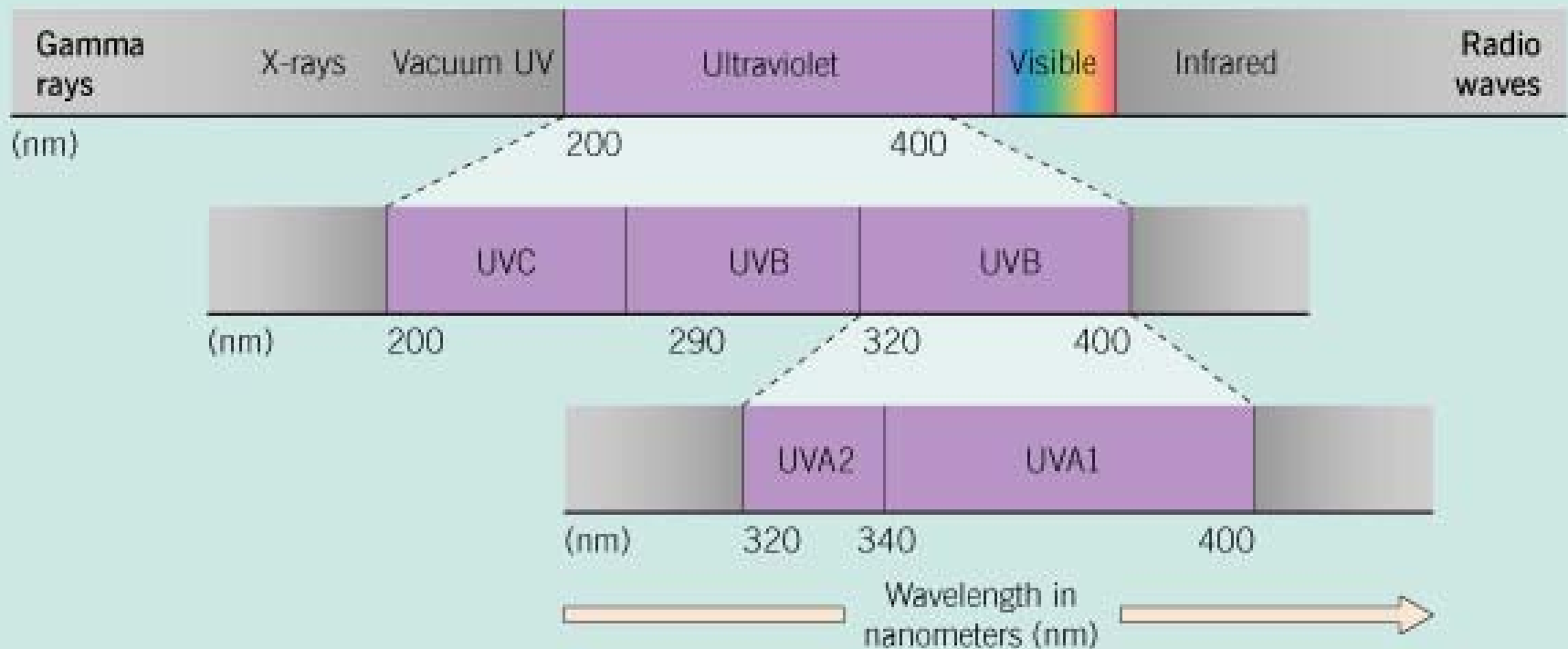


Photodermatology

Physics

- Electromagnetic radiation can be described theoretically in two ways:
 - Waves
 - Photons (packets of energy or quanta)
- The following equations are important in understanding how these are related:
 - $E = hc/\lambda$
 - $E = v h$
 - E =energy of photon; h =Planck's constant; c =speed of light;
 λ =wavelength; v =frequency
- Take home points:
 - As wavelength increases, the energy decreases (inverse relationship)
 - As frequency increases, so does the energy

ELECTROMAGNETIC SPECTRUM WITH EXPANDED UV REGION



Electromagnetic Spectrum

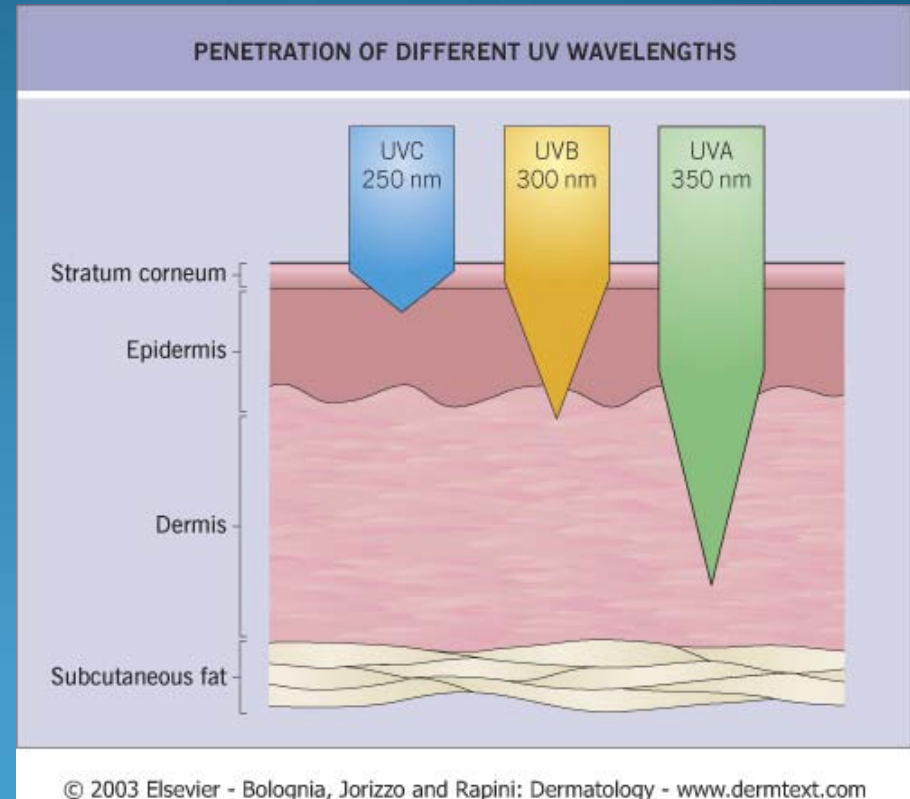
- The sun emits UV radiation as part of an electromagnetic spectrum.
- $<10\text{nm}$ (Gamma & X-rays) = *Ionizing Radiation*
 - Particles at this energy generally remove or ionize electrons
- UVC (200-290nm)
 - Filtered by the ozone
 - Well absorbed by DNA, RNA, and proteins
 - Lethal to viable cells in the epidermis (Often called *Germicidal radiation*)
- UVB (290-320nm)
 - 1000X more erythemogenic than UVA
 - Ordinary window glass filters UV less than 320nm

