Health Effects of Sunlight Exposure in the United States

Results From the First National Health and Nutrition Examination Survey, 1971-1974

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• **The dermatologic component of the first National Health and Nutrition Examination Survey (N = 20 637), conducted from 1971 through 1974, documented the deleterious effect of ultraviolet radiation on selected skin and eye conditions. Actinic skin damage was more frequent in white men with high as compared with low sunlight exposure, 36.7% vs 23.3%, respectively. Among white women, the corresponding figures were 34.1% vs 18.6%, respectively. Actinic damage was found more often in individuals with light eye color. Basal cell epitheliomas were found in 11.3% of white men aged 65 to 74 years who had severely actinic-damaged skin as compared with 1.0% of those with undamaged skin. Sunlight exposure was positively associated with localized hypomelanism, localized hypermelanism, seborrheic keratoses, senile lentigines, freckles, acne rosacea, spider nevi, varicose veins, venus star, dry skin, wrinkled skin, pterygia, arcus senilis, and a variety of minor oral lesions of the tongue, palate, and buccal mucosa. These findings suggest that a large number of dermatologic conditions, which may in part result from overexposure to sunlight, may be preventable.**

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The oncogenic effect of solar radiation has been clearly recognized for many years.[1-8] A causal relationship has been proposed for ultraviolet radiation exposure and the common skin conditions of localized hypermelanism and hypomelanism,[2,6,8-10] seborrheic keratoses,[11] lentigines,[7,8,11,12] freckles,[7,13,14] acne rosacea,[6,8,13] surface blood vessel lesions,[4,13,15,16] and skin wrinkles.[17] The surface eye condition pterygia has been attributed to excessive ultraviolet radiation.[18-20] Sunlight exposure has not been linked directly to oral pathology with the possible exception of lupus erythematosus, pemphigus, recurrent herpes simplex, and a variety of lip lesions.[8,13,21,22] However, the known ability of sunlight exposure to trigger autoimmune reactions in the case of lupus erythematosus and the predilection of oral mucosa for autoimmune reactions raises the possibility that sunlight exposure may play a significant role in inducing oral pathology.[23,24]

Several methods have been employed to provide evidence for the positive association of sunlight exposure to pathologic conditions. The most widely cited has been the predominant confinement of particular conditions to sun-exposed areas of the skin, such as the face. The gross and/or microscopic association of a lesion with sun-damaged skin has also been used as evidence. Investigators have correlated the prevalence of specific conditions with individual ultraviolet dosage as determined by sunlight exposure history, latitude, occupation, and season.[1,2,6,8]

The first National Health and Nutrition Examination Survey (NHANES I) bears the distinction of being the only major population-based survey containing a comprehensive dermatologic examination. This examination provided an important database for the study of sunlight exposure within the framework of such a survey. This study presents prevalence rates for a variety of skin and eye conditions according to evidence of sunlight exposure among the 16 191 white and 4104 black persons examined in NHANES I. Documentation of the risks of sunlight exposure may serve to facilitate the development of preventive strategies for ultraviolet radiation-associated conditions. Data from the survey might also be used in public education programs to inform the public of the deleterious effects of sunlight exposure.

PATIENTS, MATERIALS, AND METHODS

Study Population

The first National Health and Nutrition Examination Survey (NHANES I) was conducted by the National Center for Health Statistics during the years 1971 through 1974.[25] The individuals examined constituted a sample of the

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civilian noninstitutionalized population of the United States aged 1 through 74 years, with the exclusion of persons residing on Indian reservations and in Alaska and Hawaii. The examinations were conducted in specially constructed mobile examination centers that were moved around the country to previously selected primary sampling units (PSUs), usually a county or group of counties. The selection of the PSUs was part of a stratified multistage probability design that assured, at the end of the survey and after appropriate statistical weighting, that the persons selected from within the PSUs and actually examined were representative of the US population. The sample design allowed for oversampling of persons in poor districts, of children (1 through 5 years old), of women of childbearing age (20 through 44 years old), and of the elderly (65 through 74 years old).

Of the 28 043 persons 1 through 74 years old selected in the national probability sample, 20 749 (74%) were examined. Of those, 20 637 received the dermatologic examination. Examinations were performed at 65 separate locations throughout the contiguous United States.

Dermatologic Examination

A total of 101 dermatologists conducted the dermatologic examinations during the four survey years. Each dermatologist was given training in the dermatologic examination protocol before commencement of the survey examinations at each particular location. Recruitment and training of the dermatologists, each a third-year resident, were the responsibility of the chairman of the Data Collection Unit for the Committee on Planning of the National Academy of Dermatology. Besides examining the entire integument, including hair and nails, the dermatologist also inspected the eyes and oral cavity.[26]

The dermatologic examination consisted of a complete clinical examination of the skin that considered normal variations in texture, certain manifestations of aging, and all pathologic changes. The diagnoses of individual conditions were made primarily by inspection with some ancillary aid from fungus cultures and a small number of skin biopsy specimens.

