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REVIEW ARTICLE

Potential skin carcinogenicity in cosmetics products

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ABSTRACT

Exposure to natural and artificial ultraviolet (UV) radiation is a primary risk factor for skin cancers which are one of the most common and growing diagnosed forms of cancer in the world. UV radiation causes carcinogenesis by damaging DNA, inducing oxidative stress, and causing genetic changes that disrupt normal cellular regulation. The basic categories of skin cancer are basal cell carcinoma (BCC), squamous cell carcinoma (SCC), melanoma, and Merkel cell carcinoma (MCC), each with distinct biological features, clinical behaviors and rates of metastases. Basal cell carcinoma is the most prevalent, and typically less aggressive type, while SCC is more invasive. Melanoma constitutes a significant cause of skin cancer mortality due to its high metastatic potential, while MCC is a rare neuroendocrine malignant disease associated with Merkel cell polyomavirus infection and immunosuppression. The incidence of melanoma has risen significantly worldwide, especially in areas with high UV radiation, such as Australia, North America and Europe. Other risk factors include fair skin, genetic susceptibility, chronic inflammation, immunosuppression, chemical carcinogens, and indoor tanning. There have also been concerns about various potentially harmful ingredients in cosmetics and personal care products, such as parabens, phthalates, preservatives containing formaldehyde, coal tar dyes, heavy metals, and oxybenzone. Some compounds can have endocrine disrupting, genotoxic, and/or carcinogenic effects, which can all lead to oxidative stress, DNA damage, and tumor formation. The biomedical literature was searched in the PubMed, ScienceDirect, and Google Scholar databases over a period of 10 years from 2016 to 2026, using key words such as skin cancer, melanoma, squamous cell carcinoma, and basal cell carcinoma. The prevention strategies are protecting from UV, using broad-spectrum sunscreens, wearing protective clothing, regular skin examinations, and choosing safer cosmetics. Epidemiology, molecular mechanisms and environmental determinants will be key to improving skin cancer prevention, early detection, public awareness and therapeutic development.

Keywords: Skin cancer, UV exposure, carcinogenesis, melanoma, oxidative stress

Abbreviations
BCC — Basal Cell Carcinoma

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BHA — Butylated Hydroxyanisole
BHT — Butylated Hydroxytoluene
DEA — Diethanolamine
DNA — Deoxyribonucleic Acid
MCC — Merkel Cell Carcinoma
MCPyV — Merkel Cell Polyomavirus
MEA — Monoethanolamine
PEGs — Polyethylene Glycols
SCC — Squamous Cell Carcinoma
TEA — Triethanolamine
US FDA — United States Food and Drug Administration
UV — Ultraviolet
Vitamin A — Retinyl Palmitate

INTRODUCTION

Among the most common cancers globally, skin cancer has become one of the leading cancers diagnosed in the United States and can be classified into melanoma and nonmelanoma skin cancer. Its incidence rates have been rising significantly and thus created a great public health burden. This type of cancer usually involves uncontrollable growth of abnormally proliferating cells and is associated with an accumulation of UV radiation damage, which leads to immunosuppression, oxidative stress, genotoxicity, and DNA mutations.[1] Apart from environmental and genetic determinants, a possible connection between skin cancer and the use of cosmetics has received significant attention. While cosmetics serve the purpose of personal hygiene, skincare, and cosmetic improvement, some components may cause skin damage due to their phototoxic, photo-sensitizing, hormonal disruptive, and skin-barrier disrupting effects.[2] Parabens, heavy metals, preservatives, artificial fragrances, and colorants have been linked to the potential long-term harm.[3] However, numerous cosmetics include sunscreens, antioxidants, moisturizers, and UV filters that can prevent UV-induced damage to the skin.

The stratum corneum, stratum granulosum, stratum spinosum and stratum basale, as well as basal melanocytes, are epidermal structures, whereas the skin capillaries, collagen fibers, sebaceous glands, arrector pili muscles, hair follicles, and nerve endings are dermal structures.[8,9,10] Squamous cell carcinoma is formed from spinous keratinocytes, and exhibits keratin pearls and intercellular bridges,[11,12] whereas basal cell carcinoma has palisading basaloid cells. Pleomorphic malignant melanocytes are asymmetric, have irregular borders and invade the skin more deeply (ABCDE rules).[13] A comparative table is provided between staging (T-stage or Breslow depth) and prognostic factors (invasion depth and lymph nodes) (**Figure -1**).

Public awareness of the safety of cosmetics, risks associated with UV light, and proper skincare practices is crucial.[4] The preventive methods involve regular application of sunscreen, wearing protective clothing, avoiding indoor tanning, minimizing peak hour sun exposure, and conducting routine skin checks for early diagnosis.[5] Regulatory controls are important for evaluation of cosmetic ingredients, banning hazardous ingredients, and encouraging evidence-based labeling of cosmetics. Further research is required to understand the pathways through which cosmetic ingredients can cause skin cancer.[6]

In this review, the connection between skin cancer and the use of cosmetics will be explored in relation to epidemiology, etiology, risk factors, potential hazards from the use of cosmetics, preventive measures, and public health consequences.[7] This review will also focus on the issues that require further research regarding the safety of cosmetics and skin cancer prevention. It is necessary to understand the connection between these two fields of study.