Electromagnetic Spectrum

- **UVA (320-400)**
 - **More than 95% of the sun's UV radiation reaching the earth's surface is UVA**
 - Sometimes called *Black Light* because it is not visible to the human eye but causes certain substances to emit visible fluorescence
 - Woods lamp approx 365 nm (UVA₁)
 - UVA₂ (320-340)
 - More erythemogenic wavelengths in UVA₂
 - UVA₁ (340-400)
- **VISIBLE LIGHT (400-760)**
- **INFRARED & RADIO WAVES (>760)**
- Remember that these are guidelines and that there is wide variation within the same spectrum
 - Example: UVB: 297nm is 100X more erythemogenic than 313nm
 - This is the principle used in Narrow-Band UVB therapy

Electromagnetic Spectrum

- Depth of penetration of UV light is wavelength dependent.
- Longer the wavelength the deeper the penetration.
- UVA readily reaches the dermis.
- UVB is absorbed in the epidermis and small part in upper dermis.
- UVC absorbed or reflected predominantly in the stratum corneum.
- Different wavelengths may have biological effects even in a layer that it does not reach secondary to secretion of an inflammatory mediator.



Electromagnetic Spectrum

- The ability to induce sunburn rapidly declines with increasing wavelength.
- 360 nm is approximately 1000 fold less erythemogenic than light with a wavelength of 300 nm.
- UVB induced sunburn reaches its peak between 6 and 24 hours after exposure
- UVA an immediate erythema is observed followed by a distinct delayed erythema after 6 to 24 hours.

Tanning

- Tanning is biphasic and wavelength dependent.
- Immediate pigment darkening occurs during and immediately after exposure due to alteration and redistribution of existing melanin.
- Most prominent with UVA.
- Delayed tanning is result of UVB.
- Peaks about 3 days after sun exposure.
- UVB induced tan is based on an increased number of melanocytes, increased melanin synthesis, and increased transfer of melanosomes to keratinocytes.
- UVA induced tan provides 5-10 times less protection against sunburn secondary to less pronounced epidermal thickening and hyperkeratosis.

Skin Phototypes

- An individual's tendency to develop sunburn and tanning after sun exposure has been used to categorize skin phototypes.
- These correlate well with susceptibility to long term effects of exposure.

Table 134.3 Skin phototypes. ^aTypes I–IV are determined by history; types V and VI by physical examination (racial descent). ^bPatients with erythrodermic psoriasis are to be classified as skin phototype I for determination of UVA dosage. ^cPatients with this phototypes should be classified into a lower skin phototype category if the sunburning history so indicates.

SKIN PHOTOTYPES		
Skin phototype ^a	Skin reaction	Recommended dose (J/cm ²)
I ^b	Always burn, never tan	0.5
II	Always burn, but sometimes tan	1.0
III	Sometimes burn, but always tan	1.5
IV	Never burn, always tan	2.0
V ^c	Moderately pigmented skin	2.5
VI	Darkly pigmented skin	3.0

- Long term effects of chronic sun exposure include photoaging and photocarcinogenesis.
- UVA penetrates deeper in dermis thus probably more important role in photoaging.
- Only UVA penetrates glass not UVB.



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SOURCES OF ELECTROMAGNETIC RADIATION

- UV Radiation comes from many sources
 - the sun is the most important
 - Incandescent and Fluorescent Lamps
 - Woods lamp (~320-420nm)
 - welder's arcs, etc.
- Luckily, the ozone filters out all EM energy <290nm since this is HIGHLY damaging to plants and animals
- Some scientists have calculated that a 1% decrease in the ozone level increases the risk of non-MM skin cancer by 2.7%

SOURCES OF ELECTROMAGNETIC RADIATION

- EM energy at the Earth's surface is also determined by the following:
 - Distance traveled through the atmosphere (Altitude and “Middle of Day”)
 - *UVA actually changes little in intensity throughout the day*
 - Scattering by atmospheric molecules
 - Scattering by water droplets (clouds)
 - can decrease UV by 10-80%
 - Surface reflection (snow, water)

PHOTOBIOLOGY

- For radiation to produce an effect it must first be absorbed by a chromophore.
- This absorption elicits photochemical and photobiological responses.
- Chromophores exist in their lowest energy state (Ground State).
- The energy necessary to raise a certain molecule from its ground state to its excited state is precisely determined by its structure.
- There is a set energy (or range of energies in more complex systems) that a molecule will absorb.
- The wavelength of the photon corresponding to that energy determines the molecule's absorption spectrum.

PHOTOBIOLOGY

- *Absorption Maxima*: Wavelengths that have the highest probability of absorption for a specific chromophore.
 - Examples:
 - DNA 260nm (thus UVC most effective)
 - DNA in basal layer 300 nm
 - Urocanic acid 280nm
 - Aromatic amino acids 280-290nm
 - Hemoglobin 410nm
 - Porphyrins 400-420nm
 - Beta-Carotene 460nm
 - ***Melanin absorbs throughout the UV and visible spectrum and does NOT have a distinct Absorption maxima*

What is the Soret band?

