



## YOUTH EYE™ COMPLEX

Dr. Charlene DeHaven, M.D.  
Clinical Director, INNOVATIVE SKINCARE®

YOUTH EYE™ COMPLEX improves existing age-related changes and decreases ongoing aging in the periorbital area (also see YOUTH EYE™ COMPLEX Clinical Study).

### THE PERIORBITAL AREA IN THE AGING FACE

The area surrounding the eye is termed the periorbital area and is the first visible area on the body to exhibit the changes of aging. Findings associated with periorbital aging include dark under-eye circles, “bagging” or loss of elasticity, shadowing in the tear trough (area beneath the inner eye where tears first track), “crow’s feet” or wrinkling, and loss of opacity (increased translucency) of the under-eye skin. Skin here is thinner than other areas of the body, vessels are nearer the surface, exposure to the sun’s rays is high and biologic protective mechanisms are less robust. Thus, the periorbital area mandates earlier and more aggressive protection to avoid early aging.

There are some differences among ethnic groups related to aging in the periorbital area. Blacks tend to report periorbital aging sooner in life than other groups. This relates to drooping in the lateral canthal area. The lateral canthal area is the cartilaginous support provided by the inner eyelid rims nearest the temple areas of the face. When structural integrity of the glycosaminoglycans forming cartilage and cartilage itself is lost, the lateral canthal angle decreases causing a “drooping” appearance of the outer eye and giving a more aged appearance. Asians tend to hyperpigment prior to wrinkling as aging occurs. Thus, individuals of Asian descent will be concerned with increasing pigmentation first and with wrinkling in their later years compared to Caucasians. This relates to the greater number of sebaceous glands in Asians, thicker skin in general and more numerous melanocytes which are the pigment-producing cells. Although the manifestations of periorbital aging seem to differ somewhat among

genetic groups, the common denominator in all groups relates to free radical damage and inflammatory mediators as well as the individual’s response to these stressors.

### MECHANISMS OF PERIORBITAL AGING AND THEIR MODULATION

Free radical damage or “oxidative stress” is a primary cause of tissue damage and aging in the periorbital area as well as elsewhere. Antioxidants are extremely helpful in addressing this primary cause of aging. These antioxidants must be of sufficiently high quality to be functional and must be formulated in a manner that favors their deliverability to the site of action. Solar exposure bombards the skin with photons, small energy packets from the sun that are themselves free radicals. Sunscreen with deflection (i.e. “blocking”) and/or absorption of photonic energy is essential in the periorbital area as elsewhere. Inflammation results from free radical damage and begets more damage and inflammatory change, thus perpetuating these processes. Wrinkling results from repeated low level inflammatory events and is actually scarring directly related to the body’s attempt to heal these repeated occurrences.

Wrinkles can be improved by injecting Botulinum toxin (Botox), which is used to mediate neural transmission and muscle contraction, lessening the appearance of wrinkles. Other peptides have also been shown to decrease muscle contraction and decrease the appearance of wrinkles. If applied topically, these must be of a small enough molecular weight to penetrate the skin and exhibit activity on the myoneural (muscle-nerve) complex. Topical peptides used in cosmetic products affect the smaller, more superficial muscles rather than the deeper, subdermal muscles of expression targeted by Botulinum toxin.

The decreased thickness of the under-eye skin compared to other areas also contributes to more rapid aging here.



Since blood vessels are nearer the surface, the bluish coloration of the capillary venules and their reduced hemoglobin are more noticeable. Thickening of the skin under the eye would make the skin less translucent and would cause the underlying vessels to be less obvious. Collagen deposition can be encouraged by growth factors which encourage growth and differentiation of healthy skin cells. Thickening of the periorbital skin also makes capillary hemoglobin leakage less likely. Hemoglobin that leaks outside these small vessels is oxidized by sunlight and turns a darker color.

Sagging of tissue results in more prominent shadowing below the tear duct. Loss of elasticity may be addressed by encouraging collagen deposition, decreasing wrinkling and limiting oxidative damage.

Dark under-eye circles are termed periorbital hyperchromia. Some ethnic groups are more subject to this occurrence. Photodamage and inflammation produce a more intense hyperpigmentation in those with more numerous melanocytes in their skin (those having darker skin). Any factors increasing vascular congestion in the under-eye area will increase swelling and eventually produce dark circles. Allergists and pediatricians have long known their allergic patients will manifest dark infraorbital circles. The inflammation and vascular congestion associated with the allergic response can be so severe that the infraorbital areas actually appear bruised. These inflammatory responses are mediated by cytokines such as Prostaglandin E2 (PGE2) and Leukotriene B4 (LTB4), as well as other inflammatory mediators such as histamine and Interleukin-6 (IL-6). The body's own intrinsic antioxidant system tries to fight these processes with antioxidants such as superoxide dismutase (SOD). It has been clearly shown in research studies that as intrinsic (produced within the body) or supplied antioxidant levels increase, levels of inflammatory mediators decrease. Therefore, decreasing inflammation and increasing antioxidant protection preserves the vascular endothelium and decreases these dark circles by limiting vascular congestion.

When vitamins and pro-vitamins (vitamin precursors)

were topically applied for 8 weeks in a group of Asian women, dark circles decreased noticeably. There was an additional associated decrease in wrinkles in the same group. In other studies, centella asiatica and its active ingredients asiaticoside, asiatic acid and madecassic acid caused a significant decrease in wrinkling. Centella asiatica is a potent wound-healer and these effects may be antioxidant, anti-inflammatory, DNA-modulating and/or other.

