Narrative Review Article

Impact of Vitamin D Production on Skin Cancer Risk: Associations With Dietary Intake and Geographical Factors

Angela Han

Port Moody Secondary School, 300 Albert St, Port Moody, BC V3J 4W7, Canada

ABSTRACT

Cutaneous malignant melanoma (CMM) and non-melanoma skin cancers (NMSC), including basal cell carcinoma (BCC) and squamous cell carcinoma (SCC), are the most frequent types of cutaneous cancer. NMSC diagnoses comprise more than one-third of all cancers. Ultraviolet (UV) exposure is a primary requirement to produce vitamin D for individuals. However, this exposure is accompanied by an increased risk of skin cancer. Some studies have observed that vitamin D synthesis may protect against skin cancer, but the relationship remains debated in the scientific literature. Alongside studies that suggest a protective role of vitamin D in skin cancer, there have also been observations related to the connection between vitamin D derived from other sources such as dietary and environmental factors, and cancer risk. The role of dietary habits and nutrient intake in skin cancer risk has gained attention in recent years, as the two naturally occurring forms of vitamin D, ergocalciferol and cholecalciferol, are found in food. However, the association between dietary vitamin D intake and skin cancer risk remains controversial. Current findings lack clarity regarding whether sun-induced vitamin D production varies in locations observed to be at a higher risk for cancer due to geographical factors. There has been difficulty examining an independent influence of vitamin D status on skin cancer risk due to confounding and contrasting effects of sun exposure and other factors such as dietary vitamin D. Additional research is needed to confirm the preventive role of vitamin D in skin cancer risks, and to eliminate potential confounding variables.

Keywords: Skin cancer risk; Non-melanoma skin cancer; Cutaneous malignant melanoma; Ultraviolet exposure; Vitamin D; Dietary intake; Geographical location

INTRODUCTION

Sun exposure is relevant to many individuals. Whether it is associated with a line of work and occupation or daily walks outside, people are routinely exposed to ultraviolet (UV) radiation. Conversely, skin cancer (including cutaneous malignant melanoma (CMM), basal cell carcinoma (BCC), and squamous cell carcinoma (SCC)) is one of the most common forms of cancer (1, 2). It is reported that non-melanoma skin cancer (NMSC) alone accounts for at least 40% of new cancer cases. Due to population aging, the incidence of skin cancer is projected to rise in the coming decades (1).

CMM and cancers classified as NMSC, including BCC and SCC, are types of skin cancers considered "sun-related" (mainly UV-related) cancers (3). A large proportion of national tumor registries do not consider NMSC, because of their high frequency and

Corresponding author: Angela Han, E-mail: Angela.han.cn@gmail.com.

Accepted May 11, 2026
https://doi.org/10.70251/HYJR2348.438697

supposedly low impact (3). Even so, NMSC is still the most commonly diagnosed cancer in North America, Australia, and New Zealand. An estimated number of over 1 million (1,042,056) new NMSC cases were diagnosed worldwide in 2018 (3). The incidence of SCC seems to be increasing as well. CMM is considered the most harmful skin cancer out of the three main types (3). CMM incidence has increased in most developed countries, especially among males. The differences among male and female rates are controversial, as behavioral, genetic, and hormonal factors may play a role (3).

Vitamin D is synthesized through sun exposure and can also be obtained through diet and supplementation, such as vitamin D-rich foods. Modern indoor lifestyles, combined with intermittent intense sun exposure, makes a major contribution to vitamin D deficiency in the population. Furthermore, the current vitamin D deficiency epidemic is accompanied by an increase in global skin cancer incidence. UV exposure leads to environmental risk factors for CMM and other NMSCs. Some studies have observed that vitamin D synthesis may protect against NMSC. The optimum vitamin D dose to reduce skin cancer risk has not been confirmed yet. As a result, there are still controversies about vitamin D and skin cancer.

Vitamin D is a prohormone, with two major forms: D3 and D2. Vitamin D2 and D3 can be acquired from dietary intake (3). Vitamin D3 may also be synthesized endogenously by photochemical modification in the skin through UVB radiation (3). This cutaneous synthesis depends on factors such as solar UV index, amount of sun-exposed and sun-protected skin, amount of time under the sun, the body mass index, age, and skin phototype. Vitamin D deficiency is an increasingly concerning global issue (3). Optimal levels are not well defined, even for bone metabolism, and especially among non-Caucasians, who usually have greater bone mass despite generally having lower serum vitamin D levels. Vitamin D levels tend to be lower in the fairest phototypes among Caucasians (3). This is partially due to higher photosensitivity and less sun exposure, but there are other factors such as vitamin D metabolism. However, certain populations show higher serum vitamin D among individuals with the fairest phototypes and pigmentation traits (3). Some studies suggest the possibility of vitamin D's association with decreased risks of skin cancer diagnosis, but no firm conclusion has been made, and the relationship remains inconsistent in the scientific literature.

This review examines the variations of vitamin D production effects, connected through UV exposure, and their relevance on skin cancer. It also explores the connections between vitamin D, diet, and geographic factors to consolidate recommendations that can maximize essential vitamin D production for proper growth required for the body while limiting cancer development.

ULTRAVIOLET RADIATION AND SKIN CANCER

Introduction to Ultraviolet Exposure and Radiation

A combination of both non-modifiable (e.g. genetic) and modifiable (e.g. environmental) risk factors contributes to most skin cancers. Exposure to ultraviolet radiation (UVR) is one of the most common and well-known modifiable risk factors for skin cancer (4). Nearly all skin cancers are related to UV exposure in some way. Melanocytes, stimulated through UV exposure, produce melanin. Melanin can appear as tanned skin, which indicates damage to the skin, skin cells, and DNA. Sunburns, which indicate cell death, can be due to more intense exposures (4).

The three subtypes of UVR consist of UVA, UVB, and UVC. Each type of UVR differs in skin penetration. UVA rays are responsible for premature skin aging and penetrate deep into the skin through the epidermal junction where the melanocytes reside in the basal layer (4). They are also connected to potentially mutagenic oxidative DNA damage (3). Both UVA and UVB exposure can result in a tanned appearance. UVB increases melanin production that confers a minimal amount of photoprotection (equivalent to about SPF 3), resulting in a tan and indications of damage to the skin. For all skin cancers, UVB radiation is considered the most important environmental risk factor (3). Lastly, UVC rays are the shortest and are absorbed by the ozone layer and the atmosphere (4).