Peak absorption of porphyrins at 400-410 nm region

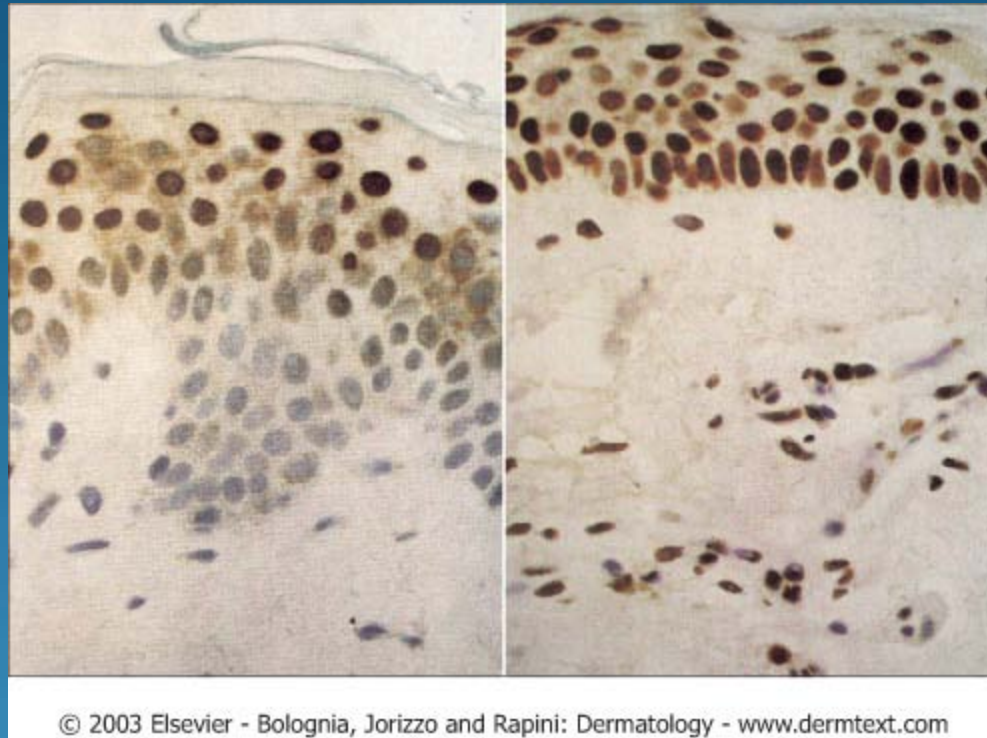


Fig. 86.8 A wavelength of 300 nm is more effective than one of 290 nm in inducing thymine dimers in the basal layer of the human epidermis. After irradiation of human skin with monochromatic 290 nm UVB (2 MED) and staining with anti-thymine dimer antibodies, most cells in the basal layer show only blue counterstaining, while suprabasal layers demonstrate pronounced reactivity. In contrast, with 2 MED of monochromatic 300 nm UVB, a pronounced immunostaining is also evident in the basal layer of the epidermis. (Reproduced with permission from Young AR, Chadwick CA, Harrison GI, et al. The similarity of action spectra for thymine dimers in human epidermis and erythema suggests that DNA is the chromophore for erythema. *J Invest Dermatol.* 1998;111:982–8.)

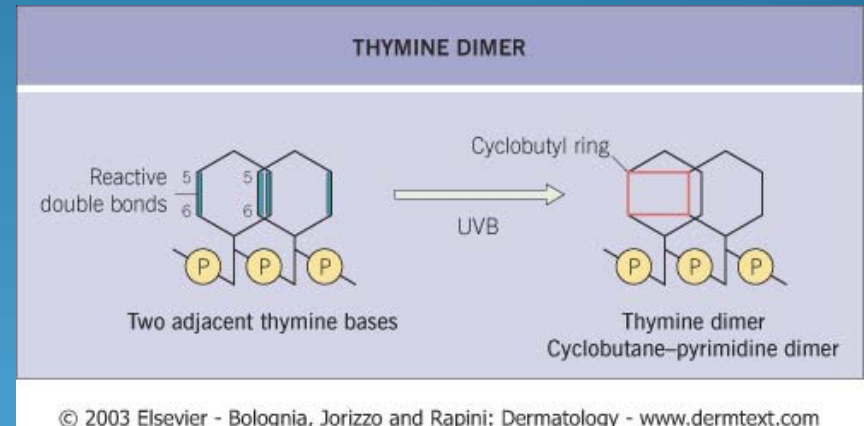
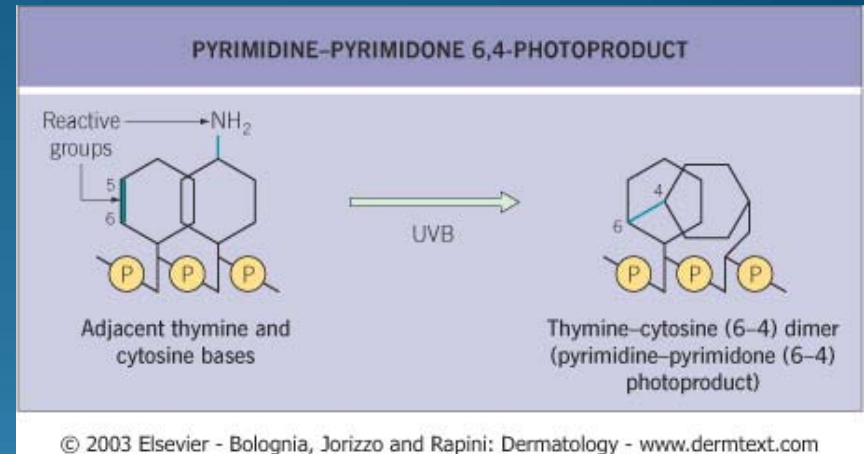
PHOTOBIOLOGY

- Upon absorption of the radiation's energy, the chromophore is elevated to an excited state.
- The excited state is unstable.
- The molecule must return to its ground state by releasing absorbed energy.
- This can be accomplished by:
 - FLUORESCENCE (Emission of light)
 - GENERATION OF HEAT
 - CONVERSION TO CHEMICAL ENERGY (PHOTOCHEMICAL REACTION)
- These reactions require energies found only at $<750\text{nm}$.
- Ensuing photochemical reactions may either lead to photoproducts by
 - Changing the chromophore directly or
 - Through energy transfer indirectly change a molecule other than the chromophore.