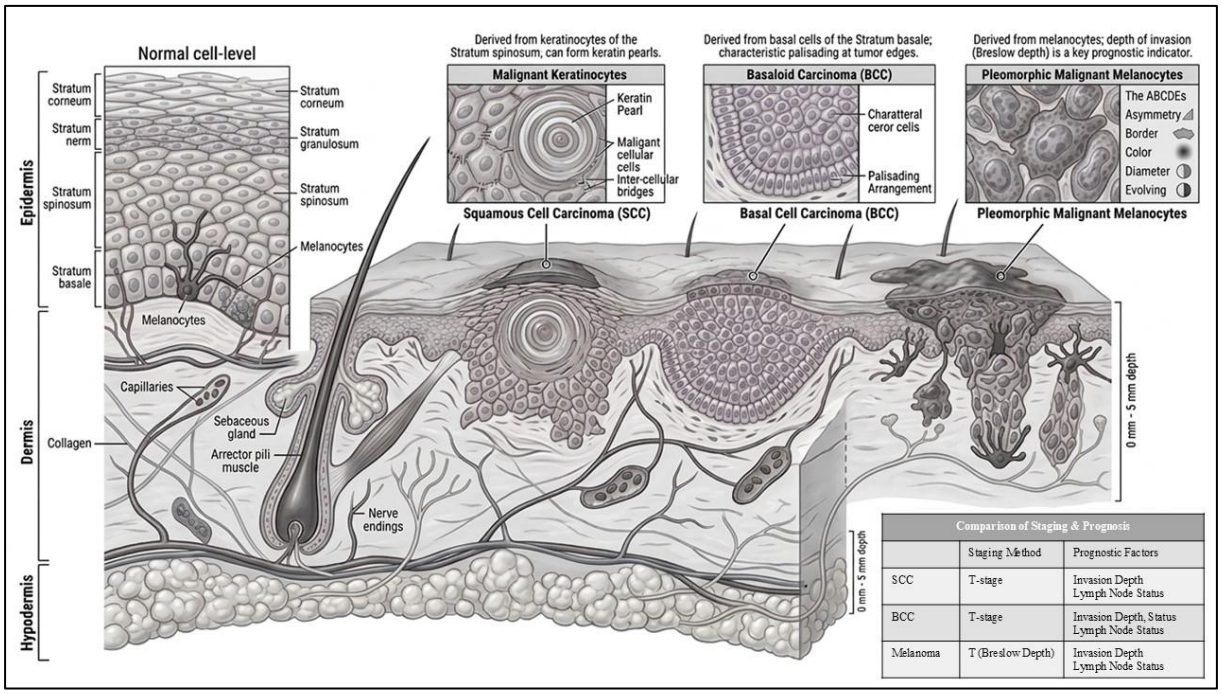


Figure 1. This schematic depicts a cross section of human skin from the epidermis to the hypodermis, and compares the normal histology to three major types of cutaneous malignancies.

METHODS

The electronic databases of PubMed, Science Direct, and Google Scholar were comprehensively and systematically searched for publications in the English language from 2016 to 2026 on the effects of clinical studies, relevant mechanistic reports, and review articles, which were all considered. Studies unrelated to skin cancers were excluded. Data from the selected studies were critically analyzed to provide an overview of cosmetics and skin cancer. Initially, 121 articles met the inclusion criteria, but ultimately, most of them were excluded due to duplication, access issues, and irrelevant topics, leaving 75 articles for analysis and synthesis**.**

Fundamentals and major types of skin cancer

Skin cancer is one of the most prevalent forms of cancer worldwide and is characterized by the uncontrolled growth of abnormal skin cells.[14] The disease primarily develops due to prolonged exposure to ultraviolet (UV) radiation from sunlight or artificial sources such as tanning beds and lamps. UV radiation damages cellular DNA, leading to mutations that promote carcinogenesis and abnormal cell proliferation. Understanding the fundamentals of skin cancer is essential for improving prevention, early diagnosis, and treatment strategies.[15] The global incidence of skin cancer has increased considerably over recent decades, making it a major public health concern and emphasizing the importance of protective measures against excessive UV exposure. Skin cancer is broadly classified into three major types: basal cell carcinoma (BCC), squamous cell carcinoma (SCC), and melanoma.[16] Basal cell carcinoma is the most common and least aggressive form, usually developing in sun-exposed areas of the skin. Squamous cell carcinoma is more invasive and may spread if untreated. Melanoma, although less common, is the most dangerous type because of its high metastatic potential and associated mortality. Each type differs in clinical presentation, severity, progression, and risk factors, highlighting the complexity of diagnosis and management in dermatological oncology.[17]

Basal cell carcinoma (BCC)

Basal cell carcinoma (BCC) is the most common type of skin cancer, accounting for nearly 75% of all skin cancer cases worldwide.[18] It originates from the basal cells located in the deepest layer of the epidermis and usually develops in areas frequently exposed to sunlight, including the face, scalp, neck, ears, shoulders, and hands. Basal cell carcinoma commonly appears as a small shiny or pearly bump, a pinkish patch,[19] or a scaly lesion that may gradually

