Photoaging

True statement: Chronic exposure to sunlight contributes to premature aging.
Chronic sun exposure can be seen when one compares the exposed skin of the face, hands, or neck to the unexposed skin of the buttocks, inner thigh, or inner arm.
Figure 3-1.
The extensor (sun-exposed) surface of the arm shows much more photodamage than the flexor (unexposed) surface of the arm. This can be used to demonstrate to patients the importance of photoprotection.
Figure 3-2.
A Wood’s lamp can be used to highlight solar lentigos caused by the sun. The advantage is that the patient can immediately see the sun damage without waiting for film to be developed.
Intrinsic vs. Extrinsic

- Intrinsic – genetic background of an individual and results from the passage of time; beyond voluntary control
- Extrinsic – external factors such as smoking, excessive use of alcohol, poor nutrition, and sun exposure than can be reduced with effort = df = prematurity skin aging
  - 80% of facial aging is attributable to sun exposure
Intrinsic vs. Extrinsic Characteristics

- Intrinsic - Skin is smooth and unblemished
- Intrinsic - Preservation of the normal geometric patterns of the skin
- Extrinsic – exposed areas (face, chest, arms)
- Extrinsic – pigmented lesions: freckles, patchy hyperpigmentation, depigmented lesions
  - Associated with increased risk of melanoma
Figure 3-3.
This is the photographic image of the same patient in Figure 3-4 using a normal camera.

Figure 3-4.
A UV camera is the best way to document photodamage. However, unless a digital system is used, the film must be developed and shown to the patient on the follow-up visit. This camera can show patients the improvement in their skin after therapies have been instituted.
Photoaging Classification

- Type 1: No wrinkles.
- Type 2: Wrinkles in motion
- Type 3: Wrinkles at rest
- Type 4: Only wrinkles
Type 1

- Typical ages 20s to 30s
- Early photoaging
- Mild pigmentary changes
- No or minimal wrinkles
Type 2

- Typically ages late 30s to 40s
- Early to moderate photoaging
- Early senile lentigines (age spots)
- Parallel smile lines beginning to appear lateral to mouth
Type 3

- Typically age 50 or older
- Advanced photoaging
- Obvious wrinkles
Type 4

- Typically age 60 or over
- Severe photoaging
- Yellow-gray skin
- No “normal” skin
Other signs of aging

- Loss of tone and elasticity
- Increased skin fragility
- Epidermal atrophy and discrete changes in collagen and elastin fibers
- Benign lesions
  - Skin tags
Severe Photoaging

• Collagen fibers are fragmented, thickened, and more soluble (Lavker, 1979)
• Elastin fibers also appear fragmented and may show progressive cross-linkage and calcification (Yaar, 1999)
• These changes worsen with continued UV exposure
Figure 3-6.
Seborrheic keratosis and other benign neoplasms often occur in areas with significant sun exposure.
Figure 3-7.
Hematoxylin and eosin (H and E) stain of sun-damaged skin demonstrates significant elastosis in the dermis and multiple solar lentigos. (Image courtesy of George Ioannides, MD.)
Characteristics of Aged Skin

• Epidermis (not as exaggerated as dermal changes)
  – Thinner in some studies (Lavker, 1979)
  – Arguable (Whitton, 1973)
  – Most dermatologists agree that the thickness of the SC is unchanged with aging
Characteristics of Aged Skin

• Dermal-Epidermal Junction (DEJ)
  – Flattens with age
  – In abdominal skin: DEJ decreased from 2.64 mm² in subjects aged 21 to 40 years to 1.91 mm² in subjects aged 61 to 80 (Katzberg, 1958)
Characteristics of Aged Skin

• Decreased Cell Turnover
  – Epidermal turnover rate slows from 30% - 50% between the third and eighth decades of life
  – SC transit time: 20 days in young adults and 30 or more in older adults
  – Leads to decelerated wound healing
  – Slow cell cycle is combined with less effective desquamation in many individuals > 65 yrs.
Characteristics of Aged Skin

- Dermis
  - Individuals 65 yrs of age and older demonstrate a loss of approximately 20% of dermal thickness
  - Changes in collagen production and fragmented elastic fibers
  - Greater changes with UV exposure: disorganization of collagen fibrils and accumulation of abnormal elastin-containing material
Collagen

• “antiwrinkle creams”
• Zyderm, Zyplast, Dermalogen, Cymetra
• Vitamin C and glycolic acid
• Little is known about wrinkle pathogenesis
  – Neither an animal model nor an in vitro model of wrinkling has been established
Figure 3-8.
The spinous layer is thinner and there are fewer keratohyaline granules in the valley of the wrinkle. Levels of collagen IV and VII are also decreased in the valley of a wrinkle when compared to the flanks.
Collagen Facts

• 70% of dry skin mass
• Ratio of collagen types changes with aging
• Overall collagen content per unit area of skin surface decreases approximately 1% per year
• Loss of a particular type of collagen may lead to wrinkle formation (type IV)
  – Affects mechanical stability of DEJ
UVR and Dermal Fibers

- UVR induces collagen damage
- The mechanism of collagen degradation by UV exposure is well-characterized
- Enzyme regulated
- Elastin degradation not as well known, but is likely similar in mechanism to collagen degradation
Other Aging Factors

• Melanocytes
  – Number of melanocytes decreases from 8% to 20% per decade
  – Loss of melanin = loss of protection from UV

• Vasculature
  – 35% reduction in the venous cross-sectional area in aged skin as compared to young skin

• Subcutaneous Tissue
  – Loss and gain site-specific (face, hands-dorsal, shins)/(waist and abdomen)
Cigarettes and Aging Skin

• Aging effects known as early as 1856
• Not as understood as the correlation between smoking and lung cancer, emphysema, chronic bronchitis, heart disease
• Aging effects can be seen – “smoker’s face” or “smoker’s mouth”
Figure 4-1. 
Smoker demonstrates premature wrinkling around the mouth known as “smoker’s mouth.”
Study Highlights

• 3 groups of postmenopausal women: lifelong nonsmokers, current smokers, and former smokers
  – Moderate to severe wrinkling for current smokers as greater than twice that for lifelong nonsmokers
Study Highlights

- Women more susceptible than men
- Duration and amount of smoking are significant factors in wrinkle development
- Facial wrinkling more prominent among smokers than among people who have never smoked
- Smoking worse than sun exposure for facial wrinkling
Mechanism

• Likely enzyme regulated
• Smoking also appears to reduce facial SC moisture as well as vit. A levels
• Genetic factors (not all patients exhibit “smoker’s face”)
• Chronic smoking diminishes capillary and arterial blood flow leading to loss of nutrients and O₂ delivered to tissue
• IR factor
Conclusions

• PREVENTION IS EASY!!!!

• WEAR SUNSCREEN AND DON’T SMOKE