After closely questioning the examinee on his occupation and the amount of leisure time spent outdoors, the dermatologist classified the examinee into one of three categories of cumulative lifetime sunlight exposure: (1) low (unimpressive); (2) moderate; (3) high (considerable). It should be noted that examinees designated as having had high sunlight exposure were found to have double the rate of plant contact dermatitis and triple the rate of fingernail trauma compared with examinees with a low sunlight exposure history. These findings provide some confirmatory evidence regarding the validity of the sunlight exposure index used in the study, particularly for the extreme categories. Although data for moderate sun exposure are presented in this report, comparisons were made only between low and high levels of sunlight exposure because we could not presume equal intervals between the three categories.

Table 1 provides distribution and sample size data for sunlight exposure history. The percentages given in this table are weighted to reflect the US population but are not age adjusted. About 1.7 times as many white males as females were rated as having had high sunlight exposure (21.8% vs 12.8%). The corresponding ratio for black males and females was also 1.7 (20.1% vs 11.5%).

After examining the skin covering the entire body, the dermatologist recorded an estimate of the presence and severity of actinic skin damage and its components, actinic keratoses, fine telangiectasia, and senile elastosis. All of these measures were graded as being either absent, minimal, moderate, or severe. The absence or presence (minimal, moderate, or severe) of actinic skin damage is used in this report as another measure of the degree of sunlight exposure.

Table 1.—Number and Weighted Percent of Persons Aged 1 Through 74 Years by Race, Sex, and Sunlight Exposure*

Race and Sex	Sunlight Exposure, No. (%)			
	Low	Moderate	High	Total
W M	2778 (38.4)	2581 (39.8)	1578 (21.8)	**6937 (100.0)**
W F	4669 (48.8)	3475 (38.4)	1110 (12.8)	**9254 (100.0)**
B M	858 (50.3)	492 (29.7)	341 (20.1)	**1691 (100.0)**
B F	1427 (58.4)	700 (30.1)	286 (11.5)	**2413 (100.0)**

*Source: National Center for Health Statistics, First National Health and Nutrition Examination Survey, 1971-1974.

Statistical Methods

All statistical analyses in this report were made using weighted data to reflect the US population. Because the design of NHANES I was a multistage probability sample, three basic operations—inflation by the reciprocal of the probability of selection, nonresponse adjustment, and poststratification by age-sex-race—were involved in the estimation procedure for prevalence estimates. The nonresponse adjustment helps alleviate nonresponse bias. From what is known about the nonrespondents and the nature of nonresponse from NHANES I and other National Center for Health Statistics examination studies, the likelihood of substantial nonresponse bias was small.[27-29] No adjustment was made for the 112 examinees who did not receive the dermatologic examination.

Age adjustment was performed using the direct method, with the reference population being the estimated US population at the midpoint (fall 1972) of NHANES I. The SEs used in the analysis were calculated by a balanced half-sample replication technique using the balanced repeated replication (BRR) computer program,[30] which computed overall variance through observation of variances among random subsamples of the total sample. The variances produced by this method are comparable in magnitude to those generated by the Taylor series approximation technique.[31] Due to the complex sample design used, these SEs are substantially greater than those that would have been generated assuming simple random sampling. Logistic regression was performed using a statistical analysis system (SAS) procedure (Proc Logist), modified to incorporate sampling weights in the estimation of the logistic regression parameters.[32,33]

All of the analyses presented herein are based on Z tests comparing prevalence rates in two population subgroups, high vs low or damage vs no damage. This test consists of dividing the difference between the two prevalences by the SE of the difference. A two-sided test of significance was made for each analysis. The SEs are included in the Tables to give an indication of the statistical reliability of the individual estimates. As noted previously, statistical tests were performed only between low and high sunlight exposure groups because equal intervals between categories could not be assumed.

Limitations of Data

Both measures of sunlight exposure used in NHANES I were subject to errors of classification. The historical information collected did not allow for the differences in

ultraviolet radiation exposure that can be attributed to the latitude and altitude of specific examination sites. Using the first 35 locations of NHANES I, constituting a separate sample of the US population, Hiller et al[34] were able to demonstrate a positive association of cataracts with ultraviolet-B counts as determined from examination site location and altitude alone. Some other confounding factors are inherent in a sunlight exposure history. For example, individuals who are particularly susceptible to actinic skin damage, ie, those with a tendency to burn rather than tan, are likely to avoid high-exposure activities such as sunbathing.[35]

