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DRAFT

NATIONAL INSTITUTES OF HEALTH

CONSENSUS DEVELOPMENT CONFERENCE STATEMENT

SUNLIGHT, ULTRAVIOLET RADIATION, AND THE SKIN

May 8-10, 1989

INTRODUCTION

It is ingrained in humans to love light and, indeed, since mankind's first wanderings from the caves, worship of the sun has been a fundamental tenet that many societies hold even to the present.

The properties of the sun that have inspired such reverence include its light (visible radiation) and its warmth (infrared radiation). Additional portions of the solar spectrum that cannot be perceived directly by the senses (ultraviolet) are capable of evoking both physiologic and pathologic events in the skin.

Sunlight is the ultimate source of energy and is vitally important to life as we know it. However, absorption of incident solar energy by chemicals in the skin can cause a variety of pathological sequelae.

Until the 20th century, the sun was the predominant source of human skin exposure to energy within the photobiologic action spectrum. More recently, artificial devices capable of mimicking some ~~or all~~ of the solar spectrum have been introduced, compounding the opportunities and risks of exposure.

Despite the undeniable importance of cutaneous exposure to ultraviolet radiation (UVR) for vitamin D homeostasis, there is little evidence to indicate that there are additional healthful effects of such exposure. Indeed, overwhelming evidence exists to support the concept that the skin

Attachment 2

is damaged in many different ways by its direct exposure to natural or artificial UVR. Some exposure is virtually unavoidable over a lifetime and is dramatically altered in different populations depending upon climate, geography, occupation, and recreational activities. The consequences of this exposure are also determined by individual factors such as melanin pigmentation. The Effects can be divided into two general types, acute and chronic. Acute effects include sunburn, and chronic effects include, among others, the development of certain forms of skin cancer. In addition, the skin is a major site of immunologic activity, and UVR is capable of affecting the immune system. The skin is also vulnerable to degenerative changes evoked by chronic UVR. These changes are a major component of the constellation of physical changes perceived as aging but in reality are due to chronic photodamage.

It is now possible to measure the effects of solar radiation on the skin, and epidemiologic studies from around the world have provided important new knowledge concerning the risks and benefits of exposure to sunlight and UVR.

Expanding knowledge about the hazards of sunlight and UVR has been accompanied by improved approaches to photoprotection, [redacted] In addition, there is preliminary evidence suggesting that the retinoids may be capable of inhibiting the development of or indeed reversing certain chronic effects of cutaneous sun exposure.

Considerable controversy remains concerning the specific adverse effects caused by various wavelengths of UVR, the magnitude of the adverse effects, and potential strategies for their prevention and/or treatment. A

Consensus Development Conference was undertaken in an effort to define the specific interactions of sunlight, UVR, and the skin and to identify methods for preventing and/or treating the adverse effects of UVR. Sponsored by the National Institute of Arthritis and Musculoskeletal and Skin Diseases, the Office of Medical Applications of Research, the National Cancer Institute, and the National Institute of Child Health and Human Development of the National Institutes of Health, the Food and Drug Administration, and the Environmental Protection Agency, the conference brought together physicians, scientists, and other health care professionals, along with representatives of the public on May 8-10, 1989. Following 1-1/2 days of presentations and discussions by the invited experts and the audience, members of the consensus panel drawn from the biomedical research community and the public weighed the scientific evidence in formulating a draft statement in response to several questions:

- What are the sources of the ultraviolet radiation, and is the extent of human exposure changing over time?
- What are the effects of sunlight on the skin?
- What factors influence susceptibility to ultraviolet radiation?
- Can ultraviolet-induced changes be prevented? If so, how?
- Are sunlight-induced adverse skin alterations treatable and/or reversible? If so, how?
- What are the directions for future research?

In applying the recommendations of this consensus conference, it is important to recognize that special circumstances may exist for each patient. These may include unavoidable exposures to UVR or the inability

to use certain of the preventive strategies. There are clearly some areas in which final recommendations cannot yet be made due to insufficient data. In these situations, physicians must use their best clinical judgment in advising patients.

1. WHAT ARE THE SOURCES OF ULTRAVIOLET RADIATION, AND IS THE EXTENT OF HUMAN EXPOSURE CHANGING OVER TIME?

There are both natural and artificial sources of UVR. Although there are many artificial sources of this energy, sunlight is the only natural source.