enlarge over time. Basal cell carcinoma is displayed in panel 1, with a pearly, telangiectatic nodule and four stages of progression from the epidermis to metastasis **(Figure 2)**. In some cases, the lesion may ulcerate, bleed easily, develop a crust or scab, and fail to heal properly. Although BCC rarely metastasizes to distant organs, it can become locally invasive and cause substantial destruction of surrounding tissues, including muscles, cartilage, and bones, if left untreated. The primary etiological factor associated with BCC is prolonged exposure to ultraviolet (UV) radiation from sunlight or artificial sources such as tanning beds.[20] UV radiation induces DNA mutations and oxidative stress in skin cells, contributing to abnormal cellular proliferation and carcinogenesis. Individuals with fair skin, light-colored eyes, blonde or red hair, a history of frequent sunburns, excessive outdoor exposure,[21] or weakened immune systems are at greater risk of developing BCC. Epidemiological studies indicate that the disease is more common among older adults, particularly those above 50 years of age, and occurs slightly more frequently in males than females.[22] Additionally, patients with a previous history of BCC are more likely to develop recurrent lesions or other forms of nonmelanoma skin cancer in the future. Clinically, BCC presents in several forms, including nodular, superficial, pigmented, and morpheaform subtypes, each exhibiting different morphological characteristics and growth patterns.[23] Common symptoms include persistent lumps, bumps, itchy or painful lesions, translucent nodules, or ulcerative sores that bleed after minor trauma.[24] Early diagnosis is essential to prevent extensive tissue damage and improve treatment outcomes. Diagnosis generally involves clinical examination followed by dermoscopic evaluation and histopathological confirmation through skin biopsy.[25] Various treatment approaches are available depending on the size, subtype, and anatomical location of the tumor. Standard treatment methods include surgical excision, electrodessication and curettage, cryotherapy, laser therapy, photodynamic therapy, topical chemotherapy, and radiation therapy.[26] In advanced or inoperable cases, targeted therapies such as vismodegib and sonidegib, which inhibit the Hedgehog signaling pathway, have demonstrated therapeutic effectiveness.[27] Preventive strategies play a critical role in reducing the incidence of BCC and include regular use of broad-spectrum sunscreen, wearing protective clothing, avoiding excessive sun exposure during peak UV hours, and performing routine skin examinations for early detection of suspicious lesions.[28]

Squamous cell carcinoma (SCC)

Squamous cell carcinoma (SCC), also known as cutaneous squamous cell carcinoma (CSCC), is the second most common form of skin cancer, accounting for approximately 16% of all skin cancer cases worldwide.[29] Squamous cell carcinoma arises from the squamous cells located in the epidermis, the outermost protective layer of the skin. It commonly develops in areas frequently exposed to ultraviolet (UV) radiation, including the face, scalp, ears, neck, arms, and hands. Panel 2 illustrates squamous cell carcinoma (SCC) arising from stratum spinosum keratinocytes containing keratotic plug and provides an overview of T1-T3 and N/M staging based on tumor size, depth, perineural invasion and nodal spread **(Figure 2)**. However, SCC may also occur in less sun-exposed regions and mucosal tissues such as the mouth, lips, lungs, and anal region, reflecting its potentially invasive nature.[30] Clinically, SCC often appears as a rough, scaly, rose-colored, or pinkish patch or bump that may crust, bleed, or ulcerate over time.[31] In advanced cases, the lesion may develop into a persistent open sore that does not heal, indicating deeper tissue invasion and progressive malignancy. The primary etiological factor associated with SCC is chronic exposure to UV radiation from sunlight or artificial tanning devices. [32] UV-induced DNA damage causes genetic mutations and abnormal proliferation of squamous cells, ultimately contributing to carcinogenesis. Additional risk factors include fair skin, light-colored eyes, blonde or red hair, older age, immunosuppression, chronic skin inflammation, previous skin injuries, and prolonged occupational sun exposure.[33] Epidemiological evidence indicates that SCC predominantly affects individuals over the age of 50 years and demonstrates increasing incidence globally due to aging populations, lifestyle changes, and excessive UV exposure.[34] Compared with basal cell carcinoma, Squamous cell carcinoma exhibits a more aggressive biological behavior and possesses a greater potential to metastasize through the lymphatic system or bloodstream if not diagnosed and treated promptly.[35] Squamous cell carcinoma is classified into several subtypes based on histopathological characteristics and disease

progression. Cutaneous SCC remains confined initially to the epidermis but may invade surrounding tissues if untreated.[36] In situ SCC, also known as Bowen's disease, represents the earliest stage of the malignancy and is restricted to the superficial epidermal layer. [37] Metastatic SCC is the most severe form and involves the spread of cancer cells to distant organs or lymph nodes, significantly increasing morbidity and mortality. Common clinical manifestations include rough scaly plaques, raised nodules with a central depression, ulcerative lesions, recurrent sores, and actinic keratosis, a precancerous condition characterized by dry, scaly, and irritated skin patches that may progress to SCC over time.[38] Early diagnosis is essential for effective disease management and prevention of metastasis. Diagnostic procedures generally include physical examination, dermoscopy, and histopathological confirmation through biopsy.[39] Treatment strategies depend on the tumor size, location, and stage of progression. Common therapeutic approaches include surgical excision, electrodessication and curettage, cryotherapy, topical chemotherapy, photodynamic therapy, laser therapy, and radiation therapy.[40] In advanced or unresectable cases, targeted therapies and systemic treatments may be considered. Preventive measures remain critically important and include regular use of sunscreen, protective clothing, minimizing sun exposure during peak UV hours, and routine skin monitoring for suspicious lesions or persistent sores.[41]