Another source of data limitation arises from the fact that neither of the measures of sunlight exposure was obtained by "blind" observers, thereby raising the possibility of measurement bias. For instance, ideally a sunlight exposure history might have been obtained without direct observation of the examinee's skin. The lack of blind observers might increase the correlation between the sunlight exposure history variable and actinic skin damage to the extent that examinees obtaining the sunlight history might have been influenced by evidence of actinic skin damage that was observed prior to the actual examination. The dermatologists who performed the examinations were not told of any hypothesis that might be tested by the study. As in any determination requiring clinical judgment, a certain amount of observer differences should be expected in each dermatologist's designation of actinic skin damage. The impact of any particular dermatologist's judgments on the overall results was minimized by the large number of examiners used in the survey. Observer differences for the conditions used in the analysis were probably minimized by the recording format that forced the dermatologist to make a specific entry for each of the items as distinguished from a "significant condition" approach.

Because this study used a cross-sectional design, definite statements regarding cause and effect relationships cannot be made. However, it is unlikely that the abnormalities described in this report preceded sunlight exposure.

RESULTS

As seen in Table 2, about 1.6 times as many white males (36.7% vs 23.3%) with high sunlight exposure had actinic skin damage as compared with those with low sunlight exposure. The corresponding rate ratios in white males for the components of actinic

Table 2.—Age-Adjusted Prevalence Rates and SEs of Actinic Skin Damage and Its Components Among Whites Aged 1 Through 74 Years by Sunlight Exposure*

	Sunlight Exposure					
	Males			Females		
Actinic Skin Damage and Components	Low (N = 2778)	Moderate (N = 2581)	High (N = 1578)	Low (N = 4669)	Moderate (N = 3475)	High (N = 1110)
Actinic skin damage	23.3 (1.43)	30.6 (1.11)	36.7† (1.35)	18.6 (0.99)	23.8 (1.11)	34.1† (1.77)
Senile elastosis	12.4 (1.13)	18.4 (1.09)	27.0† (1.27)	10.0 (0.76)	13.9 (0.95)	23.3† (1.81)
Actinic keratosis	5.6 (0.76)	9.6 (0.79)	14.4† (1.05)	2.7 (0.42)	4.8 (0.55)	11.4† (1.68)
Fine telangiectasia	17.3 (1.14)	23.1 (1.04)	30.1† (1.40)	11.6 (0.89)	17.3 (0.97)	26.2† (2.00)

*Values in parentheses are SEs. Significance test: low exposure vs high exposure. Source: National Center for Health Statistics, First National Health and Nutrition Examination Survey, 1971-1974.

†Significance level: $P < .001$.

Table 3.—Age-Adjusted Prevalence Rates and SEs of Actinic Skin Damage, Hypermelanism (Localized), Hypomelanism (Localized), Senile Lentigines, and Freckles, Among Blacks Aged 1 Through 74 Years by Sunlight Exposure*

	Sunlight Exposure					
	Males			Females		
Actinic Skin Damage and Other Conditions	Low (N = 858)	Moderate (N = 492)	High (N = 341)	Low (N = 1427)	Moderate (N = 700)	High (N = 286)
Actinic skin damage	3.4 (1.07)	3.7 (1.46)	4.1 (1.60)	3.5 (1.00)	3.0 (0.73)	0.6† (0.33)
Localized hypermelanism	23.6 (2.52)	26.6 (3.42)	36.2‡ (5.96)	22.9 (2.15)	32.1 (2.78)	37.5† (4.89)
Localized hypomelanism	8.7 (1.67)	11.2 (2.22)	20.7‡ (4.75)	10.6 (1.68)	10.8 (1.49)	17.0 (4.31)
Senile lentigines	0.7 (0.31)	0.3 (0.15)	0.3 (0.19)	2.5 (0.78)	1.2 (0.31)	1.8 (0.69)
Freckles	1.6 (0.79)	1.7 (1.14)	2.2 (1.50)	2.8 (1.07)	4.1 (1.53)	1.3 (0.81)

*Values in parentheses are SEs. Significance test: low exposure vs high exposure. Source: National Center for Health Statistics, First National Health and Nutrition Examination Survey, 1971-1974.

†Significance level: $P < .01$.

‡Significance level: $P < .05$.

skin damage, ie, senile elastosis, actinic keratoses, and fine telangiectasia, were 2.2, 2.6, and 1.7, respectively. Similar results were found for white females, with rate ratios of 1.8 for actinic skin damage, 2.3 for senile elastosis, 4.2 for actinic keratoses, and 2.3 for fine telangiectasia. In subsequent analyses, the composite actinic skin damage measure will be employed rather than its components.

Table 3 shows rates of actinic skin damage and other dermatologic conditions for blacks. Overall, the prevalence of actinic skin damage was much lower for blacks than for whites. Black women with considerable sunlight exposure showed significantly less actinic skin damage than black women with low sunlight exposure (0.6% vs 3.5%) ($P < .01$). This relationship was opposite that previously noted for white women.

