

# Structural Changes Associated with Aging Skin

*by Dr. Diana Howard*

If we look at skin that has only endured normal intrinsic or chronological aging, without environmental influences, it is smooth and generally unblemished. There are exaggerated expression lines on the skin, but by and large, the skin is well preserved. Under the microscope, we will see some signs of aging, which include a flattening of the epidermal-dermal interface and some breakdown of the dermal tissue.

In direct contrast, extrinsically-aged skin (such as that found on our face, hands and chest) is wrinkled, sallow in color and has areas of hyper- and hypopigmentation. Skin may show a loss of tone and elasticity, increased fragility, areas of purpura caused by blood vessel weakness and benign lesions such as keratoses, telangiectasis and skin tags. Under the microscope, discreet changes are evident in the collagen and elastin, which are now fragmented and thick, indicative of the cross-linking that is associated with wrinkle formation.

Scientists have found more modern ways to address the issue of wrinkles, skin discoloration, the breakdown of collagen and elastin, dehydration and the slowdown of cell turnover through understanding the biochemical reactions that trigger these structural changes.

## **Wrinkles**

Wrinkles are depressions in the skin's surface that may be coarse or fine, depending on their depth. Wrinkle depth may extend from a few micrometers to several millimeters. Coarse wrinkles, often referred to as expression lines, appear on the forehead, outer corners of the eyes (crow's feet) and as vertical lines on either side of the mouth (laugh lines). Fine wrinkles are the shallower network of lines or indentations that appear on our skin, especially in areas of facial movement (such as the eyes, mouth, upper lip, etc.).

Wrinkles occur as a result of:

- A reduction in muscle mass and skin thickness.
- Cross-linking of collagen and elastin in the dermis.
- Dehydration of the Stratum Corneum (SC).

This results in visible wrinkles on the surface of the skin and a loss of mechanical strength and elasticity.

## **Skin Discoloration**

Changes in skin color are often associated with aging. Skin color is a composite of red, blue, yellow and brown coloration. This is the result of red oxygenated hemoglobin, yellow carotenoids and flavins and the brown melanin pigment of our skin. Hyperpigmentation spots are due to erratic melanocyte activity that is the result of cumulative Ultraviolet (UV) exposure. This is often associated with hypopigmentation (white spots), which also accompanies aging. The result is a

mottled, older skin appearance made up of darker and depigmented areas. When we see an increase in yellow coloration in aged skin, it is the result of a decrease in brown melanin pigment along with a decline in red and blue-colored capillaries. In the case of cigarette smokers, the toxins cause a breakdown of elastin that also contributes to the yellow color of skin. This overall skin discoloration is often accompanied by an increase in broken veins.

While hyperpigmentation is most often associated with skin aging, we also see hypopigmentation due to a reduction in the number of melanocytes; there is a decline of 6-8% per decade after age 30, which accounts for the lighter skin color. This not only leads to a reduction in melanin (hypopigmentation), but it also accounts for a diminished protective capacity against UV exposure. Along with the decline in melanocytes, there is a reduction in both the number and functionality of the other dendritic cells of the epidermis (the Langerhans cells), which creates a lowered immune response for the skin. This results in decreased immune surveillance, which may account for the heightened incidence of premalignant and malignant lesions in aging skin.

### **Breakdown of Collagen and Elastin**

The majority of age-dependent changes that occur in our skin happen in the dermis, which can lose from 20-80% of its thickness during the aging process. This is the result of changes in the fibroblasts, the cells responsible for collagen, elastin and glycosaminoglycan (GAG) biosynthesis. Not only is the collagen and elastin produced at a slower rate, which impacts the skin's inability to repair itself, but the organization of the protein also changes, affecting the skin's structure.

The breakdown of collagen and elastin is controlled by the activity of Matrix Metalloproteinase (MMP) enzymes known as collagenase and elastase, respectively. Studies have shown that UV radiation activates these enzymes within hours of UVB exposure. Long-term elevation of the MMPs, which is typically found in people with prolonged exposure to sunlight, results in disorganized and clumped collagen and elastin that is characteristic of photodamaged skin.

Changes in elastin fibers are so characteristic in photoaged skin that the condition known as elastosis is considered a hallmark of photoaged skin. This is characterized by an accumulation of amorphous elastin protein and a breakdown in the typical structural layout, which results in decreased skin elasticity and tensile strength. This phenomenon accounts for why more mature skin takes longer to assume its original position when extended or pulled.

### **Dehydration**

Like the collagen and elastin proteins of the dermis, the ground substance, or intercellular glue, that holds these proteins in place also undergoes age-related changes. There is less ground substance as we age, and distribution of GAGs, such as Hyaluronic Acid, changes as well. Studies have shown that the amount of Hyaluronic Acid found in the dermis starts to diminish as early as our forties. This loss, along with a compromised barrier layer in the epidermis, is most likely the cause of dehydration and loss of turgidity, which contributes to altered elasticity in aging skin.

In addition to dehydration in the dermis, studies have indicated a reduction in the moisture content of the epidermal Stratum Corneum (SC), which is most likely due to a reduction in the SC lipids, resulting in an inefficient ability to bind and retain water. The result is the appearance of fine lines and scales. Fortunately, application of moisturizers and the regular use of exfoliants (in particular, exfoliants containing Lactic Acid) can alleviate this problem.

### **A Slowdown of Cell Turnover**

A discussion of the effects of aging on the epidermis would not be complete without including the effects of aging on cell turnover rates. Studies indicate that the epidermal turnover rate slows from 30-50% between our thirties and eighties. Studies have demonstrated that in young adults, the Stratum Corneum transit time was as quick as 20 days, whereas in older adults it stretched to 30 days or more.

This prolonged Stratum Corneum replacement rate also coincides with a subsequent slowing of the wound healing process that is typical in older people. In fact, doctors report that older patients take twice as long to reepithelialize after dermabrasion/resurfacing procedures when compared to younger patients. The slow down in the cell cycle is combined with a less-than-efficient desquamation process, and it accounts for the characteristic dull, rough skin surface seen in maturing skin.

### **Understanding Biochemical Reactions**

The treatment of skin aging used to rely on addressing the structural manifestations of photoaged skin, such as wrinkles and loss of elasticity. Today, we can more effectively treat this skin condition by addressing the actual biochemical reactions that trigger these structural changes. Understanding these structural changes that occur in the epidermis and dermis, as well as the biochemical reactions that trigger them, will give us greater success in their treatment.