Melanoma

Melanoma, also known as malignant melanoma, is the most aggressive and life-threatening form of skin cancer despite being less common than basal cell carcinoma and squamous cell carcinoma.[42] It originates from melanocytes, the pigment-producing cells responsible for skin coloration. Melanoma develops when these cells undergo uncontrolled proliferation due to genetic mutations and DNA damage, primarily caused by excessive exposure to ultraviolet (UV) radiation from sunlight or tanning devices.[43] Unlike other forms of skin cancer, melanoma can occur on any part of the body, including areas not regularly exposed to the sun, such as the soles of the feet, under fingernails or toenails, the genital region, and even within the eyes.[44] Clinically, melanoma often presents as a darkly pigmented lesion or an irregular mole with asymmetrical borders and uneven coloration. It may arise from a pre-existing mole or appear as a completely new lesion on previously normal skin.[45] Common warning signs include changes in the size, shape, texture, or color of a mole, as well as the development of itching, bleeding, or ulceration. Panel 3 shows the stages of melanoma (0-IV) with a focus on Breslow thickness and spread to lymph nodes and distant organs **(Figure 2)**. The American Academy of Dermatology recommends the "ABCDE" criteria for early recognition of melanoma: asymmetry, irregular borders, color variation, diameter greater than 6 mm, and evolving appearance.[46] Another important indicator is the "ugly duckling sign," where a mole appears distinctly different from surrounding moles. Several factors increase melanoma risk, including fair skin, light-colored eyes, red or blonde hair, frequent sunburns during childhood, excessive sun exposure, family history of melanoma, immunosuppression, and the presence of multiple or atypical moles.[47] Epidemiological studies indicate that melanoma accounts for the majority of skin cancer-related deaths because of its strong metastatic potential. Men commonly develop melanoma on the upper trunk and back, whereas women more frequently develop lesions on the legs.[48] Melanoma is classified into different stages according to tumor thickness and the extent of spread. Stage 0, also known as melanoma in situ, remains confined to the epidermis, while Stage I and II represent localized disease with increasing tumor thickness.[49] Stage III involves spread to nearby lymph nodes or surrounding skin, and Stage IV indicates distant metastasis to organs such as the lungs, liver, brain, or bones. [50] Early diagnosis is critical because prognosis and survival rates decline significantly once metastasis occurs. Diagnostic evaluation typically includes dermoscopic examination and histopathological confirmation through biopsy. [51] Additional investigations such as sentinel lymph node biopsy, computed tomography (CT), magnetic resonance imaging (MRI), and positron emission tomography (PET) scans are used to assess metastatic spread in advanced disease. [52] Surgical excision remains the primary treatment for localized melanoma, whereas advanced cases may require immunotherapy, targeted therapy, radiation therapy, or chemotherapy. Immune checkpoint inhibitors such as pembrolizumab and nivolumab have significantly improved survival outcomes in metastatic melanoma.[53] Targeted therapies including vemurafenib, dabrafenib, and trametinib are particularly effective in patients

with BRAF gene mutations. Preventive strategies such as regular skin examinations, avoidance of excessive UV exposure, sunscreen use, and early dermatological consultation remain essential for reducing melanoma incidence and mortality.(54)

Merkel cell carcinoma (MCC)

Merkel cell carcinoma (MCC) is a rare but very aggressive form of neuroendocrine carcinoma affecting the skin arising from the Merkel cells which are specialized epidermal cells that act as the receptors for touch sensation. Even though it is less common compared to melanoma, basal cell carcinoma, and squamous cell carcinoma, it poses high chances of recurrence, progression and metastasizing early. The most common presentation is usually a rapidly growing, painless, hard, shiny, reddish or purplish nodule. (55) Panel 4 shows Merkel cell carcinoma, which develops adjacent to Merkel cells, which are mechanoreceptors, and has a high rate of progression **(Figure 2)**.

Risk factors for MCC development are chronic exposure to UV light, advanced age, immunosuppression and Merkel Cell Polyomavirus (MCPyV) infection. And 80% of MCC cases are known to be caused by MCPyV infection with the other 20% developing as a result of UV light induced DNA damage.(56) Advanced age, especially in men and fair skinned patients and those with organ transplants and/or HIV infection among others are at higher risk.

MCC staging is based on the tumor burden, nodal status, and the presence of metastasis. Localized disease primarily equates to stages I-II, whereas stage III is associated with nodal disease, and stage IV reflects distant metastasis. Diagnosis is established via skin biopsy with histopathologic and immunohistochemical assessment, while CT, MRI, PET, and sentinel lymph node biopsy contribute to staging.(57)

Therapy is dictated by the disease stage, with surgical resection being common, along with postoperative radiation therapy. Treatment for advanced or metastatic MCC may include systemic chemotherapy or immunotherapy. Immune checkpoint inhibitors like avelumab and pembrolizumab have proven useful. The prevention strategy is aimed at reducing UV exposure and skin screening.(58)