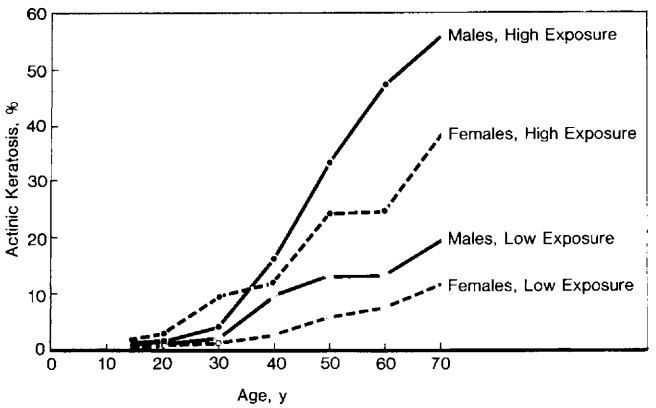

Fig 1.—Prevalence rates (per 100) of actinic keratosis among US whites (source: First National Health and Nutrition Examination Survey). Rates are plotted by age for each of four sex, sunlight level exposure groups.

Due to its role as a precancerous lesion,[13] actinic keratosis is clinically the most important manifestation of actinic skin damage. As seen in Fig 1, increasingly higher prevalence rates for actinic keratoses were found in successively older white, adult-aged groups, regardless of sex and sunlight exposure status. In each of these groups, actinic keratoses were found more often in high as compared with low sunlight exposure. The absolute difference in the prevalence of actinic keratoses for high vs low sunlight exposure was widest in the 65- to 74-year-old age group, 55.4% vs 18.5% for white men, and 37.3% vs 11.9% for white women.

Figure 2 shows the association of actinic skin damage with eye color. Age-adjusted skin damage prevalence rates for white persons with dark brown eyes were compared with those with lighter eye colors. Significant elevations in actinic skin damage rates were found for all of the lighter eye-colored white males with the highest rates found among males with dark blue eyes (35.0%). Among males, rate ratios for actinic skin damage comparing dark

