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Prevention of Skin Cancer: Healthy Sun Exposure and No Sunscreen for Intense Intermittent Exposure; Photoaging Theories Questioned and New Strategies

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Abstract

Currently the FDA adopts a zero-tolerance policy toward sun exposure in order to prevent skin cancer and premature skin aging (photoaging). This is apparently based on classical concept that damage from sunlight, a carcinogen, is cumulative and un-reparable. Such concept is apparently flawed both theoretically and in reality. The nature's design to achieve genomic stability of body health and appearance for smooth passage of generations in our normal daily lives would require virtually complete repair of damages of DNA and other tissue components from daily Exposure to Non-Burning Sunlight (ENS). In other words, ENS is generally not expected to cause skin cancer and photoaging. Such notion is evidenced by, for example, low worldwide skin-cancer incidences, severe sunburn as overwhelming skin-cancer etiology, and intrinsic aging as overwhelming skin aging. Since ENS can provide numerous health benefits, such exposure can be regarded as healthy sun exposure and used to help prevent skin cancer. Due to unintended sunburn effect, use of sunscreens for intense intermittent exposure is strongly discouraged. As photoaging and skin cancer may be closely related, some questions related to conventional theories and practices in photoaging are also raised. They include the following: Schuster's pioneering study in 1975; invalidation of accelerated aging theory; questionable theory on etiology of wrinkles and age spots; Fisher's studies on metalloproteinases; bolus doing vs constant-rate dosing in irradiation; moisturizers as anti-photoaging/anti-cancer agents; inclusion of blood and water in skin-aging exosome; wind

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effect; differences in usage pattern between countries in sunscreen evaluation; replacement of UVA in tanning beds.

Keywords

Skin Cancer; Melanoma; Sun Exposure; Sunscreen; Photoaging

Background

Skin cancer is the most common cancer. In the United States about 1 in 5 people is estimated to have skin cancer before age of 70 [1]. It affects millions of people and causes billions of dollars in medical care annually [1]. Most importantly, skin cancer incidences have been increasing exponentially in the last several decades in spite of efforts by health professions and authorities [1,2]. Skin cancer is commonly regarded as a preventable disease mainly due to exposure to sunlight, a known carcinogen that calls for zero tolerance by conventional guideline [3]. In this regard, damage from exposure to sunlight, especially Ultraviolet (UV) A and B rays, has been universally regarded as cumulative in the last century [1,3-6].

In the last decade the thinking and advice of the US Food and Drug Administration (FDA) on skin cancer prevention is to advise infants less than six months old not to be exposed to any sunlight and to advise the general public to minimize or avoid sunlight exposure by measures such as seeking shelters, wearing long-sleeve shirts, long pants, broad-brimmed hats, broad-spectrum sunscreens, and sun eyeglasses [3]. Since premature skin aging (photoaging) has also commonly been attributed to exposure to sunlight, the above recommended method is also used to help prevent premature skin aging [3]. It is to be noted that there are no disagreements in the literature that excessive sun exposure can cause skin cancer and premature skin aging such as cumulative deposits of elastotic materials in the dermis and therefore should be minimized or avoided.

After reviewing numerous published studies, Chiou recently concluded that instead of preventing sunburn as shown on the product label, use of sunscreens for intense intermittent sun exposure can ironically cause sunburn primarily due to virtually unavoidable, SPF-independent uneven or missing application of sunscreens and prolonged stay for sun exposure [7-9]. Chiou further suggested that increased use of sunscreens in recent decades may mainly account for the exponential increase of melanoma incidences [8,9]. Since basal cell carcinoma and squamous cell carcinoma could be caused by Severe Sunburn (SS), sunscreen use may also be largely responsible for their reported increases in incidences [10]. Therefore, use of sunscreen should be strongly discouraged for intense intermittent sun exposure [9,10]. In 2013 Petersen, et al., published an alarming study titled “A sun holiday is a sunburn holiday” [11].

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Apparently, such an important work has received relatively little attention by health professions and authorities as well as by general public to date. It seems that wearing protective clothing and avoiding peak sun hours may be the best ways to prevent sunburn and skin cancer when engaging in intense intermittent sun exposure [12].