Apoptosis is a process of programmed cell death in which damaged cells are eliminated. Encouraging the death of damaged cells through apoptosis acts on important checkpoints in the synthesis of new cells. This can either delay cellular development until DNA repair occurs and the cell is made functional again or can facilitate the death of a cell too damaged to be repaired. Apoptosis is important during any type of cellular damage but particularly so in photoaging. When sunburn from UVB damage occurs, the damaged cells, termed "sunburn cells", are actually cells experiencing apoptotic death. Although the apoptotic process incorporates biologic stopgap measures to limit inflammation accompanying this type of cell death, some inflammation still occurs with apoptosis – thus leading to some increase in free radical damage.

#### YOUTH EYE™ COMPLEX IMPROVES THESE PERIORBITAL AGING PROCESSES

YOUTH EYE™ COMPLEX contains several potent ingredients, many of which act in concert with each other to affect aging in the periorbital area. As previously discussed, this area of the face ages first and most severely in all ethnic groups, although the exact aging appearance may vary somewhat according to ethnicity.

Peptide technology—also known as protein or amino acid technology, or sometimes epigenetics—is one of the fastest-growing areas of biochemistry. The short-chain peptides in YOUTH EYE™ COMPLEX are beneficial in a number of ways. They decrease wrinkling by direct action on the myoneural terminal and encourage growth and development of thicker



infraorbital skin that is less translucent, firmer, and causes deeper dark circles to be less visible. Other proteins and peptide chains provide the “building blocks” for synthesis of healthy collagen. This also thickens skin and decreases wrinkles. The glycosaminoglycans (GAGs) within the product are plant-derived. Soy and wheat proteins are designed to locate, adhere to and repair damaged sites. Copper tripeptide-1 signals fibroblasts to commence collagen synthesis and repair, incorporating these amino acid building blocks to thicken periorbital skin and improve collagen architecture.

Vitamins and pro-vitamins in the formula are encapsulated within liposomes. This provides a delivery system that carries the active ingredients to the desired site where they support healthy metabolism.

The visibility of wrinkles is immediately improved as the wrinkle is smoothed. YOUTH EYE™ COMPLEX is also intensely hydrating which instantly decreases the appearance of wrinkles.

Although powerful, this “intelligent” formula is gentle and designed to repair and rebuild skin in an orderly way. Individual ingredients are able to recognize where they should go for optimum benefit and are packaged within delivery systems to reach their site of action. The damaged portions of cells transmit specific signals which the formula ingredients are able to recognize. The particular ingredients are then able to bind to the damaged area and begin repair.

The product lessens inflammation which also improves the aging periorbital area. Anti-irritant and anti-inflammatory effects protect the oil-soluble and water-soluble parts of the skin cell at all times and decrease free radical damage.

## REFERENCES

- J Drugs Dermatol. 2010 Sep;9(9):1065-71.
- Facial Plast Surg. 2010 Aug;26(3):201-8. (Epub 2010 Jun 3)  
J Drugs Dermatol. 2010 Aug;9(8 Suppl ODAC Conf Pt 2):s118-28.
- J Cosmet Dermatol. 2010 Jun;9(2):108-16.
- Pathol Biol (Paris). 2010 Jun;58(3):226-31. (Epub 2009 Nov 5)  
J Plast Reconstr Aesthet Surg. 2010 May 21. (Epub)  
Facial Plast Surg Clin North Am. 2010 Feb;18(1):19-33.
- Plast Reconstr Surg. 2010 Feb;125(2):699-708.
- Semin Plast Surg. 2009 Nov;23(4):274-82.
- Clin Dermatol. 2009 Sep-Oct;27(5):469-74.
- J Cosmet Dermatol. 2009 Sep;8(3):228-33.
- Plast Reconstr Surg. 2009 Sep;124(3):946-51.
- Semin Plast Surg. 2009 Aug;23(3):198-206.
- Dermatol Surg. 2009 Aug;35(8):1163-71.
- Dermatol Surg. 2009 Aug;35(8):1163-71.
- J Drugs Dermatol. 2009 Jul;8(7 Suppl):s15-8.
- J Drugs Dermatol. 2009 Jul;8(7 Suppl):s4-7.
- J Cosmet Dermatol. 2009 Jun;8(2):127-35.
- J Drugs Dermatol. 2009 May;(5 Suppl Skin Rejuvenation):4-13.
- J Am Acad Dermatol. 2008 Sep;59(3):397-404.
- Clin Dermatol. 2008 Jul-Aug;26(4):364-6.. 2008 Jun;10(2):104-9.
- J Cosmet Laser Ther. 2008 Jun;10(2):104-9.
- Int J Cosmet Sci. 2008 Jun;30(3):167-73.
- Skin Res Technol. 2008 May;14(2):127-34.
- J Craniofac Surg. 2008 May;19(3):812-6.
- Plast Reconstr Surg. 2008 May;121(5):1793-802.
- Plast Reconstr Surg. 2008 Mar;121(3):1002-8.
- J Drugs Dermatol. 2007 Nov;6(11):1141-8.
- Plast Reconstr Surg. 2007 Oct;120(5):1367-76.
- Dermatol Ther. 2007 Sep-Oct;20(5):350-9.



Dermatol Ther. 2007 Sep-Oct;20(5):343-9.

J Cosmet Dermatol. 2007 Sep;6(3):211-5.

Facial Plast Surg. 2007 Aug;23(3):162-7.

Eur J Ophthalmol. 2007 Mar-Apr;17(2):143-50.

J Drugs Dermatol. 2007 Feb;6(2):197-201.

Facial Plast Surg. 2006 Aug;22(3):204-14.

J Dermatolog Treat. 2005 Apr;16(2):79-86.

Skin Res Technol. 2005 Feb;11(1):47-52.

J Cosmet Dermatol. 2004 Apr;3(2):73-5.