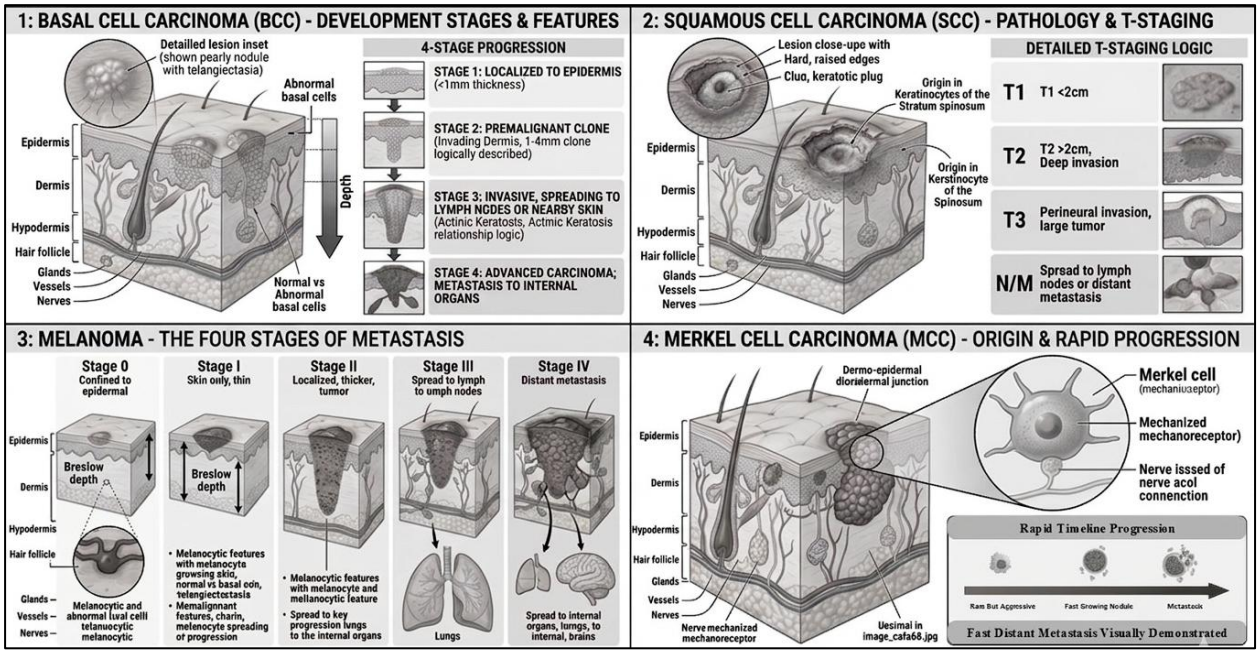


Figure 2. This four-panel schematic illustrates the pathogenesis and staging of major cutaneous malignancies in cross-sections of epidermis, dermis and hypodermis

Epidemiology of skin cancer

Skin cancer is a complex disorder arising from the interaction of many factors, including genetic, environmental, demographic, and behavioral ones. Intrinsic as well as extrinsic factors, including genetic disposition, skin type, UV radiation, and lifestyle factors, play a role in the pathogenesis of skin cancer.(59) Australia demonstrates the highest incidence and ASR of skin cancer among the listed countries, particularly among males, reflecting the strong influence of ultraviolet radiation exposure and

fair-skinned populations. The United States, Germany, and the United Kingdom also report high incidence rates. In contrast, Asian countries such as China exhibit substantially lower ASR values, likely due to greater melanin-mediated photoprotection and differing environmental and genetic factors. Across most regions, males show higher incidence rates than females, emphasizing the influence of occupational exposure, behavioral factors, and cumulative UV radiation exposure **(Table 1)**.

Overexposure to UV radiation, whether natural or artificial, is the key modifiable risk factor. UV radiation induces DNA damage, oxidative stress, immunosuppression, mutations, and malignancy. People with fair skin, light-colored eyes, blond and red hair color, and low concentration of melanin have increased risk for skin cancer. Sunburns, especially in childhood, working in the open air, playing in the sun, insufficient protection from the sun's rays are some other risk factors. Genetic predisposition, particularly in people with DNA repair disorders like xeroderma pigmentosum, is another risk factor. Family history of melanoma, atypical and multiple moles, immunosuppression, chronic inflammatory skin diseases, and skin cancer are other risk factors.

Cancer of the skin is the most prevalent type of cancer across the world, and it affects many people in Australia, New Zealand, North America, and some regions of Europe. Basal cell carcinoma is responsible for 75 to 80 percent of non-melanoma skin cancers, whereas squamous cell carcinoma contributes to about 20 to 25 percent of the cases.[60] Melanoma is the less prevalent form of skin cancer, but it causes the majority of deaths in patients suffering from skin cancer, owing to its metastatic nature. High incidence is seen in males and elderly people, whereas low incidence is seen in Asians and Africans.[61] Prevention through sunscreen application, wearing protective clothes, limiting exposure during peak UV times, not using indoor tanning beds, and proper screening of the skin can prevent skin cancer.[62]

Pathogenesis and etiology of skin cancer 294 words

Skin cancer arises as a result of a complex multi-step phenomenon characterized by genetic mutations, hyperproliferation, exposure to the environment, and faulty DNA repair. The principal environmental factor responsible for the development of both melanoma and non-melanoma skin cancers is ultraviolet (UV) radiation. This form of energy causes DNA damage, oxidative stress, inflammation, and immunosuppression, thus contributing to carcinogenesis. The UVB (290-320 nm) component of UV radiation produces direct DNA damage by forming cyclobutane pyrimidine dimers, while the UVA (320-400 nm) component triggers production of reactive oxygen species, resulting in indirect DNA damage.[63] Non-melanoma skin cancers, primarily basal cell carcinoma (BCC) and squamous cell carcinoma (SCC), often develop because of chronic UV damage to the epidermal keratinocytes. The SCC skin cancer type is known for TP53 (tumor protein p53) mutations, whereas BCC is often linked to the PTCH1 (patched 1) mutation and the Hedgehog signaling pathway activity.

Table 1. Global Distribution of Skin Cancer Incidence 2022 and Age-Standardized Rates (ASR).

Rank	Country	Total cases	ASR/100,000	Male cases	Male ASR/100,000	Female cases	Female ASR/100,000
—	World	331,722	3.2	179,953	3.7	151,769	—
1	United States	101,388	16.5	60,762	19.3	40,626	14.4
2	Germany	21,976	16.5	11,667	13.4	10,309	10.9
3	United Kingdom	19,712	15.3	10,170	15.1	9,542	15.8
4	Australia	16,819	37.0	10,902	45.9	5,917	29.1
5	France (Metropolitan)	15,729	13.5	7,880	14.5	7,849	12.6
6	Italy	13,769	12.7	7,325	13.3	6,444	12.2
7	Russia	12,903	5.3	5,223	5.2	7,680	5.5
8	Canada	11,383	14.5	6,434	15.9	4,949	13.4
9	Brazil	9,676	3.3	4,958	3.6	4,718	3.1
10	China	8,789	0.37	4,402	0.37	4,387	0.36