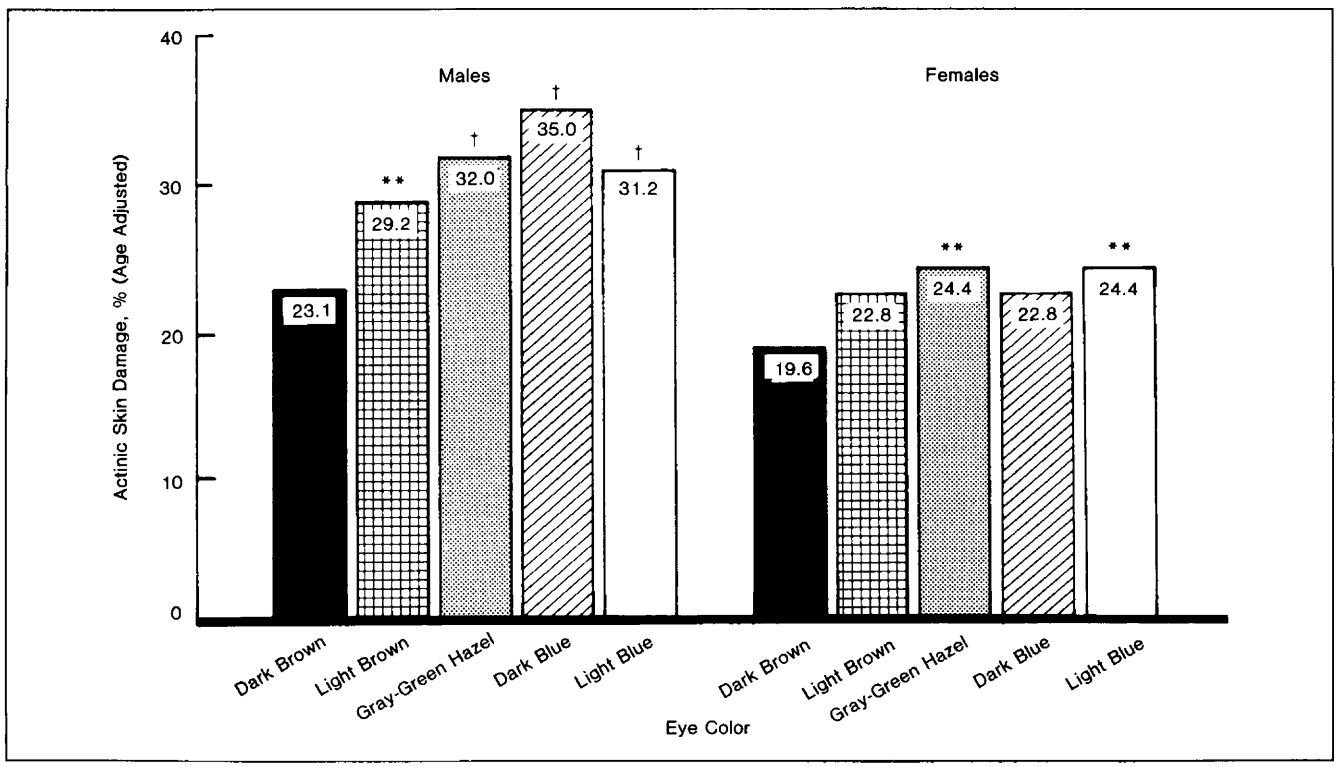

Fig 2.—Age-adjusted prevalence rates of actinic skin damage among US whites, aged 1 through 74 years (source: First National Health and Nutrition Examination Survey). Rates are plotted according to eye color and sex. Values with bars represent prevalence rates (per 100) of actinic skin damage. Significance testing within sex compares rates of actinic skin damage for dark brown eyes with rates of actinic skin damage for eyes of specific other colors. Significance levels: two asterisks indicate $P < .01$ and double daggers indicate $P < .001$.

Table 4.—Age-Adjusted Prevalence Rates and SEs of Pigment Aberrations, Senile Lentigines, and Freckles Among Whites Aged 1 Through 74 Years by Sunlight Exposure*

Selected Pigment Aberrations	Sunlight Exposure					
	Males			Females		
	Low (N = 2778)	Moderate (N = 2581)	High (N = 1578)	Low (N = 4669)	Moderate (N = 3475)	High (N = 1110)
Localized hypermelanism	4.0 (0.61)	6.0 (0.62)	7.9† (0.94)	5.3 (0.51)	6.6 (0.54)	9.5‡ (1.29)
Localized hypomelanism	5.0 (0.66)	9.0 (0.89)	15.3† (2.33)	5.2 (0.54)	9.0 (0.80)	15.2† (2.33)
Senile lentigines	11.4 (1.14)	12.3 (0.74)	13.9 (1.06)	12.5 (0.64)	15.3 (0.81)	17.0‡ (1.50)
Freckles	24.5 (1.31)	30.0 (1.22)	31.8† (2.00)	22.3 (0.94)	27.0 (1.15)	34.4† (1.97)

*Values in parentheses are SEs. Significance test: low exposure vs high exposure. Source: National Center for Health Statistics, First National Health and Nutrition Examination Survey, 1971-1974.

†Significance level: $P < .001$.

‡Significance level: $P < .01$.

Table 5.—Age-Adjusted Prevalence Rates and SEs of Vascular Conditions, Minor Oral Lesions, and Miscellaneous Conditions, Among Whites Aged 25 Through 74 Years by Actinic Skin Damage*

Miscellaneous Conditions	Actinic Skin Damage			
	Males		Females	
	No Damage (N = 1442)	Damage (N = 2270)	No Damage (N = 3511)	Damage (N = 2070)
Basal cell epithelioma	0.1 (0.09)	0.8† (0.20)	0.2 (0.10)	1.2† (0.29)
Seborrheic keratosis	6.5 (1.00)	10.5† (0.96)	7.2 (0.62)	9.7‡ (0.88)
Pterygium	0.7 (0.22)	1.8† (0.36)	0.4 (0.14)	0.7 (0.17)
Arcus senilis	2.8 (0.55)	6.3§ (0.68)	1.9 (0.33)	4.1† (0.58)
Dry skin	12.8 (1.72)	21.4§ (1.59)	20.6 (1.33)	35.4§ (1.86)
Wrinkled skin	6.1 (1.18)	7.1§ (0.79)	10.0 (1.19)	14.1§ (1.06)
Varicose veins	8.6 (1.03)	12.9† (1.18)	20.4 (1.36)	26.2† (1.54)
Nevus araneus	1.9 (0.62)	3.8‡ (0.68)	2.0 (0.47)	8.9§ (1.93)
Venus star	6.0 (1.07)	15.5§ (1.68)	28.9 (1.94)	40.9§ (2.51)
Acne rosacea	2.1 (0.76)	2.8 (0.50)	0.9 (0.24)	2.1‡ (0.45)
Tongue fissures	0.1 (0.33)	4.9§ (1.09)	1.2 (0.29)	3.4‡ (0.91)
Tongue serrations	0.6 (0.22)	2.7‡ (0.87)	0.7 (0.18)	3.5† (1.04)
Tongue papillae	0.5 (0.18)	3.0† (0.76)	1.3 (0.28)	1.9 (0.37)
Palate conditions	1.3 (0.48)	6.6† (1.63)	0.6 (0.18)	3.0† (0.76)
Fissured buccal mucosa	0.6 (0.34)	2.9‡ (1.09)	0.3 (0.21)	2.4‡ (0.82)
Angular lip lesions	0.2 (0.13)	1.1† (0.30)	0.2 (0.07)	0.6‡ (0.20)

*Values in parentheses are SEs. Significance test: no damage vs damage. Source: National Center for Health Statistics, First National Health and Nutrition Examination Survey, 1971-1974.