The Types of Skin Cancers

NMSCs are increasing worldwide, and comprise more than one-third of all cancers (5). There are two common types of NMSCs, including BCC and SCC. These cancers, including CMM, are the most frequent types of cutaneous cancer, also considered "sun-related" (mainly related to UV) cancers (3). BCC and SCC account for about 90% of all skin cancers diagnosed globally, and occur at a ratio of about 4:1 (5). Precise statistics of NMSC are generally not available as these cancers

are not reported to cancer registries in most countries (5). Because of the high frequency and apparently low impact of these cancers, a large proportion of national tumor registries do not consider NMSC (3). Despite this, NMSC cases are large, and it is estimated that between one and three million people are diagnosed worldwide each year (5). In 2018, there was an estimate of 1,042,056 new NMSC cases that were diagnosed worldwide, alongside 65,155 deaths (about 6% of those diagnosed) attributable to NMSC (mainly SCC) (3). It is important to note that global incidence rates vary by skin complexion and geographical region. However, due to growing exposure to UV sunlight related to increased sun-seeking behaviors and depletion of stratospheric ozone, cases are expected to continue to rise in the coming years (5).

Skin Cancer Beyond Sun Exposure

UV rays can cause DNA damage, which can lead to inflammation and secondary immunosuppression. These mutations are frequently identifiable in tumor suppressor genes (3). The risk of CMM can be increased due to sunburns which are usually caused by common types of sun exposure, such as sunbathing or sunny outdoor activities for typical individuals who spend time outside occasionally. Photoaging, cutaneous immune suppression, NMSC, and lentigo maligna CMM subtypes are more serious aftermaths of skin damage that may be caused by chronic sun exposure (3).

Intense UV is exposed to users to tan the skin for cosmetic purposes in indoor tanning (4). Tanning devices usually emit UV exposure significantly more intensely than the UV exposure individuals come in contact with outdoors (4). UV-emitting tanning devices have been classified as group 1 carcinogens by the World Health Organization (3). Recreational indoor tanning can be seen as a health hazard, known to cause premature photoaging and has been proven to increase the risk of BCC and SCC (3). Such devices should be used with caution.

VITAMIN D

Vitamin D Production in the Body

Vitamin D can be obtained from direct sunlight, which prompts our skin to produce this essential vitamin. Exposure to UVR synthesizes most vitamin D. This happens in a process of conversion from 7-dehydrocholesterol to vitamin D3 (6) by photochemical modification in the skin with UVB radiation (3). Vitamin D is important for health and well-being as it ensures bone strength (7). It is a fat-soluble vitamin which occurs in two types of natural forms; ergocalciferol (vitamin D2) and cholecalciferol (vitamin D3) (6). Alongside its role in bone health, vitamin D also plays an important role in different biological functions such as anti-proliferation, anti-angiogenesis, and immune system modulation (6).

Vitamin D is first converted in the liver into 25-hydroxyvitamin D [25 (OH) D], also known as calcidiol, through a process called hydroxylation. Vitamin D levels are assessed by measuring the serum concentration of 25 (OH) D (7), which has a long circulating half-life of about 2 to 3 weeks (8). Vitamin D dosage is estimated through this measurement because this is the main circulating form (8). This form of vitamin D circulates in the bloodstream.

Vitamin D Sufficiency: How is Vitamin D Obtained?

When serum concentrations of calcidiol 25 (OH) D are below 75 nmol/L, hypovitaminosis D occurs. Vitamin D deficiency is classified when serum concentrations are below 50 nmol/L. This deficiency is associated with increased risk of poor bone health. When serum concentration is between 50 and 74 nmol/L, it is considered vitamin D insufficiency, and between 75 and 125 nmol/L as sufficiency (8). However, a vitamin D concentration above 125 nmol/L can cause other risks too (7).

Sun exposure, mainly UVB, produces around 90% of vitamin D in the skin (3). Because the production of vitamin D is dependent on UVB absorption, some studies suggest that geographic location plays a considerable factor when determining the need for increased vitamin D (9).

The two natural occurring forms of vitamin D, ergocalciferol and cholecalciferol, may be found in food. This includes some food groups such as fish, dairy products, and cereal products, fortified foods such as some dairy and cereal products, as well as dietary supplements (6).

Differences in Vitamin D Production Amongst Individuals: Skin Color

Skin color may have detrimental effects and contribute to vitamin D production. The Fitzpatrick scale is used to measure skin color qualitatively. Vitamin D production decreases when the Fitzpatrick type goes from lighter skin to darker skin, increasing from I to VI (9). Compared to lighter skin, darker skin contains a higher melanin level, providing UVR protection. This suggests that the same amount of vitamin D generation in lighter skin requires lower doses of UVR compared

to the same amount of vitamin D generation in darker skin (9). Scale types I and II contain lower minimal erythema doses, where less amount of UVR is needed to induce sunburn. Although this boosts higher amounts of vitamin D production through higher amounts of UV penetration, the risk of developing skin cancer increases too. Types V and VI have higher minimal erythema doses. Individuals with darker pigmented skin may experience vitamin D deficiency due to the differencing UV absorption circumstances (9). 30 minutes of daily sun exposure allows individuals with lighter skin to generate serum levels of 25 (OH) D above 50 nmol/L, while the process takes over two hours of sun exposure for individuals with darker skin to achieve the same amount of vitamin D production (9). These differences in vitamin D synthesis across skin types may also influence variability in skin cancer susceptibility observed across populations.

THE RELATIONSHIP BETWEEN VITAMIN D AND SKIN CANCER

Vitamin D Levels as a Potential Helpful Solution?

Moderate UV exposure plays an important role for the production and preservation of adequate vitamin D levels (5), yet it is also the primary environmental risk factor for skin cancer which leads to difficulty in distinguishing whether observed effects are due to vitamin D itself or UV exposure (4). Despite this, some research argues that there is a correlation between vitamin D and skin cancer protection.