In addition, melanomas, originating from the melanocytes, contain mutations of BRAF (B-Raf proto-oncogene), CDKN2A (cyclin-dependent kinase inhibitor 2A), and MC1R (melanocortin 1 receptor) genes. Unlike BCC and SCC, melanomas are more closely related to intensive UV exposure and sunburns especially in childhood and adolescent years.[64]

Other risks include genetics, family history, light complexion, fair hair color and eye color, multiple and unusual moles, immunosuppression, chronic inflammation, old age, artificial tanning, arsenic and other carcinogens, and oncogenic infections including human papilloma virus (HPV). Fitzpatrick types I and II skin are more prone to UV-induced damage owing to reduced pigmentation, though any skin type can develop skin cancers. In general, skin cancer development is a result of DNA damage, oxidative stress, chronic inflammation, immune system malfunctioning, and cellular repair failure, leading to abnormal cell growth and invasion, formation of new blood vessels and metastasis in later stages.[65]

Multifactorial mechanisms of skin cancer progression

Skin cancer arises because of the interplay between exposure to the environment, immune system dysfunctions, and genetics. Exposure to ultraviolet (UV) radiation, especially UV-A and UV-B rays, is the main risk factor in the environment. This causes DNA damage, oxidative stress, and mutations and leads to cell transformation into malignant forms. Therefore, frequently exposed sites like the face, neck, scalp, and arms become commonly affected. [66] Ionizing radiation, specifically gamma rays, might act as another risk factor via causing oxidative and genetic damage.

Immune dysfunction further acts to increase the risk by limiting the immune system's monitoring function, leading to the survival of the cancerous cells. Immune dysfunction includes conditions such as AIDS, diabetes, chronic cancers, aging, and immunosuppression. Malignant cells evade immunity by limiting antigen presentation, T-cell activation, and cytokine signaling. These mechanisms have formed the basis for development of immunotherapy methods like immune checkpoint blockers, peptide vaccines, and adoptive T-cell therapy.[67]

Genetic mutations play a critical role as well. Genetic mutations in DNA repair lead to increased susceptibility to DNA damage caused by UV light, as exemplified in cases of xeroderma pigmentosum. Mutations in genes associated with proliferation and signal transduction such as BRAF and MEK mutations also have a significant role in the pathogenesis of melanoma. Changes in chromosomes 1, 3, 6, and 8 have been linked to the development of melanoma. Skin cancer is caused by a combination of radiation damage, immune dysfunction, genetic mutations, and DNA repair abnormalities.[11]

Cosmetics and skin cancer risk

The increased use of cosmetic products has created apprehension that these products may have a link with causing skin damage and cancer. While cosmetics, such as moisturizers, sunscreens, makeup, hair dyes, fragrance and skin care products offer beauty and protection, their continued use may result in oxidative stress, DNA damage, endocrine disruption and carcinogenesis.[68]

There are various cosmetic products whose safety has come into question. Parabens act as weak estrogens, although the link between parabens and hormone-related cancers has not been conclusively established. Formaldehyde and formaldehyde-releasing preservatives serve as antimicrobial preservatives, while formaldehyde is a human carcinogen. Coal tar dyes may contain carcinogenic polycyclic aromatic hydrocarbons whereas heavy metals such as lead, arsenic, cadmium and mercury can increase oxidative stress and DNA damage. Phthalates also have the potential for disrupting the endocrine system. [13] There have also been some studies on the endocrine disrupting properties of some sunscreens like oxybenzone, benzophenone and octinoxate.[69] These ingredients in external exposure sources like shampoos, nail polish, lotions, hair dyes and sunscreen include parabens, BHA/BHT, formaldehyde, toluene, phthalates, heavy metals and oxybenzone.[70-72] In the cell, carcinogens can cause DNA adducts and chromosomal aberrations, which result in irreversible mutations. Endocrine-disrupting compounds bind to membrane receptors and simulate the action of hormones, and mitochondrial dysfunction increases levels of reactive oxygen species (ROS) resulting in oxidative stress.[73] Uncontrolled proliferation is allowed due to impaired apoptosis. The right-

hand side of Figure 3 illustrates the process of cellular progression from normal to malignant through the phases of initiation (mutation), promotion (clonally expanded) and progression (malignant).[74]

Ultraviolet (UV) rays continue to be the biggest environmental risk factor associated with skin cancers, including malignant melanomas. UV rays lead to DNA damage, mutations, and oxidative stress that contribute to development of cancerous changes in the body. Hence, preventative methods such as use of protective clothing, hats, sunglasses, and application of sunscreen cream with SPF equal to or greater than 30 should be considered. Antioxidant enriched moisturizing creams could also help in reducing the oxidative stress experienced by the skin.[75]

Chemical-induced toxicity associated with cosmetic products

The global cosmetics and personal care industry has seen significant growth due to the rising demands of people for beauty, skin protective, and hygienic products. The global cosmetics and skincare market produced close to 138.9 billion US dollars in revenue in 2022.[72] Nevertheless, there is a growing body of evidence indicating that some of the cosmetic ingredients may be toxicologically and genotoxic agents via dermal absorption, inhalation, or accidental ingestion, thereby playing an adverse role in endocrine disturbance, organ toxicity, reproductive diseases, genetic instability, and carcinogenesis.[73]

Some of the important cosmetic contaminants are heavy metals such as Pb, As, Cd, Cr, and Hg, which could be accumulated in body tissues, promote oxidative stress, and cause DNA damages.[39–41] Genotoxic exposure results in mutations, chromosomal aberrations, DNA repair impairment, and altered cell proliferation processes; hence, a higher risk of cancer development.[42,76] Another list of hazardous substances includes formaldehyde and formaldehyde-releasers, coal tar dyes, 1,4-dioxane, acrylates, oxybenzone, parabens, phthalates, hydroquinone, methylisothiazolinone, methylchloroisothiazolinone, BHA, and BHT. All these substances may be involved in skin sensitization, endocrine and reproductive or developmental toxicity, oxidative stress, DNA damage, and potential carcinogenicity.[74]

The production and disposal of cosmetics can be responsible for environmental pollution with heavy metals and various toxicants.[75] As a result, some regulatory bodies like the US FDA and the European Union have limited or banned some harmful ingredients used in cosmetics.[54,55] However, regulation is not always consistent around the world and thus some of these products may get to the market without undergoing any toxicological tests.