†Significance level: $P < .01$.

‡Significance level: $P < .05$.

§Significance level: $P < .001$.

brown eyes with other colors ranged from 1.3 for light brown eyes to 1.5 for dark blue eyes. As in males, all of the white females with lighter-colored eyes had higher rates of actinic damage than those with dark brown eyes. However, the only statistically significant elevations were for "gray, green, hazel" and "light blue eyes" as compared with dark brown eyes.

As seen in Tables 3 and 4, the prevalence of the pigmentary aberrations of localized hypermelanism and hypomelanism varied directly with sunlight exposure for both whites and blacks. For localized hypermelanism, there was approximately a twofold increase in prevalence for both blacks and whites, from the low- to high-exposure groups. In whites, the most notable differences were observed for localized hypomelanism prevalence, which increased threefold in both sexes from the low- to high-exposure groups. In whites, the differences in prevalence rates were significant for all listed conditions, except senile lentigines in white males. Unlike whites, senile lentigines and freckles were relatively infrequent in black persons, with no consistent pattern observed in relation to the degree of sunlight exposure.

As previously noted, sunlight exposure as obtained by history in NHANES I may be subject to the confounding influences of latitude, differences in sunscreen utilization, and a probable tendency for fair-skinned individuals to avoid high sunlight exposure. To avoid these potential biases from historical data, the objective evidence of actinic skin damage, conceptually the more accurate of the two indicators of sunlight exposure, is used for the remainder of the analyses. Results are given for white adults only, as actinic skin damage is not a useful measure of sunlight exposure in blacks and children due to its low prevalence in these groups. In Table 5, age-adjusted actinic skin damage is presented as a dichotomy. The designation "no damage" indicates the absence of actinic skin damage, and the designation "damage" indicates the presence of either minimal, moderate, or severe actinic skin damage.

As seen in Table 5, actinic skin damage was

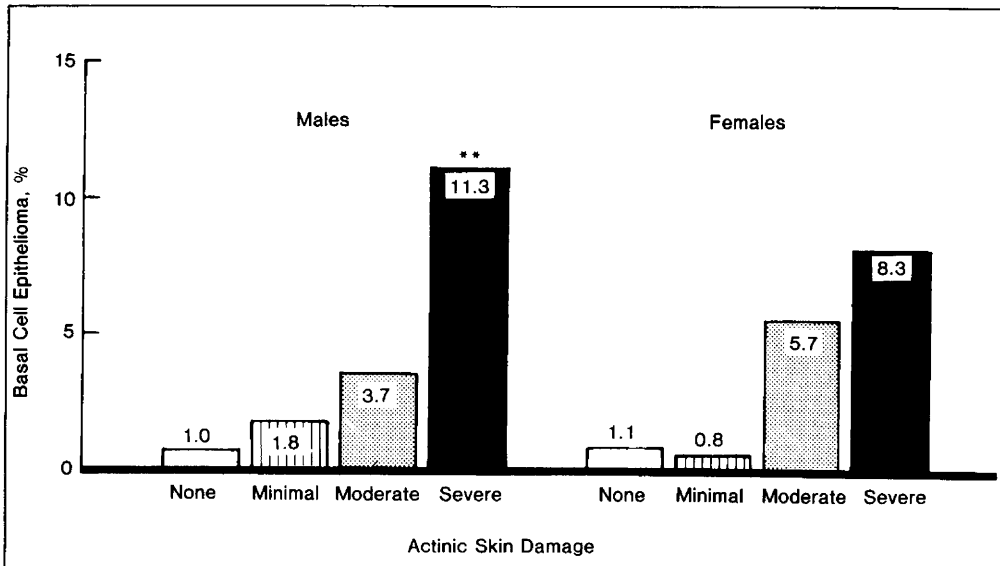

Fig 3.—Prevalence rates of basal cell epithelioma among US whites, aged 65 through 74 years (source: First National Health and Nutrition Examination Survey). Rates are plotted according to severity of actinic skin damage and sex. Values within or above bars represent rates (per 100) of basal cell epitheliomas. Significance testing within sex compares rates of basal cell epitheliomas for persons with severe actinic skin damage with rates of basal cell epithelioma for persons with no actinic skin damage. Significance level: two asterisks, $P < .01$.

associated with an increased prevalence of a variety of conditions that involve blood vessels, including varicose veins, nevus araneus (spider nevus), venus star, and acne rosacea. Statistically significant differences were found between the no damage and the damage groups with the exception of acne rosacea in men.