In implementing the current sun avoidance policy, one may need to be very certain that radiation effects from mild or moderate sun exposure without incurring sunburn as occurring daily in the general public are truly cumulative and become a major risk factor for both skin cancer development and photoaging [3]. It appears that to date no convincing clinical evidence has ever been published to support such a notion. In this regard, Chiou recently reported that most skin cancers may be solely initiated by SS and the DNA damages due to exposure to mild or moderate sunlight may not be cumulative and thus may not result in skin cancer development [10]. The notion of a non-cumulative, non-harmful UV-caused DNA lesions was used to rationalize such a hypothesis [10]. Chiou also presented some evidence to support the applicability of the above rationale to photoaging [10].

The main scope of this perspective article is in the following areas:

1. To articulate more clearly the justification for the proposed new approach for prevention of skin cancer in general public, namely, healthy sun exposure without SS or erythema, a mild form of sunburn
2. To comment on some widely accepted photoaging theories related to non-burning sun exposure that is commonly occurring in our daily lives
3. To briefly highlight the health benefits of non-burning sunlight exposure
4. To propose a new safe approach of using special moisturizers for reversing skin aging, and preventing photoaging and skin cancer
5. To provide some practical tips for implementation of healthy sun exposures. It is hoped that this provocative work may stimulate further discussion and study on these important issues

Rationale for non-cumulative, non-harmful UV-caused DNA lesions: Sustaining genomic stability for good health and appearance

It has been reported that each normal sun-exposed skin cell in humans may be subjected to 70,000 DNA lesions or damages per day from both endogenous (metabolic) and exogenous (mainly UV) assaults [13-15]. In order to maintain genomic stability to ensure smooth passage of generations an extremely sophisticated, signal-orchestrated DNA repair mechanisms are developed by our body to repair and remove the lesions [13-15]. A mutation rate of $1.6 \times$

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10–7/bp/division in skin cell has been estimated that is extremely low and can be practically regarded as zero [16]. Furthermore, the probability of an un-repaired UV-damaged mutants to eventually develop into a skin cancer is also be quite low as numerous steps need to be overcome [13-15]. Therefore, the above suggests that contrary to conventional concepts, generally speaking, the DNA lesions from chronic non- burning UV exposure are non-cumulative, non-consequential and non-cancer-forming [10]. The validity of the above discussion seems to have been robustly confirmed by the fact that most people in the world do not develop skin cancer in their lifetimes even though most of them may probably have ever experienced SS [10]. It is also probably unequivocally supported by data showing SS as the dominant or sole etiology for the 3 most common skin cancers based on re-analysis of published data from 2 very large prospective cohort studies in the US [10,17]. Of note, it was stated in a recent report from an expert panel that skin cancer is the most important chronic effect of solar radiation [18]. It appears that to date no human or animal studies have ever shown convincingly the formation of skin cancer being attributed to chronic exposure to non-burning low UV doses. The notion of the above non-cumulative UV-caused DNA lesions are supported, for example, by no differences in lesion products like cyclobutene pyrimidine dimers in the body following single or multiple UV doses in humans or fish [19,20]. Furthermore, their products were quickly eliminated from the body [19,20].

SS Overwhelms the DNA Repair Mechanisms

It is now commonly accepted that when the magnitude of DNA lesions overwhelms the skin's DNA repair capacity, an un-repaired or un-removed mutant may have an opportunity to eventually develop into a skin cancer [21-23]. Interestingly, strong sunlight can produce up to 100,000 DNA lesions per skin cell per hour [24-26]. It was suggested that the SS, caused by strong sunlight primarily attributed to UVB rays, can overwhelm a skin cell's DNA repair capacity and can potentially trigger the development of skin cancer [10]. Therefore, trying to avoid to have a SS from the sun, tanning beds and sun lamps may be the most important and effective way to prevent skin cancer. It was suggested that only SS, not erythema, can cause skin cancer [10]. Apparently, the erythema-induced DNA lesions can be all eventually repaired or removed. Interestingly, an erythema can virtually completely heal by itself in days with or without any minor intervention further supporting the concept of non-cumulative UV damage [10]. Until now, erythema has been commonly used as a biomarker for skin cancer [10,27]. The scientific justification of such a practice may thus need to be reconsidered. The above discussion may provide a theoretical reason as to why chronic moderate sun exposure may not initiate skin cancer development for ordinary people as commonly assumed [18]. This is also in line with early reports that overall, UVA induces little DNA damages in skin cancer development [28,29]. Since it may be practically difficult for the general public to control the

degree of sunburn, it is advisable for the public to avoid incurring erythema in daily life in order to prevent skin cancer.