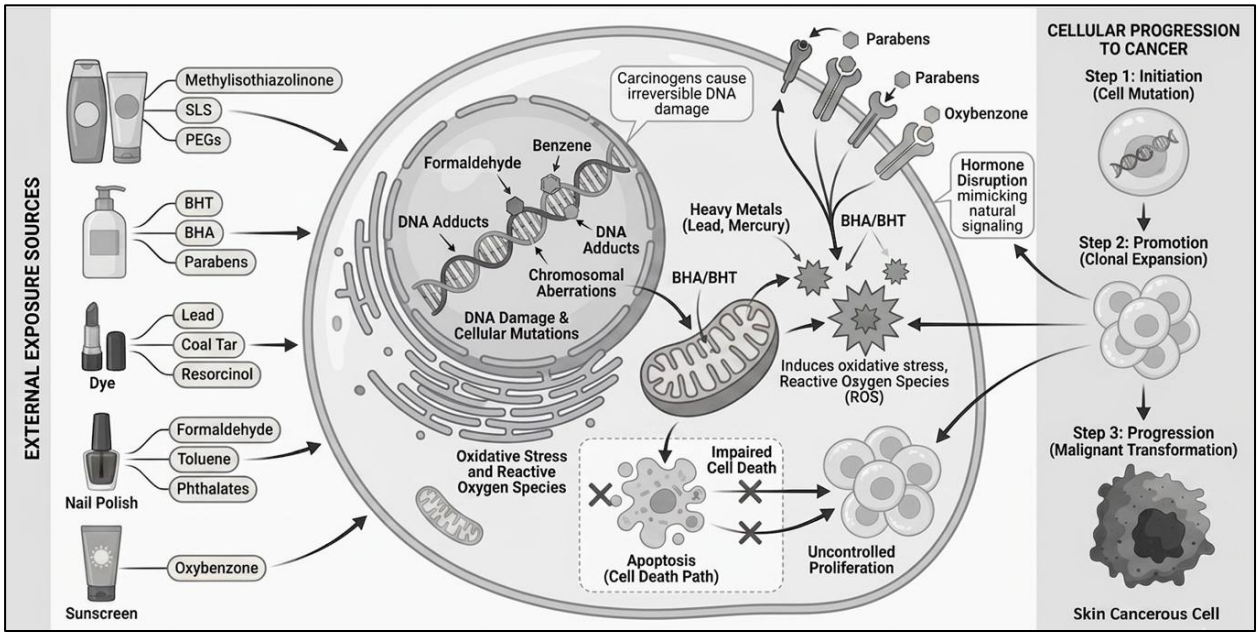


Figure 3. This diagram shows proposed mechanisms for how chemicals in personal care products can cause skin cancer

Table 2. This table catalogs 30 chemicals commonly found in cosmetics and personal care products, organized by functional category, typical product type, and associated health hazards.

Category	Chemical Name	Common Products	Health Effects/Hazards
Preservatives	Methylisothiazolinone [84]	Shampoos, lotions, conditioners	Skin irritation, contact dermatitis, allergies
Preservatives	Methylchloroisothiazolinone [13]	Haircare products, soaps	Skin irritation, contact dermatitis, allergies
Preservatives	Butylated Hydroxyanisole (BHA) [72]	Lip products, moisturizers	Skin irritation, endocrine disruption, carcinogenicity
Preservatives	Butylated Hydroxytoluene (BHT) [87]	Skin creams, lipsticks	Skin irritation, endocrine disruption, carcinogenicity
Preservatives	Formaldehyde-releasing preservatives [95]	Hair products, nail polish	Skin irritation, carcinogenicity concerns
Preservatives	Imidazolidinyl Urea [89]	Various cosmetics	Skin irritation, formaldehyde release
Preservatives	Diazolidinyl Urea [90]	Skin creams, lotions	Skin irritation, formaldehyde release
Preservatives	Parabens (e.g., Methylparaben) [12, 79]	Various cosmetics	Hormone disruption, breast cancer concerns, NHL
Preservatives	Propylparaben [79]	Lotions, deodorants	Skin irritation, hormone disruption, allergies
Sunscreen Chemicals	Oxybenzone [53]	Sunscreens	Hormone disruption, potential allergies
Plasticizers	Phthalates (e.g., Dibutyl Phthalate) [80]	Nail polish, fragrances	Hormone disruption, reproductive toxicity
Plasticizers	Diethyl Phthalate [80]	Fragrances	Hormone disruption, allergies
Heavy Metals	Lead (in certain color additives) [94]	Lipsticks, hair dyes	Neurological damage, reproductive issues
Heavy Metals	Mercury (in some skin-lightening products) [94]	Skin creams	Neurological damage, kidney and lung toxicity
Coal Tar Dyes	Coal tar [85]	Hair dyes	Carcinogenicity, skin irritation
Fragrances	Artificial fragrance chemicals [86]	Various cosmetics	Allergies, skin irritation, respiratory issues
Fragrances	Fragrance [87]	Cosmetics, perfumes	Allergies, skin irritation, respiratory issues
Antimicrobial Agents	Triclosan [88]	Antibacterial soaps, toothpaste	Hormone disruption, antibiotic resistance
Powders	Talc [85]	Powders, eyeshadows, blushes	Lung problems, ovarian cancer risk
Oils and Emollients	Mineral oils [57]	Lotions, creams	Skin irritation, clogged pores, long-term skin issues
Surfactants	Sodium Lauryl Sulfate (SLS) [89]	Shampoos, body washes	Skin and eye irritation, allergies
Amines	Ethanolamines (MEA, DEA, TEA) [90]	Shampoos, body washes	Skin irritation, allergies, carcinogenicity (DEA)
Solvents	Toluene [91]	Nail polish, hair dyes	Nervous system damage, developmental issues
Skin-Lightening Agents	Hydroquinone [69]	Skin lightening products	Skin irritation, ochronosis, carcinogenicity concerns
Hair Dye Chemicals	Resorcinol [92]	Hair dyes, acne treatments	Skin irritation, allergies, thyroid dysfunction
Polymers	Polyethylene Glycols (PEGs) [93]	Lotions, creams, cleansers	Skin irritation, allergies, potential contaminants
Preservatives	Formaldehyde [95]	Nail products, hair straighteners	Skin and respiratory irritation, carcinogenicity