A study published in 2019 conducted an evaluation report on a population of active fishermen older than 18 years of age, regarding sun exposure and vitamin D production. The observational and analytical study, which consisted of 174 individuals of both genders, focused on the clinical changes in the skin caused by constant sun exposure (8). The results of the study showed that there was a low frequency of cases of vitamin D deficiency among the participants. This is speculated to be due to the absence of the habit of using sunscreen, as patients who use photoprotection are more likely to experience vitamin D deficiency (8). The fishermen were exposed to the sun every day in 91.83% of the cases, and worked 21 to 28 hours per week on average (32.19% of the time).

It was reported that 87.7% of the patients in the study had vitamin levels above 30 ng/mL (75 nmol/L). It was mentioned that sunlight is a big factor in vitamin D production, as most individuals in the study obtained an adequate amount of serum levels (8). In the study, individuals with levels of 25 (OH) D of 32 ng/mL (>75 nmol/L) were also noticed to potentially have a 40% lower risk of developing NMSC. The data results suggested a low incidence of NMSC in the fishermen may have been due to the elevated levels of vitamin D. This indicates the possibility of higher levels of 25 (OH) D becoming a protective factor for the development of skin cancers (8). The results of the study showed protective effects involving vitamin D in many diseases, including skin cancer, demonstrated by the production and implementation of vitamin D (8).

Some studies suggest that the endogenous production of vitamin D obtained through exposure to UVB rays can inhibit protective, antitumor, and photoadaptive activities against solar radiation exposure after the initial exposure. However, it is important to note that the relationship between sun exposure, vitamin D levels, and clinical skin changes caused by UV exposure was not statistically significant (8).

Vitamin D Status Effects on Skin Cancer

Studies have shown that vitamin D alters cancer cell differentiation, proliferation, and apoptosis in vitro and preclinical animal models. This makes vitamin D a candidate agent for cancer regulation (3). It is still unclear whether vitamin D can limit progression or prevent cancer in humans.

Because of vitamin D's effects on the regulation of growth, cell death, angiogenesis, and cell differentiation, it may play a role in cutaneous carcinogenesis (3). Vitamin D is considered to possibly have the abilities to be a tumor-suppressing agent in the skin. There is an increase in speculation that it has protective actions against UV-induced epidermal cancer formation, as vitamin D receptor (VDR) has variants that are thought to alter its function when encoded by a gene located on chromosomal region 12q13 (3). From a study of the relationship between vitamin D levels, polymorphisms in the vitamin D receptor, and dietary supplementation with the incidence and survival of various neoplasms, there has been an evaluation of actions of vitamin D protecting against cutaneous cancer (3).

Studies have made efforts to determine an effective amount of vitamin D daily intake for protection against cancer. It was shown that male cancer mortality rate can be reduced by 30% with daily doses of 1500 international units (IU) of vitamin D3 in the United States (3). There have also been attempts to relate incidence of some cancers to blood levels of vitamin D3 (25-OH vitamin D) (3). To compare, studies used levels of 75-

87.5 nmol/L (30-35 ng/mL) as the minimum values. The serum levels of vitamin D used for reference are values recommended to obtain the maximum benefits of vitamin D (3). It has been clinically proven that adequate vitamin D serum levels have the ability to protect against multiple malignancies. Studies have shown findings in different tissues, and in vitro, in animal models and in cell culture (3). On the other hand, evidence in findings conflict with each other. The capability of vitamin D in protecting against skin cancer is not supported by enough epidemiologic evidence.

UV-Induced Vitamin D

Vitamin D production obtained from regular sun exposure has also been suggested to yield a protective effect on different types of internal cancers. Epidemiological studies based on environmental and occupational factors have shown connections between sunlight exposure and a reduced risk for cancers including colorectal, breast, kidney, CMM, and non-Hodgkin lymphoma (5). Several studies suggest that vitamin D immune-modulatory mechanisms and regulatory effects on the cell cycle play a protective role against various cancers (5). However, evidence from a nested case-control study contradicts such suggestions. The study, based on Swedish population registries, compared around 100,000 patients with BCC with around 1 million control patients. They found that patients with BCC (example of people with more sun exposure and therefore producing vitamin D) could be found with a higher risk of being diagnosed with other cancers before BCC (3). These findings suggest that higher vitamin D levels may be correlated to an increased risk of BCC and CMM. However it cannot be confirmed if increased skin diagnosis in people with high vitamin D levels is not due to sun exposure.

A recent meta-analysis published in 2020 compared thirteen prospective studies which suggested the association of vitamin D status with greater risks of CMM, and NMSC (6). Each serum level of 30 nmol/L increment in 25 (OH) D was revealed to be associated with 42%, 30%, and 41% increased risks of CMM, SCC, and BCC (6). The findings suggest that increased risks of CMM, SCC, and BCC are associated with a higher vitamin D status. However, sun exposure may be a confounding factor in higher risk of CMM, SCC, and BCC as serum levels in 25 (OH) D are mainly obtained from sun exposure (10). As a result, differences in adjustment for UV exposure across studies may therefore explain contradictory findings in literature (6).

Vitamin D and Non-Melanoma Skin Cancer (NMSC)

Evidence of a non-linear dose-response association with risks of NMSC and higher 25 (OH) D levels has been found. Current findings suggest that there is a consistent trend that higher levels of vitamin D are significantly associated with a higher incidence of skin cancer. In the non-linear trend analysis, the peak hazard ratio seemed to be around a vitamin D status of 80 nmol/L (6). However, current evidence is still lacking due to missing information on the range of vitamin D levels, and is unable to define if vitamin D may decrease the risks of getting NMSC. Furthermore, the results may have been related to the dual effect of UVB – allowing vitamin D synthesis but also the generation of DNA damage – as previously stated (3).

Basal-Cell Carcinoma (BCC) and Squamous-Cell Carcinoma (SCC)

In an 11-year prospective study published in 2012, the association between vitamin D status and NMSC risk was assessed. The study took place in an Australian subtropical community. Results suggest that higher vitamin D serum levels (above 75 nmol/L) are associated with increased risk of BCC, while lower levels (50-75 nmol/L) are most likely not associated with increased risk (11). These findings indicate that serum levels above 75 nmol/L may be associated with an increased chance of morbidity due to skin cancer in a group of normal, community-based adults (11). Observations indicate that this applies most for individuals with a past history of skin cancer (11).