Basal cell epithelioma, the most common form of skin cancer, was positively associated with actinic skin damage, with rates increasing from 0.1% in men with no skin damage to 0.8% in men with skin damage ($P < .01$). The corresponding increase in women was from 0.2% to 1.2% ($P < .01$). In the 65- to 74-year-old group, where the bulk of basal cell carcinomas is found, the basal cell epithelioma rates for minimal and no actinic skin damage are essentially the same (Fig 3). Major increases in rates were present only for greater degrees of actinic skin damage. The increased prevalence of basal cell epitheliomas with severe vs no actinic skin damage was statistically significant only in men. In this group, there is an 11-fold increase from 1.0% for men with no actinic skin damage to 11.3% for men with severe actinic skin damage ($P < .01$). In women, there was a nonsignificant eightfold increase from 1.1% to 8.3%.

Actinic skin damage was also significantly associated with seborrheic keratoses (Table 5), a common proliferative skin lesion. Dry skin was more common in skin-damaged women, 35.4%, as compared with 20.6% for non-skin-damaged women ($P < .001$). The rate ratios for dry skin were 1.7 in both men and women. Wrinkled skin was significantly more prevalent in women with actinic skin damage as compared with those with no damage (14.1% vs 10.0%) ($P < .001$). Prevalence rates for pterygia in men were significantly increased from 0.7% for the non-skin-damaged group to 1.8% for the skin-damaged group ($P < .01$). In addition, prevalence rates for arcus senilis were more than doubled for both men and women with actinic skin damage as compared with those without damage.

The data in Table 5 also demonstrate a positive association of actinic skin damage with a variety of minor oral lesions, including fissures of the tongue, fissured buccal mucosa, tongue serrations, tongue papillae, palate conditions, and angular lip lesions. The rate ratios for actinic skin damage vs no skin damage in men was approximately 5:1 for all the conditions listed. The rate ratios in women were similar to those found in men, with the exception of tongue papillae conditions.

Since the association of actinic skin damage with oral lesions has not been observed previously, further analyses were performed to compare the prevalence of at least one oral lesion in white individuals with sunlight exposure history. For both men and women, and all nine age groups, the prevalence of oral lesions was greater in the high vs low sunlight exposure groups. Thus, the association with oral lesions was found using both measures of sunlight exposure.

To control for the possible confounding of this association from tobacco use, age, and eye color (as a surrogate for skin type), logistic regression analysis was performed. The association of actinic skin damage with the presence or absence of any oral lesions remained significant ($P < .001$) after controlling for the above variables; the estimated rate ratio was 5.47 for men and 4.98 for women. When tobacco use and eye color were omitted for the analysis, the estimated rate ratios remained the same. This specific analysis was performed on a subsample (n = 3126) of the examinees who had tobacco history data.

Since the NHANES I survey was a cross-sectional study, we cannot determine whether the skin damage occurred before or after the oral lesions. The confirmatory data presented, however, with history of sunlight exposure support the interpretation that the damage or high sunlight exposure preceded the occurrence of oral lesions. A prospective study would be necessary to adequately address the antecedent-

consequence relationship of these variables. Overall, the results of NHANES I would appear to demonstrate a positive association of sunlight exposure and actinic skin damage with a wide variety of common dermatologic conditions.

COMMENT

Notwithstanding the possible classification errors already discussed, the results of NHANES I are in general agreement with commonly accepted associations between ultraviolet radiation and certain cited conditions. The degree of association between ultraviolet radiation and the studied conditions was probably somewhat understated due to sunlight exposure classification errors and the fact that only qualitative rather than quantitative measures of sunlight exposure were employed. Given accurate quantitative ultraviolet exposure data, the relationships may have been more marked. The unique value of the NHANES I findings springs from the fact that the data represent the only large body of dermatologic examination data that can be generalized to the US population.

The risk factors found in the NHANES I study for actinic skin damage and actinic keratosis (ie, ultraviolet dose, age, male sex, light eye color, and white race) closely resemble the risk factors that have been reported in the literature for nonmelanoma skin cancer. The causative role of ultraviolet light in the development of both squamous and basal cell skin cancers has been demonstrated in previous epidemiologic investigations.[3,36,37]

In the period 1977-1978, a special survey of nonmelanoma skin cancer was conducted in eight geographic locations in the United States by investigators from the National Cancer Institute.[36] From over 30 000 patients, the data presented by Scotto et al[36] clearly demonstrated a latitudinal and ultraviolet-B gradient with skin cancer incidence rates, the latter being highest in geographic areas of relatively high ultraviolet-B exposure. In addition, about 80% of all nonmelanoma skin cancers occurred in the head and neck. Nonmelanoma skin cancer was extremely rare in blacks and relatively infrequent in Hispanics. The risk for males was greater than for females with a twofold excess risk apparent in many localities.[36] Vitaliano and Urbach,[3] in a Philadelphia case-control study, and Silverstone and Searle,[37] in a Queensland, Australia study, also demonstrated an increased risk of skin cancer among individuals with light eye color, fair complexion, a tendency to burn rather than tan, and born of Celtic ancestry.