PHOTOBIOLOGY

- Different wavelengths of UV light induce different types of DNA damage.
- UVC and UVB are capable of exciting the DNA molecule directly.
 - DNA is regarded as the chromophore for most of the biological effects of UVB and UVC
 - These include erythema, tanning, immunosuppression, mutagenesis, and carcinogenesis.

- Photoproducts lead in a stepwise fashion to
 - Biochemical reactions
 - Cellular changes
 - Observable organ (skin) responses
- Examples of photoproducts:
 - Pyrimidine dimers (T-C, T-T, C-C)
 - *Cyclobutane Dimer is most common*
 - T-T is the most common
 - Followed by C-T and T-C
 - Then C-C
 - 6-4 Pyrimidine-Pyrimidone 2nd most common
 - T-C is the most common
 - C-C and T-T are also observed
 - Free radicals
 - Oxidized lipids
 - Pre-Vitamin D₃

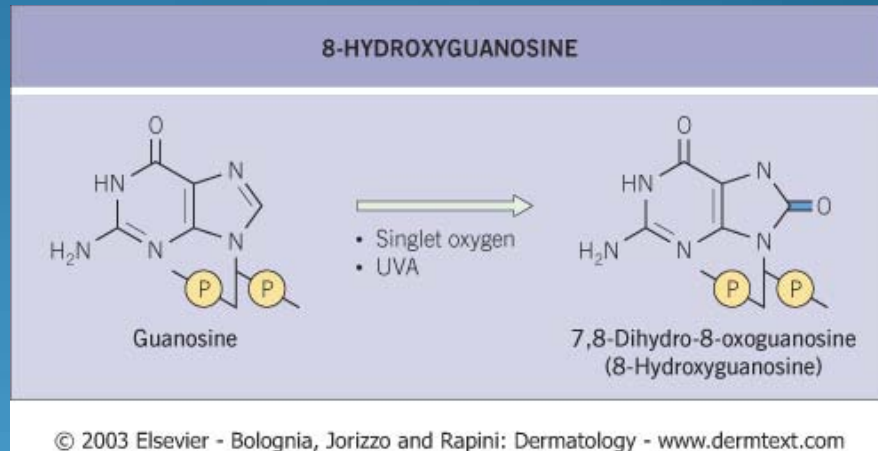


PHOTOBIOLOGY

- Photoproducts can also be from exogenous chromophores that can lead to photosensitizing reactions (toxic and allergic)
 - This is done indirectly by transfer of energy to
 - DNA (type I photosensitized reaction) or
 - To molecular oxygen, with reactive oxygen species in turn being able to damage DNA (type II photosensitized reaction).
 - This indirect generation of DNA damage is relevant to UVA.
 - UVA is barely able to excite the DNA molecule directly thus rarely produces pyrimidine dimers.
 - Many of the biological properties of UVA is strictly dependent upon oxygen.
 - UVA has been shown to be responsible for almost all guanine oxidation products in DNA.
 - We take advantage of this every day with PUVA treatments.

PHOTOBIOLOGY

- UV induced reactive oxygen species include singlet oxygen, hydrogen peroxide, and superoxide radical.
- Singlet oxygen reacts predominantly with guanine and generates 8-Hydroxyguanosine.
- UVA still produces few pyrimidine dimers.
- Raises a question of debate—are the mutagenic properties of UVA (particularly UVA₁) really mediated by oxidative DNA damage or by the weak ability to form a few pyrimidine dimers?



PHOTOBIOLOGY

- Example using Sunburn
 - Nucleic Acids (chromophores) absorb UV light and thymidine dimers (photoproducts) are formed
 - This leads to
 - inhibition of DNA (biochemical reaction)
 - cell death (cellular change)
 - desquamation of the epidermis (organ response)
- In order for absorption to occur, the EM energy must penetrate the epidermis and dermis.
- The depth of penetration depends on the wavelength.

Repair of UV-induced DNA Damage

- UV induced DNA damage requires excision and replacement of damaged nucleotides by DNA repair pathways.
- DNA photoproducts are mutagenic.
- They can be repaired by nucleotide excision repair (NER) pathway.
- A defect in this pathway (XP) increases UV sensitivity and cancers.
- XP includes seven genetic complementation groups (XPA-XPG).
- These represent different proteins in the NER pathway.
- There is no backup pathway for NER.