The meta-analysis, previously referenced above, showed a slightly increased risk of BCC among individuals who received at least 100 daily IU of dietary or supplemental vitamin D (6). Vitamin D also showed no benefit in preventing BCC in a randomized clinical trial of supplementation (3). An observation from another study contradicts these findings, suggesting that obtaining serum levels of vitamin D above 60 nmol/L may reduce BCC recurrence rates significantly (3). Higher vitamin D serum levels and increased incidences of SCC have been observed as well (may have been confounded by excessive photodamage). However, some epidemiologic evidence suggests that SCC may be prevented from vitamin D supplementation (3).

Critical Appraisal Of Confounding Data and Conflicting Evidence

The differences in study design, population characteristics, and statistical power explain inconsistent

findings found across studies that examine vitamin D status and skin cancer (Table 1). Observational studies including cohort and case-control designs are especially susceptible to the difficulties of isolating the effect of vitamin D itself. Cross-sectional or snapshot studies have limitations to capturing long-term exposure or latency periods relevant to carcinogenesis as they measure vitamin D at a single time point. In contrast, longitudinal studies generally provide higher evidential weight along with stronger temporal relationships.

Compared to observational studies, randomized controlled trials (RCT) provide causal inferences. However, its findings do not strongly support a protective effect of vitamin D supplementation on skin cancer risk. 25,871 participants took part in the large-scale VITAL trial where individuals were randomized to vitamin D3 supplementation of 2000 IU/day versus placebo over a median follow-up of around five years (12). The primary statistical outcomes of the trial did not demonstrate a significant total cancer incidence reduction, and further secondary analyses did not show a clear protective effect against skin cancer (12). However, statistical power for skin-cancer specific analysis is reduced as there are relatively low event rates for skin cancer outcomes available to interpret. Similarly, a more recent RCT evaluated vitamin D supplementation and did not report any significant reduction in skin cancer incidence or recurrence compared with placebo (13). Findings from both articles suggest that skin cancer risk is unlikely influenced by vitamin D supplementation alone in controlled settings. Results of the RCTs contrasts with mixed associations found in some observational studies, supporting the role of confounding factors such as UV exposure and dietary patterns rather than the effect of vitamin D itself.

Reliability of studies is further influenced by sample size. Subgroup analyses may produce inconsistent estimates with higher likelihood of false-positive or

Table 1. *Summary Of Key Studies Examining Vitamin D, UV Exposure, Diet, and Skin Cancer Risk.*

Study	Study Design & Population	Exposure Type	Main Findings
Coutinho *et al*. (8)	Observational; Brazilian fishermen	Chronic UV exposure	High vitamin D; low NMSC incidence; possible protective association
van der Pols *et al*. (11)	Prospective cohort; Australian adults (11-year)	Serum vitamin D	Higher vitamin D (>75 nmol/L) associated with ↑ BCC risk
Mahamat-Saleh *et al*. (6)	Meta-analysis (13 studies)	Serum vitamin D	Higher vitamin D associated with ↑ CMM, SCC, BCC risk
Caini *et al*. (10)	Systematic review/meta-analysis	Vitamin D intake + serum	Inconsistent associations; possible ↑ NMSC risk
Malagoli *et al*. (16)	Case-control; Italy	Diet	Certain foods protective (legumes, olive oil); others ↑ risk
Ibiebele *et al*. (20)	Prospective cohort	Dietary patterns	Meat/fat ↑ SCC; fruit/veg ↓ risk
Vinceti *et al*. (22)	Case-control; Italy	Dietary vitamin D	Inverse association with melanoma
Gamba *et al*. (21)	RCT (WHI dietary trial)	Low-fat diet	No strong BCC association
Manson *et al*. (12)	Large RCT (~25,000 participants)	Vitamin D supplementation (2000 IU/day)	No significant reduction in cancer incidence; limited skin cancer effect
De Smedt *et al*. (13)	RCT; melanoma patients	High-dose vitamin D supplementation	No improvement in melanoma outcomes
Surdu *et al*. (5)	Multinational occupational study	UV exposure	Occupational UV ↑ NMSC risk
Qureshi *et al*. (24)	Cohort; U.S. women	Geographic UV	Latitude variation affects risk
Berman-Rosa *et al*. (25)	Ecological; Canada	Environmental + UV	Higher UV/temp ↑ melanoma incidence

false-negative associations, whereas studies with larger sample sizes reduce the likelihood of misinterpreted associations and thus have greater power. Specifically, studies with restricted populations such as occupational groups like fishermen may have higher variability in results. Additionally, findings can diverge across studies even when studying similar endpoints due to population variability that can act as effect modifiers, including latitude and supplementation use. Overall, a combination of confounding factors, variability in exposure measurement, and differences in study design are all potentially important elements that play a role in conflicting evidence.

MODIFYING FACTORS: POTENTIAL ASSOCIATIONS WITH VITAMIN D PRODUCTION, AND SKIN CANCER RISK

Obtaining Vitamin D From a Food Source

Although much of the evidence focuses on vitamin D produced through sun exposure, vitamin D can also be obtained through dietary sources. Examining dietary intake may help clarify whether observed associations are due to vitamin D itself or confounded by UV exposure. As a fat-soluble micronutrient, vitamin D-rich foods provide several health benefits (14). Vitamin D plays a role in the regulation of bone metabolism, maintenance of calcium and phosphate homeostasis, which increases the gut absorption of calcium and phosphate (14).

The best food sources of vitamin D include the flesh of fatty fish (such as trout, salmon, tuna, and mackerel) and fish liver oils (15). When measured as IU per 100 grams (IU/100g), the category of fatty fish and fish oils ranges from 632-10,000 IU/100g (14). The diet of an animal also affects the amount of vitamin D in its tissues (15). Foods such as beef liver, egg yolks, and cheese contain small amounts of vitamin D, usually in the form of vitamin D3 (15). In addition to vitamin D3, animal-based foods often provide some vitamin D in the form of 25 (OH) D (15). Eggs (raw and hard boiled), dairy alternatives (soy yogurt/milk), pork, and dairy products (including milk, butter, cheese and yogurt) contain around 30-115 IU/100g (14).

