, most commonly on the trunk or proximal extremities. Initially described by Campbell de Morgan in 1872, a cherry angioma is formed by numerous newly developed capillaries and prominent endothelial cells arranged in a lobular fashion in the papillary dermis (Kim et al, 2009). Early lesions appear as flat, red macules that look like petechiae. As they develop, they become 1-5mm red papules. Cherry angiomas are usually asymptomatic but may bleed with trauma.

Senile purpura

Senile purpura is a common, benign and self-resolving condition affecting more than 10% of people aged over 50 years. It is characterised by the recurrent formation of irregularly shaped, dark purple ecchymoses, about 1-4cm in diameter, often appearing on the forearms after a minor trauma (Trozak et al, 2006). They do not undergo the colour changes of a normal bruise and take up to three weeks to resolve. Risk factors include chronic sunlight exposure and the use of oral or topical corticosteroids and anticoagulants.

Senile purpura is caused by the thinning of dermal tissues and increase in the fragility of blood vessels. As a result, superficial vessels tear and rupture, even with negligible trauma. The subsequent leakage of blood into the surrounding dermis results in senile purpura.

Assessment and care

Although age-related skin problems are not usually life threatening, they can cause distress and decrease quality of life. Skin conditions that commonly affect older people are eczema, psoriasis, infections and pruritus (Davies, 2008), many of which are associated with dry skin and itching. Decreased sebaceous secretions, loss of oil glands and circulatory changes contribute to dry and scaly skin in the

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**Box 2. Pressure ulcers**

- Pressure ulcers are a particular risk in older people with compromised blood circulation, reduced mobility or faecal and/or urinary incontinence
- Most pressure ulcers will harbour a bacteraemia and this can be the cause of localised infection, cellulitis or osteomyelitis
- Pressure ulcers, which are preventable, can cause great pain, distress and even death; sepsis is a very serious complication

**Box 3. Ageing nails**

- Ageing results in thickening of the nails, particularly the toenails
- Thick, hard nails become difficult to cut and older people may not have the strength required to do so
- If nails overgrow, because they are confined in shoes, they may begin to curl under the toes; this results in a condition called ram’s horn nails, in which the nail curls over the top of the toe and grows into the flesh on the bottom, causing pain
- Fungal infections become increasingly common in old age, causing thick, brittle, misshapen and discolored nails; they are more likely in people who have underlying conditions such as diabetes
- If in severe discomfort due to nail problems, older people may avoid walking, thereby becoming less mobile
In tissue integrity may increase the older age (Tobin, 2017). However, any breach of barrier or eczema. Such as steroids, for conditions like psoriasis, are often used alongside other treatments, effectively alleviate skin dryness. They are especially useful for older people who have dry and itchy skin; these increase the risk of infection (Laube, 2004).

Pruritus, a common complaint in older adults, may be caused by dryness, irritation or infection. It is also linked to diabetes, kidney disease and anaemia. About 85% of older people develop ‘winter itch’, as dry, overheated indoor air compounds skin dryness. Pruritis can substantially decrease quality of life, especially if it leads to sleep deprivation (Patel and Yosipovitch, 2010).

Emollients increase the amount of water held in the skin and so are useful for older people. They lower extremities, and skin becomes more susceptible to inflammation, infection and rashes.

Skin assessment and skin care are essential components of nursing care for older people. Skin assessment should be undertaken regularly and encompass detailed visual inspection and assessment of texture, moisture, turgor and temperature; personal skin hygiene should be encouraged (Cowdell and Radley, 2012). Skin care should include the care of nails.

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