Conventional Photoaging Theories Questioned

It is commonly accepted that photoaging could account for about 80% to 90% of skin aging [30-32]. Chiou has discussed potential shortcomings of such a concept or theory for ordinary people without exposure to excessive sunlight and has suggested the intrinsic factor, cardiac output/index, as the dominant or overwhelming factor in affecting skin aging [9,33]. This is consistent with nature's ability to sustain genome integrity for maintaining skin appearance. In an extensive review, it was stated that in photoaging, various skin damages, abnormalities and deteriorating conditions from sun exposure can be totally repaired through apoptosis and the regeneration of the cells [21]. Therefore, these UV-caused lesions can be only temporary and not cumulative with age; this is largely consistent with the present non-cumulative DNA damage notion. However, some conditions like deposits of elastotic materials in the dermis probably due to intense chronic sun exposure may accumulate over time. Several other questions related to conventional photoaging theories are discussed below.

Long-ignored pioneering work of Dr. Sam Shuster on skin collagen content in ordinary people: In 1975, Dr. Shuster, et al., published a widely cited (678 times per Google search on October 22, 2022) paper on the influence of age and sex on skin thickness, skin collagen and density in 154 Caucasians aged from 15 to 93 years [34]. They found that skin collagen content per unit skin surface area for each subject in skin samples taken from the sun-protected or sun-exposed area was precisely the same. This paramount finding was only mentioned in one short sentence in the text without elaborating on its potential significance in skin aging theory. Since it was not mentioned in the abstract, the sentence might have been easily overlooked by most readers in the last half a century. Another reason why this seminal work did not receive proper credit for showing the dominant role of intrinsic factor in skin aging may possibly be due to publication of enormous volumes of studies in molecular biology detailing damaging effects of sun or UV irradiation on skin tissue in the last several decades.

Dr. Shuster recently cited his 1975 paper reaffirming the dominance of intrinsic factor in skin aging [35]. It appears that there is an urgent need for other researchers to confirm the findings of Dr. Shuster especially using subjects who have not got sunburned in their entire lives. It is of interest to note a study indicating that there were practically no differences in collagen content, collagen solubility property and collagen synthesis of skin samples taken from sun-exposed or sun-protected area from each of 15 subjects with a mean age of about 68; in these old-age subjects, major differences in collagen content were expected based on current photoaging theories, thus further supporting the work of Dr. Schuster [34,36]. In a study on 8

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healthy subjects aged between 47 and 79, both sun-protected and sun-exposed skin appeared equally normal without any clinical and molecular abnormalities although there were disappearance of elastic fibers and more elastotic materials in the dermis of sun-exposed skin suggesting only minor photoaging effects in these subjects [37]. Interestingly, a study commenting on overestimation of photoaging by histochemistry has been published [38]. I have observed (unpublished) several senior Asian women living in Chicago and Taiwan who had rarely used sunscreens but had a very smooth healthy-looking skin.

Kinetic analyses effectively invalidating accelerated photoaging theory and supporting the dominant or overwhelming intrinsic aging theory in normal skin aging: Kinetic analysis has been commonly employed as a powerful tool to study mechanisms of processes or actions in biological systems. Due to reported increases in metalloproteinases that will degrade collagen and elastin in skin, a decrease of synthetic enzyme for collagen, as well as the action of free oxygen radicals causing oxidative stress, chronic sun exposure has been universally accepted in the last century to be able to cause acceleration of skin aging [10,33,39-41]. A review of the literature suggests that virtually no convincing clinical data have been ever published to support the accelerated aging theory. It appears that Chiou was the first one to employ the first-order kinetic method to unambiguously demonstrate that accelerated skin aging did not occur in normal subjects [33]. This is because aging kinetics of skin collagen per unit surface area of skin in 70 males with 8 decades of life span clearly followed a first order kinetics without showing an acceleration in rate constant with an increase in age [10,33]. Excessive sunlight exposure is known to degrade superficial skin blood vessels [42]. Chiou also re-analyzed published clinical data and found that microcapillary densities at three different skin sites all surprisingly followed first-order kinetics as shown in Fig. 1 for quick reference [10,33]. Furthermore, the regression line from the sun-exposed skin was parallel to those from sun-protected areas indicating total lack of damaging effect of sun exposure on capillaries [33]. The above discussion suggests that although skin tissues were bombarded with sun rays trillions of times, these lesions appeared amazingly to be fully repaired or removed. Taken together, the above data and discussion seem to present irrefutable evidence supporting notion of the dominant or overwhelming role of the intrinsic factor in driving skin aging in individuals who have never experienced sunburns in their lives. `This notion is different from a widely accepted view of “Long-term exposure to UV radiation causes premature skin aging (photoaging) characterized in part by wrinkles, altered pigmentation, and loss of skin tone” ; more discussion below [43].