The Relationship Between Dietary Intake and Cancer Risk

The role of dietary habits and nutrient intake in skin cancer risk has gained attention in recent years. Trends of higher intake of beta-carotene and vitamins A, C, D, E associated with reduced skin cancer risk have been shown in several epidemiological investigations (16, 17).

Healthy dietary intake such as vegetables, fruit, and fish have been suggested to have a protective effect. Consumption of specific foods in dietary patterns may be associated with detrimental effects, such as alcohol, sugars/carbohydrates, and diet high in glycemic load (16, 17).

Dietary risk factors of CMM and the population in Northern Italy have been assessed in a recent population-based case-control study. The study was based in the provinces of Bologna, Ferrara, Modena, Parma, and Reggio Emilia, where resident patients with newly diagnosed CMM were attending the local dermatological clinics (16). Frequency and quantity of daily consumption for each food item were collected from each participant (16). Findings suggest that higher consumption of cereal products, sweets, and cabbages are associated with increased CMM risk. On the other hand, higher intake of legumes, olive oil, eggs, onion, and garlic are connected to a decreased risk (16).

High consumption of foods characterized by high contents of refined flours and sugars results in a high glycemic index (16). Foods that contain high glycemic index have been observed to be associated with increased cancer risk. Possibly due to increased postprandial glucose and insulin levels, the association has been observed at sites such as colon-rectum, breast, and endometrium (16). High circulating levels of leptin (involved in glucose metabolism and obesity, females, and insulin levels) have been suggested to possibly play a role in CMM development (16). In the study, it was found that high intake of cereals and sweets could increase CMM risk, especially in women (16). This supports previous findings based on observations of the glycemic load of dietary intake.

Conversely, dietary patterns of high vegetable consumption have a beneficial influence upon cancer risk, and possibly CMM. Observations of the study suggest an indication of onion, garlic, and vegetable consumption containing a protective effect on CMM risk, especially in women and in younger participants (16). However, trends of cabbage consumption and associations with higher CMM risk were found (16). Additionally, these findings conflict with past studies that suggest the connection between the intake of Brassicas species (all types of cabbages, broccoli, cauliflower, brussel sprouts) and a possible protective role (16). Though, heavy metals such as cadmium, in which cabbages are a possible source of, has been associated with increased CMM risk (16).

The study has also observed a correlation between increased consumption of legumes, olive oil, and eggs, and decreased risk of CMM (16). Eggs contain the major dietary sources of vitamin D, which could influence a protective effect on CMM risk (16). However, high intake of other vitamin D-rich foods including fish and dairy products did not show a clear association with disease risk (16). Legumes, consisting mainly of beans and peas, are observed to possibly be able to prevent CMM carcinogenesis in initiation and progression phases (16). Protection against cancer and other inflammation-related diseases may be associated with olive oil consumption (16). This has been evident in several other studies. The preventive activity of olive oil has been related to phenolic compounds. The ability to protect against DNA damage has also been demonstrated in olive oil through several other studies (16). Food categories such as fish and fruit were not associated with CMM risk (16). The association of meat and dairy products with disease risk was not detected, and still remains unclear (16). Although previous studies have found negative and positive associations with coffee, fruit juices, and alcohol (18), no beverages assessed showed a clear association with CMM risk (16).

Some studies have also observed and suggested a direct relation between dietary intake of fatty acids (particularly polyunsaturated fatty acids, known as PUFAs) and CMM risk (19). However, evidence did not suggest a major role of fatty acids on the risk of CMM (19).

A study on the association between dietary pattern and skin cancer based in Nambour, Australia, identified two major dietary patterns (20). It was discovered that the development of SCC tumors were positively associated with meat and fat patterns (20). The association was observed to be stronger in participants with skin cancer history. In contrast, there was a trend of decreasing SCC tumor risk that accompanied a higher intake of vegetables and fruits (20). This supports the trend of vegetable consumption containing a protective effect on cancer found in previous studies, specifically SCC risk. However, this contradicts another study that suggests no association between vegetable intake and the risk of NMSC (21). No association between the dietary patterns and BCC tumors were found (20).

Interpretation and Limitations of Dietary Vitamin D Evidence

Current evidence suggests that an adequate amount of vitamin D should be obtained from a healthy diet instead, as unprotected sun exposure should be avoided in order to reduce underlying risks (6). Alongside studies that suggest a protective role of vitamin D in skin cancer, results of the association between dietary intake of vitamin D and risk remains controversial (14, 22).

Difficulty in separating UV-induced vitamin D synthesis from the independent effects of dietary vitamin D may be an explanation for these conflicting findings. It is primarily challenging to determine whether vitamin D itself or UV exposure is associated with observed cancer risks, if any, as individuals with higher vitamin D levels often experience greater sun exposure. In addition, dietary patterns are often accompanied by other lifestyle-related factors that vary from individual to individual which may further influence study outcomes.

Variability present in study design is also a possible contributing factor to inconsistent findings across literature. For instance, several studies rely on food-frequency questionnaires or self-reported dietary intake which may not necessarily reflect long-term vitamin D status accurately. As a result, comparisons between studies remain limited due to differences in methods of assessing vitamin D exposure.

It is still unclear if the association between skin cancer risk and vitamin D-rich foods levels is due to vitamin D itself or other confounding factors, such as protective properties that those foods contain themselves. Although there have been suggestions of the correlation between diet and the risk of skin cancer, there are relatively few studies available on this topic. Additional longitudinal and controlled investigations are recommended to clarify whether dietary vitamin D has an independent role in modifying skin cancer risk.

The Role of Geographical Location in Vitamin D Production

In addition to dietary intake, environmental factors such as geographic location play a significant role in both vitamin D production and skin cancer risk. A recent study analyzed the impact of latitude and season on serum vitamin D levels through previously collected data from another study. Vitamin D production results were collected from 2,000 children in the U.S. from different geographic areas with various Fitzpatrick skin types. Results suggested that during the summer, children in northern latitudes produced different amounts of vitamin D compared to children in southern latitudes (9). Sufficient vitamin D production was only evident from children situated in southern latitudes. Nevertheless, summer UV exposure was still effective at generating

vitamin D in northern latitudes. On the other hand, both groups did not produce an adequate amount of vitamin D during the winter (9).