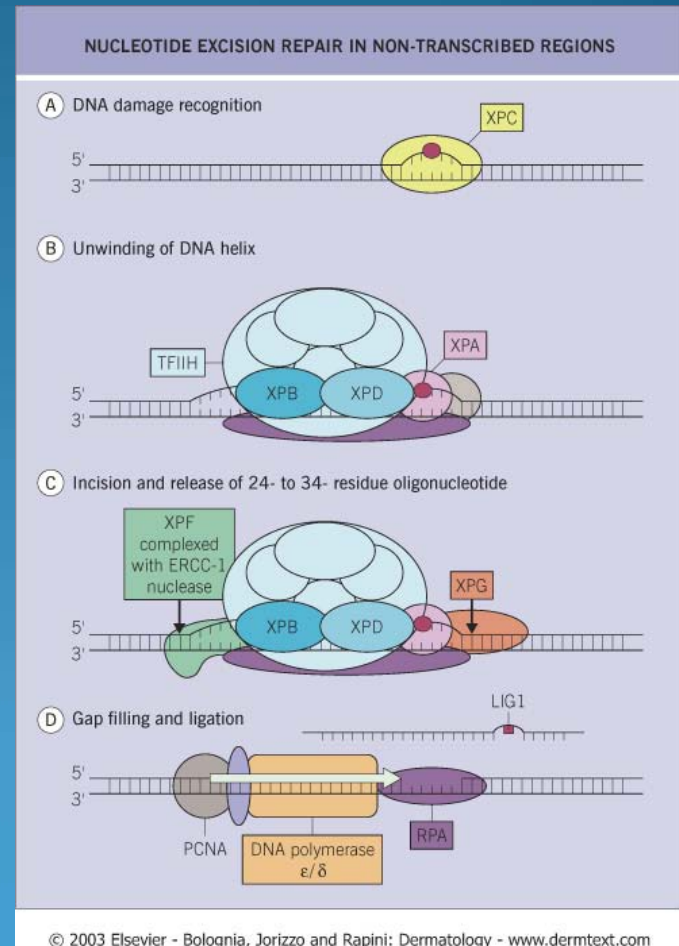
Table 86.1 Deficient DNA repair genes. Deficient DNA repair genes in xeroderma pigmentosum (XP) complementation groups XPA to XPG, trichothiodystrophy (TDD), Cockayne syndrome (CS), and xeroderma pigmentosum variant (ERCC1, excision repair cross complementing gene 1; NER, nucleotide excision repair; TFIIH, transcription factor IIH).

DEFICIENT DNA REPAIR GENES		
Complementation group or inherited disorder	Function of affected gene product in DNA repair	Also associated with
Nucleotide excision repair		
XPA	High affinity for injured DNA (single strands) Has many interactions with other NER proteins Might have a role in assembling the DNA repair machinery around the DNA lesion	–
XPB	Subunit of the transcription factor TFIIH Unwinds the DNA helix around the DNA lesion with its 3'→5' helicase function	XP/CS ^a , TTD
XPC	DNA damage recognition Only required for global genome repair, not for transcription-coupled repair	–
XPD	Subunit of the transcription factor TFIIH like XPB Unwinds the DNA helix around the DNA lesion with its 5'→3' helicase function	XP/CS ^a , TTD
XPE	Affinity for UV-damaged DNA Might have an auxiliary role in DNA damage recognition	–
XPF	5'-repair endonuclease The ERCC1/XPF complex cuts at the single-strand to double-strand transition 5' of the DNA lesion	–
XPG	3'-repair endonuclease Cuts at the single-strand to double-strand transition 3' of the DNA lesion	XP/CS ^a
TTD	Possibly another subunit of TFIIH See XPB and XPD	–
CSA	Only required for transcription-coupled repair Might be necessary for dissociation of RNA polymerase upon stalling at DNA lesion	–
CSB	Only required for transcription-coupled repair Might be necessary for dissociation of RNA polymerase upon stalling at DNA lesion	–
Translesional DNA synthesis		
XP variant	DNA polymerase-η Bypasses T–T dimers with correct insertion of two A residues	–

^aXeroderma pigmentosum/Cockayne syndrome overlap syndrome.

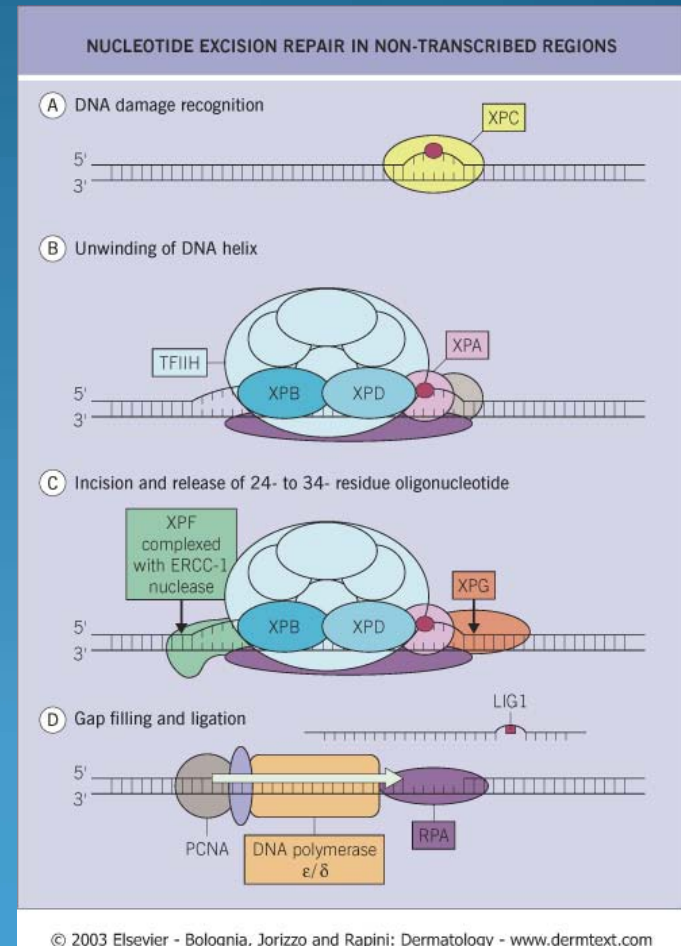
Repair of UV-induced DNA Damage

- Nucleotide excision repair involves
 - Recognition of DNA damage
 - Unwinding of DNA helix
 - Incision of the DNA strand containing a lesion
 - DNA synthesis and ligation to replace an excised oligonucleotide



Repair of UV-induced DNA Damage

- DNA damage recognition requires that the DNA photoproduct distort the DNA helix.
- A key intermediate is an open, unwound structure formed around a DNA lesion in a reaction that uses the helicase activities of XPB and XPD
- This creates sites for cutting by the endonucleases XPG on the 3' side and the XPF-ERCC1 complex on the 5' side
- The oligonucleotide is released.
- The gap is filled by DNA polymerase delta or sigma.
- It is sealed by DNA ligase 1.



What other 2 diseases are defects in NER found?