Etiology of wrinkles and age or dark spots: Statistical impossibility, defense mechanism, plant experiment, mouse wrinkle model and human studies: Formation of wrinkles has been commonly accepted as a direct result of chronic, cumulative exposure to sunlight [33]. Such a concept seems highly problematic from a statistical point of view as the whole face, not just the wrinkled line, is exposed to sunlight. Chiou proposed that wrinkling in ordinary people

mainly results from the body's defense mechanism by reducing effective surface area in order to minimize water evaporation from the skin surface [33]. Such a concept is consistent with dramatic results (reproduced in Fig. 2 for quick reference) obtained from a recent experiment showing that a totally withered plant resembling deep facial wrinkle after withholding of watering for 3 days could be completely restored to the original beautiful texture just within 8 hours after watering [10]. The plant study seems highly relevant as genomic principles can be similarly applied to both humans and plants [44]. The dehydration and hydration experiments can be easily duplicated at home or in a laboratory by placing a flower or a vegetable with leaves on a table for about 1 to 3 days until it withers almost completely and then place it in a water container. The flower or vegetable should then be quickly restored or rejuvenated in hours to its original texture.

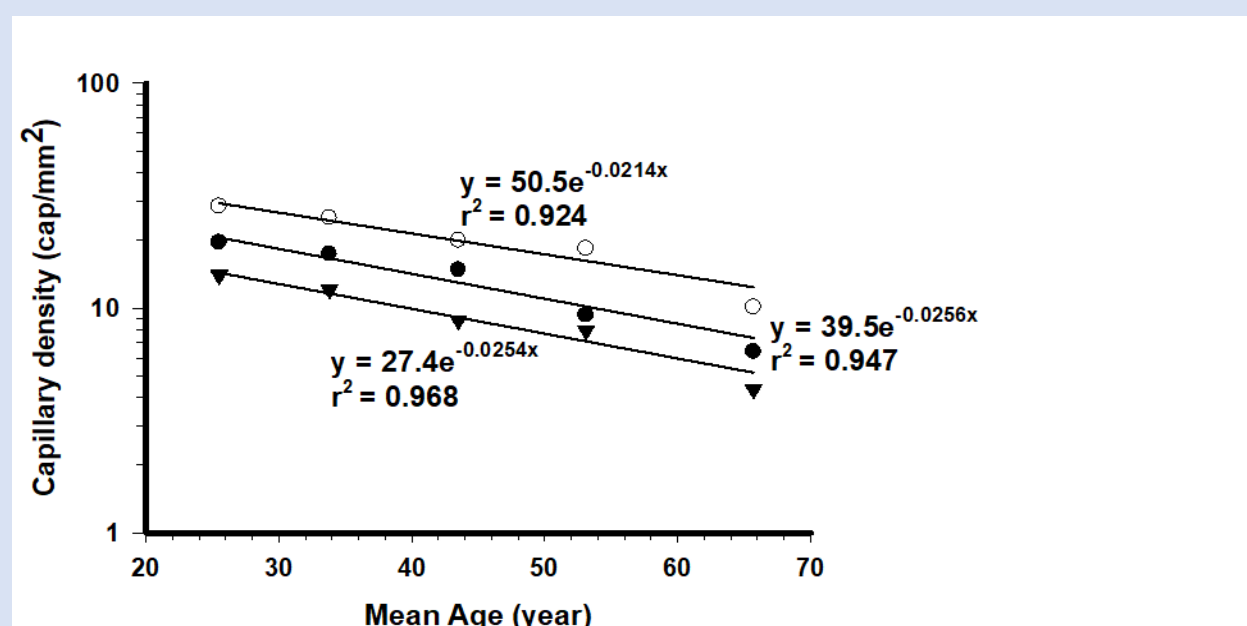


Figure 1: Semi-logarithmic plots of mean capillary density versus mean age of five female groups at three sites: volar forearm (●) representing the non-photo-exposed site; the back of hand (○) and forehead (▼) representing photo-exposed sites [33].