The sun emits a wide variety of electromagnetic radiation, including infrared, visible, and ultraviolet A (UVA;320 to 400 nm), ultraviolet B (UVB;290 to 320 nm), and ultraviolet C (UVC;200 to 290 nm). The only UVR that reaches the Earth's surface are UVA and UVB. UVA radiation is 1,000-fold less effective than UVB in producing erythema. However, its predominance in the solar energy reaching the Earth's surface (tenfold to hundredfold more than UVB) permits UVA to play a far more important role in contributing to the harmful effects of sun exposure than previously suspected.

Sunlight is by far the greatest source of human UVR exposure, affecting virtually everyone. The extent of an individual's exposure, however, varies widely depending on a variety of factors such as occupation, lifestyle, age, altitude, latitude, and attire. There is greater UVR exposure with decreasing latitude. UVR exposure increases with a concomitant decrease in ozone. Further, residing at highest altitude results in a greater UVR exposure. For every 1,000 feet above sea level,

there is a compounded 4-percent increase in UVR exposure. Other factors that can influence exposure include heat, wind, humidity, pollutants, cloud cover, snow, season, and time of day.

Solar flares (sunspots) also influence the amount of UVR reaching the Earth. Solar flares increase ozone concentration in the stratosphere (above 50 km) thereby reducing the amount of surface UVB. In periods of inactivity of solar flares, there is a decrease in the ozone concentration, allowing increased UVB to penetrate to the Earth's surface. This 11-year cycle of solar flares causes as much as 400-percent variation in UVB at 300 nm.

There is serious concern about depletion of the stratospheric ozone by manmade chlorofluorocarbons (CFC). These extraordinarily inert chemicals are used in numerous products, including aerosols and refrigerants. The U.S. Environmental Protection Agency is charged with estimating the health effects associated with changes in stratospheric ozone levels. In a recent risk assessment document, the Agency predicted that without controls on CFC production, there would be a 40-percent depletion of ozone by the year 2075. The Agency further concluded that for every 1-percent change in ozone, there will be compounded a 2-percent increase in UVB levels at the Earth's surface.* Such a rise is predicted to result in an additional 1- to 3-percent increase per year in nonmelanoma skin cancers (NMSC).

Recent satellite measurements already indicate a worldwide 16-percent decrease in stratospheric ozone over the last decade. Both satellite- and land-based measurements have revealed a seasonal hole in the ozone over the Antarctic secondary to its destruction by CFC's. Although increased surface UVB has

* This change will all occur in the more damaging short ~~[illegible]~~ ~~the UVB radiation which [illegible]~~ ~~[illegible]~~ problems.

been measured in the Antarctic, there has not yet been a measurable change in UVB in the United States.

Over the past several decades, the average American's exposure to UVB has increased considerably due to changing lifestyles: more outdoor activities, more emphasis on tanning, scantier clothing, and a population shift to the sunbelt, and a increase of fluorescent light fixtures.

The most common sources of artificial UVR exposure are various kinds of lamps that emit this form of energy. These lamps are used primarily for recreational tanning and phototherapy of skin diseases (e.g., psoriasis and mycosis fungoides). UVR lamps can emit UVA, UVB, and/or UVC. Those lamps currently used for recreational tanning emit primarily or exclusively UVA. However, contamination with UVB remains a problem with most of these sources. Even 1 percent UVB emission can cause a significant increase in the potential for skin cancer. Some UVA lamps generate more than 5 times more UVA per unit time than solar UVA radiation reaching the Earth's surface at the Equator. At these doses, even "pure UVA" is likely to have adverse biologic effects.

The tanning industry is rapidly growing in the United States. Currently, more than 1 million Americans use commercial tanning facilities every day. The biggest categories of users are adolescents and young adults, especially women.

The use of artificial ultraviolet sources for the phototherapy of dermatologic diseases has increased substantially in recent years. This has exposed a group of people to markedly increased doses of UVR. Epidemiologic studies of these patients have shown an unequivocal dose-

dependent increase in the incidence of NMSC, especially squamous cell carcinoma (SCC).

Another potential but as yet ~~undefined~~ unexplored source of artificial UVR is unshielded fluorescent bulbs used for illumination. An unresolved issue is the amount of UVA emitted and the long-term effects of this exposure.