Vitamin Derivatives	Retinyl Palmitate (Vitamin A) [12]	Anti-aging creams	Skin irritation, photosensitivity, potential carcinogenicity
Hair Treatment Chemicals	Ammonia [94]	Hair dyes, hair color products	Eye and lung irritation, potential allergies
Environmental Contaminants	Microplastics [96]	Exfoliating scrubs, toothpaste	Environmental harm, potential ingestion and absorption
Nanomaterials	Nanoparticles [56]	Various cosmetics	Potential skin penetration, long-term health effects

Therefore, increased awareness, strict regulation, ingredient labeling, and more toxicological research are important in minimizing the dangers of harmful cosmetic chemicals.[76] Categories include preservatives (isothiazolinones, parabens, BHA/BHT, formaldehyde releasers), sunscreen agents, plasticizers (phthalates), heavy metals (lead, mercury), coal tar dyes, fragrances, surfactants, solvents, and emerging contaminants such as microplastics and nanoparticles **(Figure 3).** Reported hazards range from dermatitis, allergic sensitization, and irritation to endocrine disruption, reproductive toxicity, neurotoxicity, and suspected carcinogenicity. Several entries, notably parabens, BHA, and talc, are linked to hormonal or cancer-related concerns, though evidence strength varies (**Table 2**).

CONCLUSION

Skin cancer remains one of the most significant global public health challenges due to its continuously rising incidence, high morbidity, and substantial mortality associated particularly with aggressive forms such as melanoma and Merkel cell carcinoma. The disease develops through a multifactorial process involving prolonged ultraviolet (UV) radiation exposure, genetic susceptibility, immune dysregulation, environmental carcinogens, and abnormal cellular proliferation. Among the major types of skin cancer, basal cell carcinoma (BCC) is the most prevalent and least aggressive, squamous cell carcinoma (SCC) demonstrates greater invasive potential, while melanoma represents the most dangerous form because of its strong metastatic capability and high mortality rate. Merkel cell carcinoma (MCC), although rare, is also highly aggressive and associated with poor prognosis. In addition to environmental and genetic risk factors, growing scientific evidence suggests that prolonged exposure to certain cosmetic and personal care products may contribute to skin carcinogenesis and systemic toxicity. Several chemical ingredients commonly found in cosmetics, including parabens, phthalates, formaldehyde-releasing preservatives, heavy metals, coal tar dyes, oxybenzone, hydroquinone, acrylates, and other synthetic preservatives, possess toxic, endocrine-disrupting, mutagenic, or carcinogenic properties. These compounds may induce oxidative stress, DNA damage, hormonal imbalance, organ toxicity, reproductive dysfunction, and genetic instability, thereby increasing susceptibility to chronic diseases, including cancer. The widespread and prolonged use of cosmetics, combined with inadequate regulatory oversight in some regions, further amplifies concerns regarding cumulative exposure to hazardous substances and their long-term health consequences.

Preventive measures such as regular application of broad-spectrum sunscreens, use of protective clothing, minimizing UV exposure during peak sunlight hours, avoidance of tanning devices, routine dermatological examinations, and early detection of suspicious lesions remain essential for reducing morbidity and mortality associated with skin cancer. Furthermore, advances in molecular biology, immunotherapy, targeted therapy, and dermatological oncology have substantially improved the diagnosis and treatment of skin cancers. Immune checkpoint inhibitors, targeted therapies against BRAF and MEK mutations, and novel immunotherapeutic approaches have significantly enhanced survival outcomes, particularly in advanced melanoma and MCC. Nevertheless, continuous research is required to better understand the molecular mechanisms linking environmental toxins, cosmetic ingredients, UV radiation, immune modulation, and carcinogenesis. Overall, increasing public awareness, strengthening cosmetic safety regulations, promoting safer product formulations, encouraging responsible sun-protective behaviors, and advancing scientific research are critical for minimizing skin cancer burden and improving long-term human health outcomes. A multidisciplinary approach

integrating dermatology, toxicology, oncology, molecular biology, environmental science, and public health is essential to address the growing challenges associated with skin cancer and cosmetic-related toxicities worldwide.

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