Figure 2: Plant Rejuvenation after water and nutrition [10].

The above dehydration theory for wrinkle formation is also consistent with an early study showing that a dehydrating phenomenon was also observed in mice following irradiation of UV rays before the appearance of wrinkles [45]. Skin dehydration also occurred in humans following irradiation of UVA rays [46]. Contrary to common belief that wrinkling occurs because of reduced collagen and elastin contents caused by sun damage, it was found to occur well before signs showing reduction of collagen and elastin in mice [30,32-34,45]. Such a finding is, however, totally expected in view of the potential extremely long half-life of collagen in mice; a mean decay half-life of 44 years in humans was reported [33]. The above discussion suggests that generally skin dehydration through reduced cardiac output with age is the major cause of wrinkling; intense UV radiation can cause both dehydration and degradation of cutaneous connective tissue thereby facilitating wrinkling process. Similarly, age or dark spot cannot be simply attributed to sun exposure and a born or acquired defect in microcirculation causing insufficient delivery of nutrients from the heart has been suggested as the major culprit for their initial occurrences [10,33]. Also, age spots can often occur on skin not exposed to sunlight and increase in number as one ages [10,33]. Furthermore, deep wrinkles can occur on skin totally protected from sunlight as shown in some Muslim women [9,10]. Prolonged exposure to wind causing skin dehydration may have negative effects on skin appearance and structure, an area largely unexplored [9]. In this regards, use of moisturizers alone was found to be capable of reducing wrinkles and pigmentation (more discussion below); this can also serve as evidence that wrinkling is mainly caused by deficiency in water, not by degradation of connective tissue from sunlight [47].

A renowned cover-story picture of unilateral dermatophilosis obtained from a veteran (28 years) truck driver has been attributed to chronic UVA radiation through the window showcasing the need of UV filters for UVA rays in sunscreens [48]. It is now postulated that the observed deeply-wrinkled skin was mainly caused by the dehydrating effect of sunlight passing through the window and by the wind blowing and sunlight radiation when the window was open for pleasant breezing [9]. In this regard, use of an effective moisturizer may be largely sufficient to prevent the problem (more discussion later).

Alternative interpretation of study from Dr. Gary Fisher: Bolus vs prolonged UV dosing responses: Dr. Fisher, et al., elegantly reported in 1997 that single dose (such as one standard erythema dose) or multiple low doses of UVB irradiation on human skin quickly caused elevation of activator protein-1, metalloproteinases and their activities [49]. Since these enzymes are responsible for the degradation of collagen and elastin (extracellular matrix), long-term exposure from sun light may result in reduction of collagen and elastin which can lead to wrinkled appearance [49]. The above notion is different from that predicted by the present non-cumulative UV damage hypothesis and from reported studies [9,33-36]. It is well known in pharmacokinetics, pharmacodynamics and therapeutics that bolus dose of a drug administered intravenously in seconds may yield pharmacodynamic or therapeutic response dramatically different from that produced by the same total dose given over a prolonged period of time such as 10 or 120 minutes [50]. Since the lowest bolus dose used in their dose range study showed negligible effect on enzyme induction, and this may be partly attributed to efficient repair of damage by the body's repair mechanisms, it is hypothesized that a slow constant UVB dosing over 30 or 120 minutes may result in negligible or insignificant induction of enzymes; a high bolus dose such as 1 or half standard erythema dose may instantaneously in seconds overwhelm the repair system and result in enzyme induction. It is hoped that such a critical experiment with potentially important far-reaching implications could soon be soon carried out by Dr. Fisher's laboratory. By the same token, use of bolus dosing and prolonged (such as over 2 to 8 hours) dosing may also yield entirely different results in studies of molecular mechanisms of skin cancers by UV radiation, an area hitherto seemingly largely unexplored. In order to simulate the real-world situation, it is suggested that the intensity of artificial sunlight, UV rays, UVA or UVB rays used in various studies should be ideally identical to that of UV Index 2, 3, 4 or 5 for chronic exposure. It is of interest to notice the following statement reported from the laboratory of Dr. Fisher in a 1995 study "Chronic UV exposure does not lead to long-term elevations for these metalloproteinases" [51]. This statement seems to be entirely consistent with the present non-cumulative DNA lesion notion for people practicing healthy sun exposure (i.e., no elevated enzyme levels in sun-exposed skin) and inconsistent with his later study discussed above (i.e., expected elevated enzyme levels in sun-exposed skin as reported in his study shown in reference). The reason for the above apparent discrepancy from the same laboratory is unknown [49].