2. WHAT ARE THE EFFECTS OF SUNLIGHT ON THE SKIN?

Marked morphologic changes in all regions of the skin, except perhaps the subcutaneous tissue, are recognized as consequences of exposure to UVR. These changes underlie the clinically observed sagging, wrinkling, leathery texture, and blotchy discoloration of skin typically associated with actinic damage. It is unclear how much exposure (over time) is required to effect these changes although it is evident that clinically normal appearing skin can show pathologic signs of sun damage upon histologic and ultrastructural examination. It is known that individuals with fair complexions are more susceptible to this damage.

In the epidermis, UVR-induced changes include aberrant tissue architecture and alterations in keratinocytes, melanocytes, and Langerhans cells. The epidermis becomes thickened as much as twofold, disorganized, and shows evidence of hyperkeratosis, parakeratosis, and acanthosis. Keratinocytes lose their typical alignment and progressive flattening, show inclusions in the nucleus, and accumulate excessive amounts of melanosome complexes above the nucleus (capping). At the ultrastructural level, clumped keratin filaments and alterations in electron density of some of the basal cells are characteristic. Keratinocytes of the more differentiated

epidermal layers (upper spinous, granular, and cornified) show few, if any, cytologic changes.

In spite of evidence for morphologic change, there are no data to support alterations in keratinocyte differentiation. Furthermore, it is not known how UVR interactions with light-absorbing molecules within the keratinocytes (e.g., DNA, keratins, lipids) correlate with the changes in morphology.

Two other cells of the epidermis are also affected by UVR. The melanocyte, with its melanin pigment-containing melanosomes, is the primary cell involved in photoprotection of the skin. In sun-damaged epidermis, they enlarge, increase in number, and migrate to higher levels of the epidermis. UVR also affects Langerhans cells in both animal and human skin by altering their immunologic function. Even low doses of UVB can reduce the antigen-presenting capability of these cells, block the normal effector pathway, and evoke an inappropriate response by activating the T suppressor pathway. It is unclear whether UVR affects Langerhans cells directly or indirectly through soluble factors released by damaged keratinocytes.

The dermal-epidermal junction loses its rete ridges forming a flattened interface between the epidermis and dermis. This kind of abutment is more susceptible to shearing forces than the normal interlocked system of epidermal rete ~~pegs~~ ridges and dermal papillae. At the ultrastructural level, regions of reduplicated lamina densa are evident. This change is not unique to photodamage but is characteristic of trauma to the epidermis by wounding and/or by disease.

UVR causes unique dermal damage: alterations in architecture, matrix composition, vascular structure and function, and cellular activities. The

connective tissue immediately beneath the epidermis (Grenz Zone) contains large bundles of densely packed, normal-appearing collagen fibrils. Beneath this region, a broad zone of electron-dense elastotic material is evident. There are no data that demonstrate how newly synthesized or degraded, previously existing elastic fibers contribute to this material. Abnormal collagen fibrils can be admixed with the elastotic substance. Other studies show changes in the type III:I collagen ratio and an increase in glycosaminoglycans. Fibroblasts appear to be metabolically active. It is not clear whether this is a transient response to the UVR or whether there is a change in cell phenotype that can be retained in vitro. The mechanisms for the altered connective tissue responses are not understood.

Dermal vessels become dilated, leaky, and accumulate excessive basement membrane-like material. Inflammatory cells collect around the vessels; mast cells are increased in number and may show evidence of degranulation and apparent physical associations with fibroblasts. Although the nature of this relationship is unknown, it is a common observation in other disorders in which fibrosis may occur.

Sunburn is UVR-induced erythema of the skin caused by vasodilatation of dermal vessels. This may be mediated through cyclo-oxygenase and lipo-oxygenase products of arachidonic acid. Generation of the prostaglandins associated with UVB erythema produced within the first 6 to 12 hours can be blocked by topical nonsteroidal anti-inflammatory agents such as indomethacin. These anti-inflammatory agents, however, cannot inhibit the delayed, post-24-hour erythema that is modulated by lipo-oxygenase products. The time-dependent release of varying mediators during the UV-induced inflammatory process underscores the need for further exploration

into selective inhibitors of both the cyclo-oxygenase and lipoxygenase pathways in the prevention and treatment of sunburn erythema.