1. Cockayne Syndrome
2. Photosensitive form of brittle hair syndrome
trichothiodystrophy

Base Excision Repair (BER)

- Repairs oxidative DNA base modifications.
- Initial step is removal of a base rather than a nucleotide.
- This step is carried out by a DNA glycosylase.
- Human DNA glycosylase 8-oxoG DNA glycosylase 1 (hOGG-1) repairs 8-Hydroxyguanosine.
- Loss of this enzyme leads to cellular hypersensitivity to UVA, not UVB.
- The backup for BER is DNA glycosylase MYH.
- This enzyme removes misincorporated A residues opposite 8-hydroxyguanosine.

OPTICAL PROPERTIES OF THE SKIN

- When radiation strikes the skin part is
 - remitted (reflected and scattered)
 - Absorbed
 - transmitted inwards
- Very small fraction is re-emitted as fluorescence.
- 5-10% of incident light is reflected by the outer surface of the Stratum Corneum.
- This is dependent on the angle of incidence.
- The real reason for our perception of skin color is actually the result of back-scattering from within the dermis
 - Caucasian skin remits 50% of visible light in this way
 - Melanin acts as a filter to *prevent dermal remittance*
 - Blood in the dermis absorbs blue and green wavelengths (but not red)

OPTICAL PROPERTIES OF THE SKIN

- White stratum corneum also transmits more radiance to the deeper layers.
- This increases the susceptibility to actinic damage.
- Things such as scale on the skin surface cause increased surface scattering of light.
 - This is why patient's with psoriasis should apply a thin layer of Vaseline before PUVA treatments

NATURAL DEFENSES OF THE SKIN

- Stratum corneum with its melanin content plays a major factor in skin protection.
- Thicker areas (palms/soles) rarely burn and are also difficult areas to treat with light therapy.
- Amelanotic skin thickens in response to UVB radiation.
- Melanin
 - Protects by absorbing energy (chromophore) and by acting as a free radical scavenger
 - *Melanin actually exists in human skin as a stable free radical*
- Constitutive (Baseline color; more protective than Facultative)
- Facultative (ability to tan in response to UV exposure)
- Beta-Carotene
 - Believed to work as free-radical scavenger
 - NO photoprotective role in UVB spectrum (doesn't work as sunscreen)
 - Used in treatment of EPP

UV RADIATION AND THE IMMUNE SYSTEM

- UV Radiation has long been known to effect the immune system
- The effects are extremely complicated and highlight the complexity involved in mounting an immune response.
- There seems to be no single definitive primary action.
- The effects of UV Radiation can be divided into
 - *Cellular Effects*
 - *Molecular Effects*

UV RADIATION AND THE IMMUNE SYSTEM

- Cellular Effects:
 - UV Radiation has been shown to decrease Langerhan's cell appearance, function, and absolute numbers.
 - Also changes the primary antigen presenting cell type to macrophages which are less efficient.
 - Darker skin suffers less Langerhan's cell depletion and returns to baseline more quickly than Caucasian skin.
 - All of the above lead to diminished delayed-type hypersensitivity reactions in UV exposed skin.

UV RADIATION AND THE IMMUNE SYSTEM

- Cellular Effects

- UVR causes a decrease in CD4 cells and a slight increase in CD8 cells.
 - This has not been absolutely proven and the significance is unknown
- IL-10 production by melanocytes and activated macrophages on exposure to UVL may bias towards a Th2 type of local immune response.

- Molecular Effects

- Isomerization of trans to cis-Urocanic acid can suppress NK-cell activity in a dose-dependent manner.
- DNA damage also plays a role in immunosuppression

SUNSCREEN AND QUICK TANNING LOTIONS

- Sunscreens are especially important for Fitzpatrick Skin Types I, II, and III
- Two Types are available
 - Chemical or Organic
 - Absorb UV Radiation
 - Physical or Inorganic
 - Reflect & Scatter UV Radiation
- *Products may also have mixtures of these two types*

SUNSCREEN AND QUICK TANNING LOTIONS

- SPF
 - MED Sunscreen Protected / MED
 - *This is a measure of UVB protection only*
- There is no set standard or agreement on how to best measure and label for UVA protection
- Suggested methods include
 - measuring IPD (Immediate pigment darkening)
 - PPD (Persistent pigment darkening)
 - PFA (Protection Factor A which measures MED much like SPF)
- Many now recommend using SPF 30 since almost no one applies the correct amount of sunscreen.
- SPF determined using 2mg/cm².
- Most apply less than ½ this amount.

SUNSCREEN AND QUICK TANNING LOTIONS

- New AAD Labeling:
 - SPF 30 is maximum
 - may use 30+ if over 30
 - Extended wear claims or use of terms such as “All Day Protection” not permitted
- Water Resistant
 - Maintains SPF after 40 minutes in water immersion
- -Very Water Resistant
 - Maintains SPF after 80 minutes
- These new regulations also include very specific standards for SPF determination.

SUNSCREEN AND QUICK TANNING LOTIONS

- Sunscreen in Children <6 months old
 - Current recommendations discourage use in this age group but there is no evidence supporting this.
 - Common sense argues that children should be protected with clothing and sun avoidance.
- Quick Tanners:
 - *Dihydroxyacetone and Lawsone*
 - Auto-oxidizers that bind the S. corneum
 - *Tyrosine rich compounds:*
 - Diffuse into the skin and increase the *in vivo* rate of melanization via tyrosinase.
 - These have little to no photoprotective value.