Moisturizers as safe, effective, age-reversing, anti-photoaging and anti-cancer agents: Since intrinsic skin aging can result in decreased delivery of water to the skin and prolonged exposure to sunlight can lead to gradual evaporation of water from skin surface as discussed above, it is then envisioned that a moisturizer may also function as an effective anti-photoaging/anti-cancer agent just like a sunscreen [33]. An aqueous solution containing 50% (w/w) glycerin was earlier found to have an SPF of 2 from an independent testing laboratory (unpublished data). This is interesting in view of the following: First, a product with an SPF 2 should be generally adequate for protection from sunlight especially for people of color [9]. This is because it can effectively reduce the sunlight effect by half, for example, from a UV Index 10 to UV Index 5 that is generally non-burning to Blacks, Hispanics and Asians [9]. For Caucasians it can reduce UV Index from 6 to 3 or 8 to 4 that may be considered to be generally safe [9]. Second, glycerin is extremely safe and non-irritating and has a moisturizing effect lasting up to 10 to 24 hours [52]. Third, it has a skin-firming property probably due to its topical rejuvenation of stem/progenitor cells [9,10]. Propylene glycol was also found to have similar properties [53,54]. It is likely that other moisturizers like hyaluronic acid may also have similar properties; for example, a topical nano-hyaluronic acid product has been found to reduce skin roughness, decrease the depth of wrinkles by up to 40%, increase skin hydration by up to 94 % and enhance skin firmness and plasticity by up to 55% [47]. Hence, safe and effective moisturizers may represent an exciting new area for future research to reverse skin aging and to prevent intrinsic and extrinsic aging as well as skin cancer. Most importantly, compared to sunscreens they are much safer to human health and also environmentally friendly and may not require re-application every 2 hours recommended for use of current sunscreens [3,55]. Of note, the tissue regenerative property of propylene glycol can be usually demonstrated in a few days by applying an aqueous solution of about 30 to 70% to the cuticle area of big toenail 2 to 3 times a day [54].

Inclusion of blood and water in skin aging exosome: Anti-photoaging, rejuvenation of topical stem/progenitor cells: It is known that the composition and flow rate of blood in the body can have profound effects on the aging and vitality of tissues perfused [33]. In this regard the function of stem cells and progenitor cells in tissues may be highly reversible and depends on blood flow as shown in parabiotic studies in animals and massage studies in humans [56,57]. Administration of progenitor cells from young hearts to old hearts of rodents was found to quickly rejuvenate not only the heart but also other organs and tissues including the skin; this suggests great importance of blood supply in affecting aging and aging reversal [58]. It is proposed that blood supply and water be included in skin aging exosome for future research [59].

Vehicle (placebo) effects ignored in most studies: Topical products had been routinely used in the treatment of acne for many decades without realization that vehicle alone might contribute up to about 90% of their anti-acne activities [60]. A preliminary review of literature indicates that potential vehicle effect has been rarely considered in the evaluation of sunscreen efficacy

for preventing photoaging and skin cancer. This may be especially significant in view of the role of even water in wrinkle formation as discussed above. Failure to consider the potential vehicle effect may result in incorrect interpretation of experimental data.