Also associated with UVR irradiation of human skin is the appearance of dyskeratotic keratinocytes, known as sunburn cells, in the superficial layers of the epidermis. The mechanisms of the development of these cells are still unclear and warrant further exploration.

Tanning is the term applied to the increase in melanin pigmentation following UVR exposure. It is mediated by a combination of immediate pigment darkening (IPD) and delayed pigment darkening (DPD). IPD is caused by UVA and is due to photooxidation of preformed melanin. It is not protective against UVB erythema. DPD occurs about 72 hours after UVR exposure and protects against UVB erythema and pyrimidine dimer formation. It is accompanied by an increase in the number of DOPA-positive melanocytes, an increase in the number and melanization of melanosomes, and an increase in dendricity of melanocytes. UVR also increases the transfer of melanosomes from melanocytes to keratinocytes. Melanosomes that were diffusely distributed within keratinocytes collect above the nucleus, forming a "cap" over it. DPD occurs with either UVB or UVA. DPD induced by UVB is more protective against UVB erythema than is DPD induced by UVA. Both UVB- and UVA-induced DPD protect equally well against UVB dimer formation.

In addition to certain genetic and metabolic disorders that are precipitated by UVR, there are many photosensitive diseases of unknown cause. These include lupus erythematosus and polymorphous light eruption, which are due to certain wavelengths of the UVR spectrum. Photosensitivity

disorders may also occur due to the interaction of UVR with many commonly used drugs, [illegible] as chemicals used in industry and consumer products.

UVR modifies local and systemic immune responses, alters Langerhans cells, and activates the T cell suppressor pathway. Soluble factors released from UV-irradiated epidermal cells also may be responsible for this altered immune response. In certain experimental systems UVR-induced tumors transplanted into genetically identical animals are normally rejected. ~~However, if host~~ If these host animals are irradiated before transplantation, the tumor will be accepted. These conclusions are based on animal studies. The role of UVR in the immunobiology of human skin cancer and in susceptibility against certain cutaneous infectious diseases is unclear. More studies on the effect of UVR on human neoplastic and infectious disease are warranted.

There is extensive epidemiological evidence supporting the direct role sunlight plays in human skin cancer. Basal cell carcinomas (BCC), the most common skin cancer in Caucasians, are found primarily on sun-exposed areas such as the head and neck in which a dose-response relationship exists. Furthermore, skin cancer patients generally have decreased melanin photoprotection; people with light complexion and ease of sunburning have a higher incidence of tumors. There is even greater evidence for the role of sunlight in causing SCC's. Although both BCC's and SCC's are more prevalent in geographic areas of high sun exposure, there is a much greater increase in SCC with decreasing latitude and increasing sun exposure. A correlation is postulated ~~to exist~~ reasonable correlation exists between sunlight exposure and melanomas, but the relationship is not as clear as with NMSC. It should be emphasized that the incidence of NMSC and melanomas has been steadily increasing. Unlike NMSC, melanomas occur most frequently on the upper back in males and lower

extremities in females. Melanoma incidence does not follow a pattern of risk with cumulative UVR exposure as in NMSC.

Extensive data also exist on UVR-induced skin cancer in experimental animals. In mice and guinea pigs, UVR induces mainly SCC whereas in rats both SCC and BCC are produced by repeated doses of UVR. In general, UVR induces SCC's in mice somewhat more effectively in young animals than in older ones. The cancer response is preceded by photodamage to the epidermal DNA, inflammation, epidermal hyperplasia, and dysplasia. Although there are several animal models in which chemical carcinogens can induce melanomas, the induction of melanomas by UVR has been very difficult. Recent studies suggest that the opossum may be a reasonable model for UVR-induced melanomas.

~~Both epidemiological~~ and animal studies suggest that UVB is more effective than UVA (320 to 400 nm) in causing all types of skin cancer; however, the data are not conclusive. Nevertheless, UVA can induce DNA damage, erythema, and SCC in both pigmented and albino mice and in guinea pigs. Recent evidence suggests that the longer UVA wavelengths (UVA I; 340 to 400 nm) of the UVA spectrum are less damaging than the shorter UVA wavelengths (UVA II; 320 to 340 nm).