SUNSCREEN	SPECTRAL RANGE
PABA	UVB
Octyl dimethyl PABA	UVB
Octyl salicylate/ salicylates	UVB
Homomenthyl salicylate	UVB
Ethyl hexyl p-methoxyl cinnamate/cinnamates	UVB
Methyl anthranilate	UVA
Dioxybenzone	UVA
Sulisobenzene	UVA
Oxybenzone	UVA
Butylmethoxydibenzoyl methane (avobenzene or Parsol 1789)	UVA
ZnO, TiO ₂ (absorption, reflection, and scatter of UVA and UVB)	UVA, UVB
Camphor derivatives	UVB
Red veterinary petrolatum	UVA

PHOTOMEDICINE

- Until the 1970's UV treatment (only broadband UVB) was mainly confined to the management of psoriasis and acne
- Now many treatment modalities are available for the treatment of over 40 skin diseases
- *Broadband UVB Therapy:*
 - Energy usually from “sunlamp” fluorescent bulbs that emit a significant amount of
 - UVC
 - all wavelengths of UVB
 - large amount of UVA
 - visible light

UVB

- Broadband UVB Therapy

- Fairly safe and simple if used properly.
- Psoriasis is main indication with ~70% of patients cleared within 30 treatments.
- Particularly good for guttate type.
- Also used for mild-moderate atopic eczema (works poorly in severe disease).
- The only quantitative study in acne actually showed no benefit!
- Limited by poor penetration; therefore ineffective for thick plaques or palms/soles.
- Frequently used in combination with tar (Goeckerman Method).
- Dose calculated by determining MED (Europe).
- Skin type (American) and then increasing in conservative increments.

UVB

- Broad Band UVB
 - Usually 3-5 treatments per week to equal 25-30 total.
 - Always apply emollient for optical effects.
 - Most patients need at least weekly maintenance treatments.
 - Avoid topical steroids
 - these actually reduce the duration of remission
 - Review
 - UVB is absorbed by endogenous chromophores.
 - Photochemical reactions mediate a variety of biological effects.
 - This leads to therapeutic effects.

UVB

- The most important chromophore is DNA.
- This causes the formation of pyrimidine dimers.
- UVB exposure reduces DNA synthesis.
- This suppresses the accelerated DNA synthesis found in psoriatic epidermal cells.
- It also induces p53 leading to cell cycle arrest or apoptosis of keratinocytes (sunburn cells) if DNA damage is too severe.
- P53 thus prevents photocarcinogenesis by this mechanism.
- UVB induces the release of prostaglandins and cytokines.
- IL 10 is important in immune suppression.
- Langerhan's cells decreased.
- Adverse effects
 - Erythema
 - long-term photodamage

Table 134.4 Differences between the US and European protocols.

DIFFERENCES BETWEEN THE US AND EUROPEAN PROTOCOLS

	US	Europe
UVA dosimetry	Predetermined dose according to skin phototype	Individualized dose according to MPD determination
Frequency of treatment	Two to three times/week	Four times/week
Dose increments	Predetermined	Individualized
Principle of approach	Regimented, cautious	Flexible, aggressive
Goal	To clear without ponderous testing and acute side effects	To clear rapidly before maximum pigmentation develops

UVB

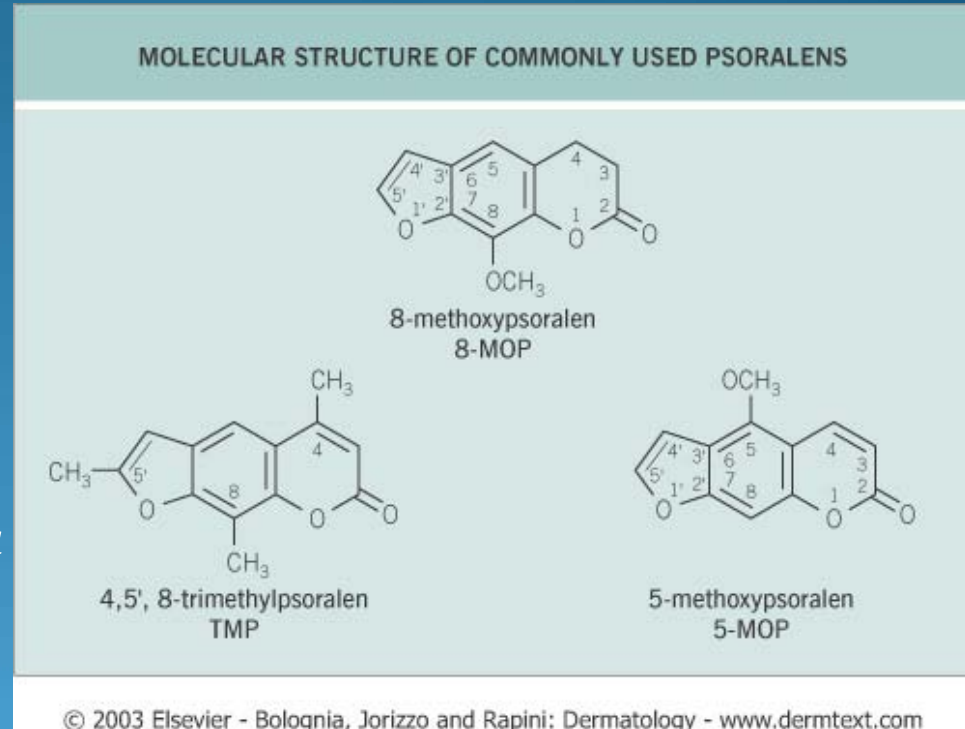
- • *Narrowband UVB (313nm)*
 - Newer treatment that takes advantage of the more therapeutic and less erythemogenic wavelengths of UVB spectrum.
 - Unfortunately the bulb (Philips TLo1) is an inch longer than standard UVB, so a dedicated unit is needed.
 - Definitely superior to standard UVB.
 - Better clearance and fewer treatments required.
 - One study showed NB-UVB to be equivalent to PUVA.
 - Also better for severe atopic eczema.

UVB

- Narrow Band UVB
 - Penetration is still an issue with NB-UVB.
 - Technique essentially same for standard UVB.
 - Adverse effects also similar.
 - NB-UVB has been shown to be more carcinogenic in mice, but this risk may be offset by its greater efficacy.