Geographic Variation in UV Exposure and Skin Cancer Risk

Some studies suggest that the gradient of annual UVB and UVA radiation may also be associated with skin cancer risk (14). In one study, incidence rates of NMSC and CMM in Scandinavia, England, New Zealand, and Australia were analyzed with the connection between solar radiation and annual exposure (23). Fair-skinned individuals who live at low latitudes have shown to be correlated with higher incidence of skin cancer (14). However, data lacks detail in terms of external factors that may relate to the increased risk of skin cancer. Possible confounding variables that have been suggested to influence skin cancer risk from previous studies such as skin type and previous cancer associations, should be taken into consideration.

The relationship between geographical locations and skin cancer was observed in an 18-year study based in the United States (24). The erythemal UV index is an estimation method of how UVR reaches the earth's surface. It has been implicated that geographic location and UVR are possible risk-modifying factors for CMM, NMSC, and breast and colon cancers (24). Factors such as ozone depletion as well as seasonal and weather variations affect the amount of UVR that reaches the surface (24). Time of day, cloud cover, haze, ozone concentrations, latitude, and altitude have been speculated to be associated with skin cancer risks (24). Geographical locations may also vary in vitamin D production rates which has been shown to possibly have a beneficial effect for some cancers (24). The results of the study suggested that a north-south UV index gradient and increasing age was associated with the risk of SCC. BCC, although less pronounced, presented similar trends (24). However, in contrast to the risks, it was observed that CMM risk showed little association with the same UV index gradient.

An analysis of geographic and environmental factors highlighted their association with CMM incidence across Canada (25). The study suggested that increases in annual average temperature and summer UVR were associated with higher expected incidence of CMM cases (25). The higher average number of annual rain events together with higher number of annual heat were associated with decrease in CMM incidence rate. However, it is speculated that the association may be explained by the impact of the factors on individual behavior, leading to higher or lower rates of sun exposure (25).

Interaction Between Diet, Geography, and UV Exposure

Inconsistent findings have been observed across various studies that examine how dietary intake and geographic location influences vitamin D levels and skin cancer risk, which likely reflects differences in both methodological design and environmental exposure. In high UV environments, geographic location plays a direct role on variability in sun exposure which remains the main contributor to vitamin D intake. Conversely, dietary sources may play a more significant role in maintaining adequate vitamin D levels in regions with limited sunlight. Additionally, behavioral patterns related to sun exposure can be influenced via geographic variation. For instance, vitamin D production and UV exposure is likely to increase for individuals living in warmer climates who may spend more time outdoors. In contrast, populations that live in colder climates may face seasonal reductions in UVR and thus rely more heavily on dietary vitamin D sources. Together, these factors complicate direct comparisons across studies and may partly explain the inconsistent findings observed.

CONCLUSION

As the central environmental driver, UV exposure is able to simultaneously increase vitamin D synthesis while inducing DNA damage that elevates skin cancer risk. However, this relationship is modified by geography as it influences UV intensity and duration, thus potentially shaping availability of vitamin D across populations. Furthermore, while dietary intake can act as a secondary source of vitamin D, note that associations between vitamin D-rich foods and skin cancer risk may not be attributable to vitamin D itself. The biological properties of vitamin D-rich foods such as fatty fish and dairy products also contain other bioactive compounds that independently influence cancer risk. Additionally, geography may drive dietary patterns, creating a cyclical interaction between diet, geography, vitamin D status, and skin cancer (Figure 1).

There have been mixed results shown in previous observational studies of vitamin D status and skin cancer. Throughout the study, there has been noticeable trends in previous research that suggests a correlation between vitamin D and skin cancer protection. Furthermore,

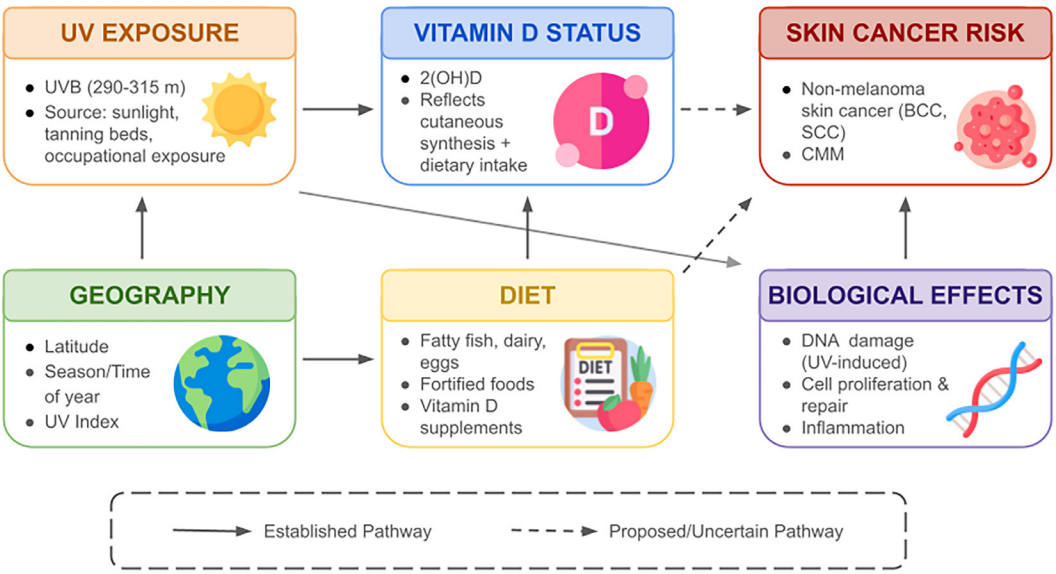

Figure 1. ***Conceptual Framework Of The Relationship Between UV Exposure, Vitamin D, Diet, and Skin Cancer Risk.*** *The relationship between UV exposure, vitamin D production, dietary intake, and skin cancer risk. UV exposure contains a dual effect, simultaneously promoting vitamin D synthesis and induces DNA damage. Diet provides an independent source of vitamin D, while geographic factors influence both UV exposure and dietary patterns, contributing to variability in observed associations across studies. Figures created using icons from Flaticon.*

studies suggest that there may be many confounding factors that should be taken into consideration to be further analyzed in future investigations. In an attempt to eliminate possible external factors that affect the correlation between vitamin D levels and skin cancer risk, observations have also been made on the link between dietary intake and risks, as well as geographical factors. External factors connected to both increased or decreased risks of skin cancer, as well as varying vitamin D levels may also substantially indicate an association between the two.