The exposure of skin to UVB is essential for the endogenous production of vitamin D_3. In areas of the world where adequate vitamin D in nutrition is not available, it is the only source.

The relationship of sunshine to vitamin D_3 and the normal growth and development of the skeleton is well known. Exposure of skin to UVR in the region of 290 to 315 nm is essential for the formation of vitamin D_3 in the epidermis.

There is evidence that vitamin D_3 synthesis is inhibited by the use of sunscreens. In the United States, this does not represent a health hazard for the pediatric population who receive adequate vitamin D supplementation in milk. Potential deficiencies in elderly populations may exist.

3. WHAT FACTORS INFLUENCE SUSCEPTIBILITY TO ULTRAVIOLET RADIATION?

Susceptibility to damage by UVR may be influenced by genetic and acquired disorders, genetic traits, age-related factors, and the use of some medications.

Genetic abnormalities can increase the susceptibility to UVR damage. These include disorders manifested in utero that may be lifelong or that may appear shortly after birth. The inherited disorders manifested in utero include disorders of keratinization and pigmentation. Severe inherited disorders that express marked susceptibility to UVR in early childhood include xeroderma pigmentosum, Bloom's syndrome, Rothmund-Thomson syndrome, ataxia-telangiectasia, the porphyrias, phenylketonuria, dysplastic nevus syndrome, and the basal cell nevus syndrome.

There are numerous and diverse acquired diseases that manifest increased light susceptibility. Examples include actinic reticuloid, polymorphous light eruption, solar urticaria, hydroa aestivale, hydroa vacciniform, actinic prurigo, lupus erythematosus, dermatomyositis, Darier's disease, and disseminated superficial actinic parakeratosis.

Significant factors related to susceptibility to UVR damage include race, ethnicity, eye and hair color, and the tendency toward formation of freckles and nevi. One approach to categorizing humans in terms of susceptibility to UVR is typing according to history of sunburning and

tanning. Six skin types have been defined. Type I individuals always burn and never tan; type VI individuals always tan and never burn.

The age of an individual may be correlated with a number of factors that influence the susceptibility to UVR. These may include age-related structural differences, behavioral differences (e.g., adolescent risk taking) and, hypothetically, age-related immunological differences.

There are numerous systemic medications that may augment UVR susceptibility. Increased UVR damage may occur with the use of oral antibiotics, antihypertensives, psoralens, immunosuppressive agents, nonsteroidal anti-inflammatory drugs, and myriad other agents. In addition, a number of topical medications may affect sun exposure. These include topical psoralens, tretinoin, and other sensitizing and depigmenting agents.

4. CAN ULTRAVIOLET-INDUCED CHANGES BE PREVENTED? IF SO, HOW?

Skin cancers due to UVR exposure are the most common form of cancer. In 1978, there were more than 500,000 new cases of skin cancer. This is probably a gross underestimate for 1989, because the number of office visits for NMSC has increased more than 50 percent in the past decade while the overall increase in office visits has increased only 11 percent. Therefore, it is imperative to consider ways to minimize deleterious effects of UVR.

What measures can be taken to minimize UVR exposure? There is considerable information that can serve as a basis for developing a policy of "low-risk" behavior.

(*) There are also data which support that a single [illegible] in childhood....

- First, susceptibility to UV damage can be reduced through use of proper clothing constructed of tight woven fabrics, (wide brimmed ~~billed~~ hats) long sleeves, long pants, etc.

- ~~Second, a significant reduction in UVR damage~~ certain types of ~~can be achieved through the proper use of physical and chemical sunscreening products.~~ Maximum Photoprotection is afforded ~~by chemical sunscreens with [illegible] today are appropriate for UVB protection, combination sunscreens that are effective against UVB and UVA are preferable. Waterproof sunscreens should be selected by swimmers and those who perspire sufficiently to wash off nonwaterproof products. Daily use is~~ recommended (during appropriate times of) the year. ~~Sunscreens should be applied 30 minutes before exposure, with frequent reapplication.~~

- Third, one must strive to enhance behavior that minimizes sun exposure. (*) There are data to suggest that 50 percent of an individual's total lifetime UVR exposure occurs by 18 years of age. Therefore, parental education with subsequent direction of the behavior of children is important during childhood. Modified schedules for outdoor activities at school, camp, daycare centers, or beach should be considered wherever possible so as to minimize UVR exposure. The time of day and the time of year have a major impact on the extent of UVR exposure. For example, on a sunny day in June between 10:00 a.m. and 3:00 p.m., fully 60 percent of the UVB solar radiation ~~UVB radiation~~ reaches the Earth's surface. If exposure during this period could be minimized, significant reduction in the number of

arrives during this period.