Implications of different patterns of sunscreen use in tropical and non-tropical countries: In countries like Australia and New Zealand, sunscreens are used regularly for sun protection while in the US and many other Western nations with a temperate climate, sunscreens are primarily used for intense intermittent sun exposure especially for males [61]. Therefore, one should be very careful in extrapolating results obtained from one country to another country without realization of dramatic differences in use pattern that can result in different outcomes. It seems that to date, this issue has been largely not considered in the drug approval process or in the literature.

Health Benefits of Moderate Sun Exposures

Reported potential benefits from moderate sun exposure are numerous and increasing yearly. It has been extensively reviewed and only a few points will be briefly highlighted below [9,18,61,62].

Protection against skin cancers: Chronic exposure to sunlight has been generally accepted to be protective against melanoma in the last two or three decade's especially in countries with temperate climate. Moore recently, outdoor workers in Sweden and Denmark were also shown to have lower risks of basal cell carcinoma compared to indoor workers [9,10,61,63]. These reports are quite interesting because they reversed the previously observed trend with higher cancer rate for outdoor workers. Since melanoma is the deadliest skin cancer and exposure to sunlight can be protective against it, it would be reasonable to argue that it can also protect against other similarly SS-induced skin cancers such as basal cell carcinoma and squamous cell carcinoma. The reasons for the protection may be mainly due to increased thickness and tanning of skin as well as enhanced immunity [9,10,61,62]. More interestingly, contrary to our common belief, radiation by UVA and visible light (320-500 nm) has been shown to decrease the formation of melanotic tumors from UVB radiation in opossums, although its clinical significance remains to be determined [64].

Protection against other cancers: Epidemiological and experimental evidence has shown that chronic exposure to sunlight could contribute to prevention of colorectal cancer, breast cancer, prostate cancer and non-Hodgkin Lymphoma [18].

Other health benefits: Other benefits include enhancement of vitamin D synthesis, reduction of osteoporosis, blood pressure, heart attack and stroke, prevention of multiple sclerosis, and metabolic syndrome, mood and sleep improvement, and immunity enhancement. In this Covid-

19 pandemic it was reported that sunlight exposure may reduce Covid infection and mortality [9]. Interestingly, it was reported that the risk of avoiding sunlight is equivalent to smoking in terms of reducing life expectancy [65]. Incidentally, sun exposure has been traditionally used for the prevention and treatment of some diseases like psoriasis, eczema, vitiligo and acne [61].

It has been shown that UVA could impair the DNA repair capacity for UVB-induced DNA lesions and may increase the chance of getting skin cancer [66]. This would provide justification for using broad-spectrum sunscreens and tinted glass window for protection against UVA radiation [66]. Since this would not affect the etiology of skin cancer, therefore this should not increase the chance of getting cancer for those practicing healthy sun exposure; in other words, no negative impact would occur. UVA has been also known to cause vascular dilation resulting in decrease of blood pressure, heart attack and stroke; this would call for exposure to UVA or sunlight for better health [18,67]. It is felt that the benefit from this alone should outweigh the risk of getting too much UVA especially for non-white populations whose incidences of skin cancer have often been described as rare compared to white populations [9]. The above discussion seems to further underscore the importance of practicing healthy sun exposure for all people including those with immune deficiency. Thus, in general, too little sun and too much sun are all not beneficial to good health and there is an urgent need to abandon the current sun avoidance policy for the general public [18,61,62,66].

It is important to note that unlike UVB, UVA-induced tanning commonly employed in tanning salons does not protect against photoproduct formation from UV radiation [68]. This and reduction of DNA repair may suggest that use of simulated sunlight or UVB should be much preferred for tanning in tanning salons [66].

How to Obtain Healthy Sun Exposure without Getting Sunburned?

The above discussion suggests that a healthy sun exposure can generally help prevent skin cancer without resulting in any significant photoaging. Obviously, there are many factors that may affect people on how to obtain healthy sun exposure. They include, for example, skin types, age, colors of eyes and hair, UV Index, time of day, season of the year, latitude and altitude, snow, clouds, immune function and genetics, as well as the length of exposure and use of sunscreen. Therefore, practical implementation of the proposed new preventative method can be highly individualized. Some general tips for people of whites and colors and for whites have been reported [8-10,18]. It is to be noted that intermittent sun exposure without getting sunburned may be generally considered as healthy sun exposure [69].

Conflict of Interest

The authors declare that they have no conflict of interest.

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