Evidence of the association between dietary factors and disease risk remains inconsistent, due to few studies that have taken place in which sunlight-derived vitamin D exists as a confounding factor in collected data. Factors to consider include possible differences in long-term and short-term dietary intake, eating habits, family history of skin cancer, population base, and consumption of organic and non-organic foods. Other indicators of vitamin D status, such as the blood levels of 25-hydroxyvitamin D, may cause inconsistency across studies due to the multiple sources from which vitamin D can be obtained (22). An alternative method of measuring vitamin D levels, such as devices specifically used to measure sun-induced vitamin D, may be able to provide consistent results for clinical usage. Further observation of populations with consumption of foods considerably rich in vitamin D, such as pescetarian diets that consist of fish or dairy, may be compared with populations with a balanced diet that do not emphasize vitamin D intake. Populations may include groups that eliminate obtaining sun-induced vitamin D during the process, through sun protection procedures, reducing the possible contributions of inconsistencies in results. A longitudinal study may follow up on individuals over the course of multiple years. Results may possibly indicate whether or not groups who obtain vitamin D from pescetarian diets have a notable difference in skin cancer risk when compared to groups with a balanced diet. In future investigations, eliminating any possible external factors that could possibly change the results of the association between skin cancer and dietary intake may provide a substantially clearer and consistent trend.

The results from various studies suggested that geographic location may be linked to skin cancer risk. However, current findings still lack detail in whether or not sun-induced vitamin D production varies in different areas, especially locations observed to be at a higher risk for cancer due to geographical factors. There are noticeable trends in higher vitamin D production posing a protective effect against skin cancer throughout several studies. Future research on the geographical effect of

vitamin D production and the comparison with areas associated with higher risks of cancer will be needed.

Nevertheless, it is safer and preferable for individuals to obtain adequate levels of vitamin D through diet than through sun exposure (3). Higher levels of vitamin D can be achieved while minimizing the risks associated with UV exposure (11, 27). Recommended sun protection procedures should be followed for personal safety against UVR (28-30).

The relationship between UV exposure, vitamin D production, and skin cancer risk is best understood as a multifactorial interaction. UV exposure simultaneously promotes vitamin D synthesis and carcinogenesis, while diet and supplementation provide alternative sources that may modify risk independently. Geographic factors further influence both vitamin D status and cancer susceptibility.

Further investigation focusing on eliminating external factors that affect the correlation between vitamin D levels and skin cancer risk may be able to provide stronger and solidified evidence. Current evidence suggests that an adequate amount of vitamin D should be obtained from a healthy diet instead, as unprotected sun exposure should be avoided in order to reduce underlying risks. In contrast to studies that examine the factors of UV exposure, vitamin D status, diet, and geographic variation in isolation, this review highlights the importance of considering these factors as interconnected influences on skin cancer risk.

ACKNOWLEDGEMENTS

The author would like to express deepest gratitude to research mentor Naa-Oye Bosompra for her invaluable support, guidance, feedback, and inspiration throughout this work. The author also extends sincere thanks to friends and family for their continuous encouragement and motivation during the research and writing process.

CONFLICT OF INTEREST

The author declares that there are no conflicts of interest related to this work.

REFERENCES

1. Li H, Colantonio S, Dawson A, Lin X & Beecker J. Sunscreen Application, Safety, and Sun Protection: The Evidence. *Journal of Cutaneous Medicine and Surgery.* 2109; 23 (4): 357-369. https://doi.org/10.1177/1203475419856611
2. Laikova KV, Oberemok VV, Krasnodubets AM, Gal'chinsky NV, *et al*. Advances in the Understanding of Skin Cancer: Ultraviolet Radiation, Mutations, and Antisense Oligonucleotides as Anticancer Drugs. *Molecules (Basel, Switzerland)*. 2019; 24 (8): 1516. https://doi.org/10.3390/molecules24081516
3. Martin-Gorgojo A, Gilaberte Y & Nagore E. Vitamin d and skin cancer: An epidemiological, patient-centered update and review. *Nutrients*. 2021; 13 (12): 4292. https://doi.org/10.3390/nu13124292
4. Watson M, Holman DM & Maguire-Eisen M. Ultraviolet radiation exposure and its impact on skin cancer risk. *Seminars in Oncology Nursing.* 2016; 32 (3): 241–254. https://doi.org/10.1016/j.soncn.2016.05.005
5. Surdu S, Fitzgerald EF, Bloom MS, Boscoe FP, *et al*. Occupational exposure to ultraviolet radiation and risk of non-melanoma skin cancer in a multinational European study. *PLoS ONE*. 2013; 8 (4): e62359. https://doi.org/10.1371/journal.pone.0062359
6. Mahamat-Saleh Y, Aune D & Schlesinger S. 25-Hydroxyvitamin D status, vitamin D intake, and skin cancer risk: A systematic review and dose–response meta-analysis of prospective studies. *Scientific Reports*. 2020; 10: 13151. https://doi.org/10.1038/s41598-020-70078-y
7. Government of Canada, S. C. Shedding light on Canada's vitamin D levels as we approach the shortest day of the year. Statistics Canada, 2023. https://www.statcan.gc.ca/o1/en/plus/5095-shedding-light-canadas-vitamin-d-levels-we-approach-shortest-day-year
8. Coutinho RCS, Santos AFD, Costa JGD & Vanderlei AD. Sun exposure, skin lesions and vitamin D production: evaluation in a population of fishermen. *Anais brasileiros de dermatologia*. 2019; 94 (3): 279–286. https://doi.org/10.1590/abd1806-4841.20197201
9. Raymond-Lezman JR & Riskin SI. Benefits and risks of sun exposure to maintain adequate vitamin d levels. *Cureus*. 2023; 15 (5): e38578. https://doi.org/10.7759/cureus.38578
10. Caini S, Gnagnarella P, Stanganelli I, Bellerba F, *et al*. Vitamin D and the Risk of Non-Melanoma Skin Cancer: A Systematic Literature Review and Meta-Analysis on Behalf of the Italian Melanoma Intergroup. *Cancers*. 2021; 13 (19): 4815. https://doi.org/10.3390/cancers13194815
11. C. van der Pols J, Russell A, Bauer U, E. Neale R, G. Kimlin M, & C. Green A. Vitamin D Status and Skin Cancer Risk Independent of Time Outdoors: 11-Year Prospective Study in an Australian Community. 2012; 113 (3): 637–641. 10.1038/jid.2012.346
12. Manson JE, Cook NR, Lee I-M, Christen W, *et al*.