NMSC would almost certainly occur. Adults and children should limit their exposure during this peak period of UVR.

- Fourth, one must be aware of photosensitizing medications and chemicals because it is known that these can exacerbate the effects of UVR exposure.
- Fifth, ~~and more important~~ the adverse effects of intentional UVR exposure must be considered. All evidence indicates that UVR-induced suntanning, whether from natural or artificial sources, is deleterious to the skin.

This reemphasizes the need for educating the public about all of these factors, consideration of which will show that low-risk strategies are compatible with normal, active lives.

5. ARE SUNLIGHT-INDUCED ADVERSE SKIN ALTERATIONS TREATABLE AND/OR REVERSIBLE? IF SO, HOW?

Sunlight-induced adverse skin alterations include NMSC (BCC & SCC), melanoma, actinic keratoses, as well as textural and pigmentary changes characteristic of chronic photodamage. All of the cancers are treated by standard ~~classical~~ surgical techniques. Precancerous lesions such as the actinic keratoses are treated by topical chemotherapy (e.g., by the use of 5-fluorouracil) and physical methods of superficial skin destruction (e.g., cryosurgery). Various therapies to improve the features of chronic photodamage--scaliness, coarse and fine wrinkling, telangiectasis, and irregular pigmentation--including chemical peels, the topical use of 5-fluorouracil, alpha-hydroxy acids, and retinoic acid have been tried. Although the beneficial cosmetic effects of some of these treatments have received wide publicity, there are insufficient data demonstrating

Long term large sca... for normal people in the general popa are in progress

sustained improvement, reversibility of tissue pathology, or the preservation of normal skin function by those agents. There is no information regarding long-term positive, negative, or toxic effects. Conflicting data exist regarding the prevention/potentiation effect of topical retinoids in the development of UVR-induced skin tumors in animals. There are indications that systemic use of β carotenes & certain retinoids may be beneficial ~~to~~ in prevention of sun damage in people w/ certain disorders.

6. WHAT ARE THE DIRECTIONS FOR FUTURE RESEARCH?

These following recommendations are not ~~arranged~~ according to priority.

- There is a need for updated epidemiologic data concerning NMSC in the United States. Evidence exists to suggest that the incidence of NMSC is increasing, but no new data have been ~~available~~ obtained since 1978. ~~[illegible]~~
- There is a need for more research to define the dynamic action spectra of sunlight and UVR in the pathogenesis of ~~premalignant and malignant lesions~~ cutaneous melanoma and to develop better animal models for the study of this disease. ~~[illegible]~~
- Studies are needed to define more precisely the phenomenon known as "photoaging" of the skin and to compare this with chronologic aging insofar as pathophysiological mechanisms are concerned.
- Studies are needed to better define the immunomodulatory ~~immunosuppressive~~ effect of UVR exposure and its potential role in the development of skin cancer and cutaneous infections.
- The biological effects of UVA radiation require further study, particularly with the increased use of UVB sunscreens and UVA tanning equipment.
- The importance of UVB radiation in vitamin D homeostasis requires additional study; alternatives to sun exposure should be considered as sources of vitamin D.

~~specifically~~ see (*)

~~The [illegible] effects of UVB radiation require further study, particularly [illegible] shorter [illegible] of the UVB [illegible] where [illegible]~~

- New approaches for behavior modification and education are needed to reduce skin exposure to UVR during childhood and adolescence because it is estimated that as much as 50 percent of an individual's total sun exposure occurs by 18 years of age.

- ~~More effective sunscreens and nontoxic anticarcinogenic agents~~ nontoxic should be developed as ~~approaches to diminishing the risk of UVR exposure~~.

- There is a need for more research in the age-related optical properties of skin and acute and chronic cutaneous responses to UVR.