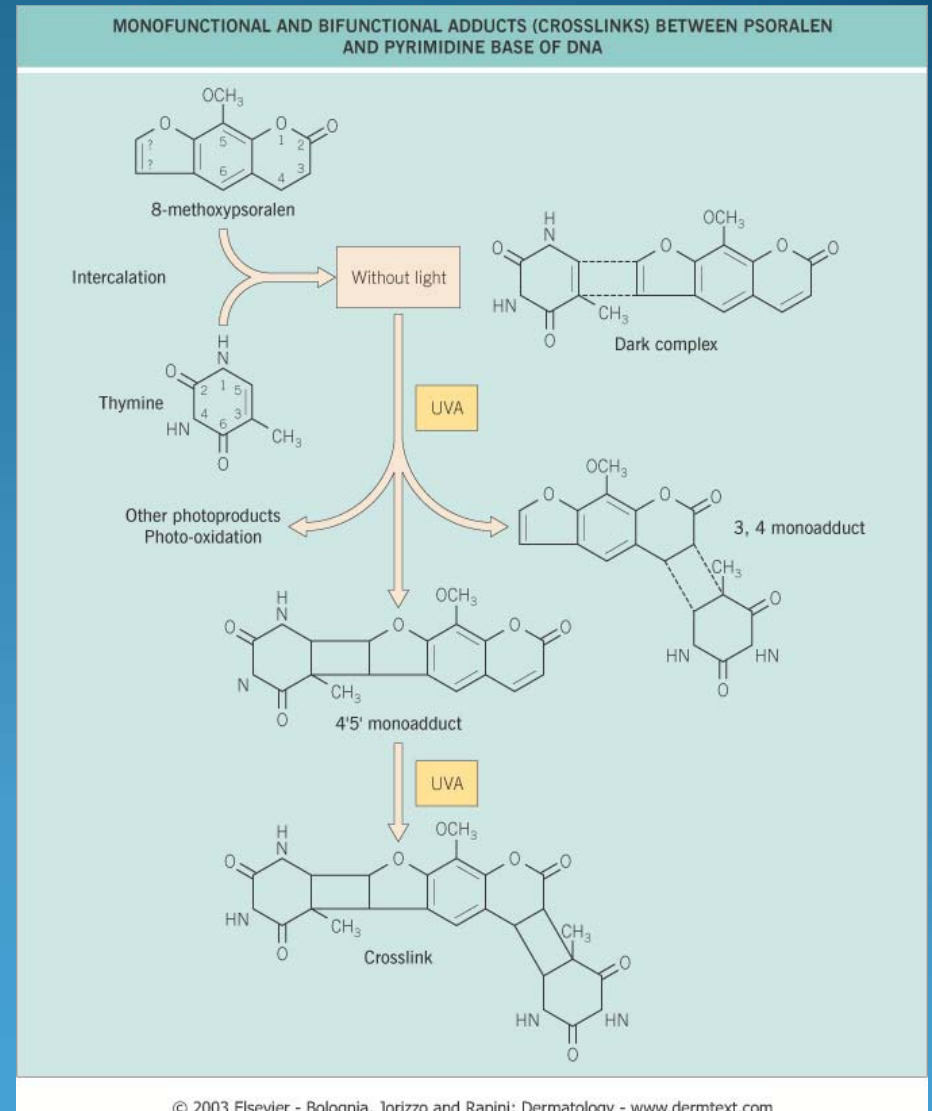
PUVA

- *PUVA (Psoralens & UVA)*
 - Available in both oral and topical forms.
 - Psoralens are naturally occurring linear furocoumarins.
 - 8-methoxypsoralen is used primarily.
 - Get GI intolerance
 - 5-MOP is less erythemogenic and not associated with GI intolerance.
 - TMP is less phototoxic than 8-MOP



PUVA

- Psoralens react with DNA in three steps
 - Psoralen intercalates into the DNA double strand.
 - UVA results in formation of 3,4 or 4',5' cyclobutane monoadduct with pyrimidine bases of native DNA.
 - This monoadduct can absorb second photon.
 - This leads to formation of interstrand cross link of the double helix.



PUVA

- Excited psoralens can also react with molecular oxygen.
- This causes cell membrane damage by lipid peroxidation.
- These reactions inhibit DNA replication and cause cell cycle arrest.
- PUVA is far more potent in inducing apoptosis in lymphocytes than in keratinocytes.
- This may explain efficacy in CTCL.
- “Gold Standard” for moderate to severe psoriasis.
- Produces more than 90% clearing within 30 treatments.
- Also induces longer remissions and requires fewer and less frequent maintenance treatments

PUVA

- PUVA
 - Adverse effects
 - Erythema
 - Photoaging (Lentigines)
 - Carcinogenicity
 - SCC highest risk
 - Melanoma
 - BCC
 - Ocular damage (?cataracts) are the main concerns
 - RePUVA
 - Combination of UVA and oral Retinoids
 - Benefit of quicker response and fewer total treatments required.

Table 134.2 PUVA-responsive diseases. †Experience is limited to a small number of patients. *May flare.

PUVA-RESPONSIVE DISEASES

Therapy of disease

Psoriasis
 Palmoplantar pustulosis
 Atopic dermatitis
 Mycosis fungoides (stages IA, IB, IIA)
 Vitiligo
 Generalized lichen planus
 Urticaria pigmentosa
 Cutaneous graft-versus-host disease
 Generalized granuloma annulare
 Prurigo nodularis
 Pityriasis lichenoides (acute and chronic)†
 Lymphomatoid papulosis†
 Pityriasis rubra pilaris†,*
 Purpura pigmentosa chronica†
 Langerhans cell histiocytosis†
 Dermatitis herpetiformis†
 Localized scleroderma†

Prevention of disease

Polymorphous light eruption
 Solar urticaria
 Chronic actinic dermatitis†
 Hydroa vacciniforme†
 Erythropoietic protoporphyria†

UVA1

- *UVA1 (320-400nm)*:
 - Takes advantage of less erythemogenic UVA spectrum.
 - Available in high and low dose protocols.
 - No reported adverse effects.
 - Carcinogenicity is still a concern.
 - Not yet available in the US.