Vitamin d supplements and prevention of cancer and cardiovascular disease. *New England Journal of Medicine.* 2019; 380 (1): 33–44. https://doi.org/10.1056/NEJMoa1809944

13. De Smedt J, Van Kelst S, Janssen L, Marasigan V, *et al*. High-dose vitamin D supplementation does not improve outcome in a cutaneous melanoma population: Results of a randomized double-blind placebo-controlled study (ViDMe trial). *British Journal of Dermatology.* 2024; 191 (6): 886–896. https://doi.org/10.1093/bjd/ljae257
14. Ombra MN, Paliogiannis P, Doneddu V, Sini MC, *et al*. Vitamin D status and risk for malignant cutaneous melanoma: Recent advances. *European Journal of Cancer Prevention*. 2017; 26 (6): 532–541. https://doi.org/10.1097/CEJ.0000000000000334
15. National Institutes of Health. Office of dietary supplements—Vitamin D. Office of Dietary Supplements, n.d. https://ods.od.nih.gov/factsheets/VitaminD-HealthProfessional/
16. Malagoli C, Malavolti M, Farnetani F, Longo C, *et al*. Food and beverage consumption and melanoma risk: A population-based case-control study in northern italy. *Nutrients.* 2019; 11 (9): 2206. https://doi.org/10.3390/nu11092206
17. Malagoli C, Malavolti M, Agnoli C, Crespi CM, *et al*. Diet quality and risk of melanoma in an italian population. *The Journal of Nutrition*. 2015; 145 (8): 1800–1807. https://doi.org/10.3945/jn.114.209320
18. Paiva M, Yumeen S, Kahn BJ, Nan H, *et al*. Coffee, Citrus, and Alcohol: A Review of What We Drink and How it May Affect our Risk for Skin Cancer. *The Yale journal of biology and medicine.* 2023; 96 (2): 205–210. https://doi.org/10.59249/EVKO3455
19. Vinceti M, Malagoli C, Iacuzio L, Crespi CM, *et al*. Serum fatty acids and risk of cutaneous melanoma: A population-based case-control study. *Dermatology Research and Practice.* 2013; 2013 (1): 659394. https://doi.org/10.1155/2013/659394
20. Ibiebele TI, van der Pols JC, Hughes MC, Marks GC, *et al*. Dietary pattern in association with squamous cell carcinoma of the skin: A prospective study. *The American Journal of Clinical Nutrition.* 2007; 85 (5): 1401–1408. https://doi.org/10.1093/ajcn/85.5.1401
21. Gamba CS, Stefanick ML, Shikany JM, Larson J, *et al*. Low fat diet and skin cancer risk: The women's health initiative randomized controlled dietary modification trial. *Cancer Epidemiology, Biomarkers & Prevention: A Publication of the American Association for Cancer Research, Cosponsored by the American Society of Preventive Oncology*. 2013; 22(9): 1509-1519. https://doi.org/10.1158/1055-9965.EPI-13-0341
22. Vinceti M, Malagoli C, Fiorentini C, Longo C, *et al*. Inverse association between dietary vitamin D and risk of cutaneous melanoma in a northern Italy population. *Nutrition and Cancer.* 2011; 63 (4): 506–513. https://doi.org/10.1080/01635581.2011.539314
23. Moan J, Grigalavicius M, Baturaite Z, Juzeniene A & Dahlback A. North-South gradients of melanomas and non-melanomas. *Dermato-Endocrinology.* 2013; 5 (1): 186–191. https://doi.org/10.4161/derm.23791
24. Qureshi AA, Laden F, Colditz GA & Hunter DJ. Geographic variation and risk of skin cancer in us women: Differences between melanoma, squamous cell carcinoma, and basal cell carcinoma. *Archives of Internal Medicine.* 2008; 168 (5): 501–507. https://doi.org/10.1001/archinte.168.5.501
25. Berman-Rosa M, Logan J, Ghazawi FM, Le M, *et al*. Analysis of geographic and environmental factors and their association with cutaneous melanoma incidence in canada. *Dermatology (Basel, Switzerland).* 2022; 238 (6): 1006–1017. https://doi.org/10.1159/000524949
26. Zambrano-Román M, Padilla-Gutiérrez JR, Valle Y, Muñoz-Valle JF & Valdés-Alvarado E. Non-Melanoma Skin Cancer: A Genetic Update and Future Perspectives. *Cancers*. 2022; 14 (10): 2371. https://doi.org/10.3390/cancers14102371
27. Lewandowska A, Lewandowski T, Rudzki G, Próchnicki M, *et al*. The risk of melanoma due to exposure to sun and solarium use in poland: A large-scale, hospital based case - control study. *Asian Pacific Journal of Cancer Prevention: APJCP.* 2023; 24 (7): 2259–2269. https://doi.org/10.31557/APJCP.2023.24.7.2259
28. Symanzik C & John SM. Sun protection and occupation: Current developments and perspectives for prevention of occupational skin cancer. *Frontiers in public health.* 2022; 10: 1110158. https://doi.org/10.3389/fpubh.2022.1110158
29. Koh HK. Preventive strategies and research for ultraviolet-associated cancer. *Environmental health perspectives.* 1995; 103 (8): 255–257. https://doi.org/10.1289/ehp.95103s8255
30. Sánchez G, Nova J, Rodriguez-Hernandez AE, Medina RD, *et al*. Sun protection for preventing basal cell and squamous cell skin cancers. The Cochrane database of systematic reviews. 2016; 7 (7): CD011161. https://doi.org/10.1002/14651858.CD011161.pub2