- ~~Stud. to better def. the actual relat of sun exp. ... expos ... ex ...~~

Intervention programs for ~~need to~~ both adults + children need to be tailored to their readiness to ...

add recomm. about research on fluor. light

- The ... ratio of ... ~~...~~ ~~...~~ ~~...~~ ~~...~~

- The effects of sunlight ~~exp.~~ exposure ~~...~~ and ~~...~~ Vitamin D and other cancers, esp. colon cancer, ~~...~~ warrant future investigation.

- There is a need for epid. study of the health of the popul. who use sunscreens that block UVB rad. ...

- Data are needed which quantitatively assess UVR exposure in normal populations at all ages + under various senarios.

CONCLUSIONS AND RECOMMENDATIONS

- Human exposure to UVR from natural sunlight and artificial sources is increasing substantially.
- UVR in sunlight is critical for vitamin D synthesis in the skin. However, it produces a variety of pathologic effects, including sunburn, pigmentary change, immunologic alterations, and neoplasia. A constellation of structural alterations to the epidermis, dermal-epidermal junction, and dermis is uniquely characteristic of photo-damage.
- Factors that influence susceptibility to UVR include genetic abnormalities, skin type, acquired diseases, and medication exposure.
- UV-induced changes can be prevented by the use of proper clothing, appropriate application of physical and/or chemical sunscreens, behavior modification, UVA I lights and awareness of photosensitizing medications.
- Sunlight-induced adverse skin alterations can be treated by standard surgical techniques and superficial destructive modalities. There is insufficient evidence concerning the reversibility of adverse effects by topical and systemic agents.
- There should be better education of the public with regard to the hazards of tanning parlors and regulation of tanning facilities to protect the public against inadvertant injury by UVR. there should be

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Presenters at the conference were:

Paul R. Bergstresser
Structure and Function of Skin: Its Relationship to Ultraviolet Radiation

David L. Correll
The Nature of Solar Radiation and its Spatial and Temporal Variation on the Earth's Surface

Peter M. Farr
Output of Artificial Light Sources

Irene E. Kochevar
Acute Effects of Ultraviolet Radiation on Skin

Robert M. Lavker
Chronic Nonmalignant Effects of Ultraviolet Radiation on Skin

P. Donald Forbes
Ultraviolet Radiation Effects--Malignant Changes

Michael F. Holick
Role of Sunlight for Producing Vitamin D_3 in the Skin

Margaret L. Kripke
Local and Systemic Effects of UV Radiation on the Immune System

John A.H. Lee
Effects of Ultraviolet Radiation on Skin: Modulation by Melanin/Skin Type

Kenneth H. Kraemer
Hereditary Disorders and Sun Sensitivity

Arthur J. Sober
Preadult Sun Exposure and Cancer (Melanoma) Risk

Robert S. Stern
Modulation of Effects of Ultraviolet Radiation Based on Presence of Photosensitivity Disease or Because of Phototherapy

Joseph Scotto
Solar Ultraviolet (UVB) Radiation and Skin Cancer: Physical and Epidemiological Measurements

Janice Longstreth
The Potential Dermatologic Consequences of Stratospheric Ozone Depletion

1 Robert T. Handren, Jr.
2 Extent of Use

3 Leonard C. Harber
4 Real and Potential Adverse Effects of High-Intensity UVA
5 Light Sources Used for Cosmetic Purposes (Tanning Parlors)

6 Maxim F. Mutzhas
7 Beneficial Effects of Intentional Ultraviolet Exposure in
8 Tanning Parlors

9 Richard W. Gange
10 Commercial Intentional Ultraviolet Exposure: Tanning Parlors

11 Nicholas J. Lowe
12 Sunscreening Agents: Chemical and Physical Design and Use

13 Kenneth H. Kraemer
14 Prevention of Ultraviolet-Induced Skin Damage By Use of
15 Systemic Agents

16 Joseph S. Rossi
17 Exploring Behavioral Approaches to UV Risk Reduction

18 John J. Voorhees
19 Reversal of Photodamage/Photoaging: Is It Possible
20 and How? Retin A

21 Eugene J. Van Scott
22 Use of Alpha-Hydroxy-Keto Acids and 5-Fluorouracil to Eradicate
23 or Ameliorate Skin Lesions Secondary to Photoactinic Damage

24 John H. Epstein
25 Reversal of Photodamage/Photoaging: Is it Possible and How?
26 The Alternative Point of View

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