In regard to basal cell carcinoma, the most prevalent form of skin cancer, the data from NHANES I would seem to indicate that prevalence rates for this condition would be greatly reduced by the prevention of moderate to severe actinic damage. This could be achieved by identifying those individuals with known risk factors for skin damage and also those persons who show signs of skin damage at some early age. Once identified, high-risk persons could be warned about the importance of protection for actinic skin damage through the avoidance of excess sunlight exposure.

The risks resulting from ultraviolet radiation exposure may be increased in the future by a partial depletion of the protective ozone layer in the stratosphere resulting from the release of fluorocarbons. The possible eventual ozone depletion has been estimated to be as high as 16%. An increase of about 4% in nonmelanoma skin cancer may be expected for each 1% relative decrease in stratosphere ozone.[1]

Ultraviolet radiation has been shown to produce a variety of deleterious changes in human and animal skin. Certain known effects of ultraviolet radiation may be involved in mechanisms responsible for increased prevalence of some of the specific skin conditions reported in the study. For example, many of those conditions, such as seborrheic keratoses, are proliferative in nature. Ultraviolet radiation increases cellular proliferation.[38] Melanocytes are responsible for the production of melanin pigment. These melanocytes may be destroyed locally by ultraviolet radiation–producing hypomelanism. Alternatively, ultraviolet radiation may also result in localized hypermelanism through the stimulation of local melanocytes and from an increase in melanocyte density.[9] In addition, wrinkles are a result of an associated structural deterioration of the elastic network, which is enhanced by solar radiation.[17]

As expected, the NHANES I data demonstrated the profound influence of black ancestry in drastically limiting actinic skin damage. Complexion skin differences existing in the US black population mask actinic skin damage but may also play a strong role in determining the damage within that group. Perhaps the most intriguing finding of the study was the demonstration for the first time of a strong association between actinic skin damage and a constellation of minor oral lesions of the tongue, palate, and oral mucosa.

The hypothesis suggested by the NHANES I findings can be stated as follows: "Sunlight exposure to skin and/or lip oral mucosa (vermilion border) triggers an immune reaction in the oral mucosa, which results in oral lesions." Testing of this hypothesis in future studies would require demonstration of immunologic reactions in oral tissue among individuals with high sunlight exposure. The most suggestive evidence for a possible immunologic relationship is the pronounced tendency of oral tissue to develop autoimmune reactions. A variety of autoimmune diseases, including lupus erythematosus, scleroderma, dermatomyositis, pernicious anemia, Crohn's disease, pemphigus, Sjögren's syndrome, aphthous stomatitis, Behçet's syndrome, and chronic graft-vs-host disease,[23,24,39] have oral manifestations. Alternatively, the NHANES I findings could be the result of some unknown confounding factors.

The most common of oral conditions, periodontitis, has been associated with lymphotoxicity for gingival epithelial cells.[40] An indication of underlying oral autoimmune activity is the finding of autoantibodies to oral mucosa in 10% of control saline homogenate specimens.[24]

Ultraviolet radiation has been shown to produce

immunologic reactions. Specific antibodies to photoproducts of irradiated skin DNA have been found in the serum samples of patients with systemic lupus erythematosus.[41] Cooper et al[42] have recently demonstrated that ultraviolet radiation, after a 24-hour delay period, acted to markedly enhance immunologic function in human skin.

Of interest is our original finding of an increased prevalence of arcus senilis in individuals with actinic skin damage. Although commonly considered as part of the normal aging process, some studies have linked the condition to coronary artery disease.[43]

In view of the deleterious effects of ultraviolet radiation on the skin and eyes, a concerted effort would seem in order to persuade the public to reduce exposure to sunlight, especially around noon when the highest dosage of ultraviolet radiation reaches the skin. The increased use of appropriate sunscreens would also be highly desirable. Outdoor workers should protect their eyes from the sun by wearing wide-brimmed hats and using wraparound sunglasses. For increased effectiveness, such an educational campaign should be able to cite a variety of conditions, other than just skin cancer, that are associated with ultraviolet exposure. As the desire to look young and look good seems to be a feature of modern society, the linkage of excess sunlight exposure to an image of unattractive-looking skin would seem desirable.

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