A review of Basal and Squamous Cell Carcinoma in Relation to Climate Change

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Abstract:

Skin cancer remains a significant public health challenge, with diagnosis rates differing significantly worldwide. This review explores the link between climate change and its impact on Squamous Cell Carcinoma (SCC) and Basal Cell Carcinoma (BCC). Both types of skin cancer are primarily caused by ultraviolet (UV) radiation. BCC is mainly associated with UVB-induced mutations in the Sonic Hedgehog signaling pathway, which generally controls cell growth and tissue regeneration. When mutated, the hedgehog signaling pathway can lead to unchecked cell proliferation and cancer development. On the other hand, SCC is primarily driven by mutations in the MAPK signaling pathway, which oversees cell survival, proliferation, and apoptosis. Mutations in this pathway result in the constant activation of RAF, MEK, and ERK proteins, leading to uncontrolled cell division. Diagnosing BCC and SCC typically involves physical exams, biopsies, and imaging tests to determine the stage of cancer. Treatment options include Mohs surgery, excision, cryotherapy, and photodynamic therapy. Climate change exacerbates the risk and incidence of skin cancer by increasing UV radiation exposure, making it a growing concern if not addressed promptly. Skin cancer rates also vary globally due to differences in geographic location, healthcare access, and cultural practices. Sunscreen remains a vital preventive measure worldwide, though in the U.S., regulatory barriers classify sunscreen as a drug, slowing the approval of new, more effective ingredients compared to countries where sunscreen is considered a cosmetic. Despite significant progress in reducing skin cancer incidence, more efforts are needed to fully address the ongoing challenges of diagnosis and mortality.

Summary:

Climate change and sun exposure are inexplicably intertwined and, unfortunately, lead to notable public health problems. Due to climate change, there is a predicted increase in skin cancer, and some countries are taking preventative health measures.

Introduction

Though the risk and prevalence can vary across geographic regions, one in five Americans develop some form of skin cancer in their lifetime in the US (*Skin Cancer*, n.d.). Every day in the U.S., approximately 9,500 people are diagnosed with skin cancer (skin cancer, n.d.-b). More than 1 million Americans are currently living with melanoma, the most severe form of skin cancer (Skin Cancer, n.d.-c). Risk factors influencing skin cancers include U.V. range, behaviors, genetics, and geography. Geographical location has a significant impact on Australia. Australia has the highest skin cancer rate, with 2 out of 3 Australians developing skin cancer by the time they are 70 years old. These high rates are due to high ultraviolet (UV) rays from the sun and the predominant skin tone. It is also high due to Australia's location in the southern hemisphere in relation to the elliptical orbit (Cancer Council NSW, 2024). The two most common types of skin cancer are Squamous cell carcinoma (SCC) and Basal cell carcinoma (BCC). Due to climate change leading to the changes in U.V. penetration, there is an increased risk of skin cancer. Climate change leads to changes in temperature and an increase in the U.V. due to prolonged periods of sun. Due to climate changes, SCC and BCC rates will increase as rising temperatures and other environmental factors lead to higher exposure levels of U.V., which

causes mutations in DNA and leads to skin cancer (Bharath & Turner, 2009). In this paper, the author examines the relationship between climate change and skin cancer and how we can better protect ourselves. This review will focus on the two most common types of skin cancer, SCC and BCC, rather than melanoma.

A. Mechanisms of Skin Cancer Pathogenesis

The main cause of skin cancers is exposure to UV-A, UV-B, and UV-C radiation. U.V. falls between visible light and X-rays. U.V. radiation falls between visible light and X-rays on the electromagnetic spectrum(Education, n.d.). U.V. light spans from a range of 10-400 nanometers. Of UV rays, UVA has the longest wavelength (320-400 nm), and its radiation is responsible for 90-99% of radiation on Earth(Narayanan et al., 2010b). The stratospheric ozone layer does not filter this kind of radiation, and it has longer wavelengths than UVB and UVC. These longer UV wavelengths have low energy, enabling the radiation to penetrate deeper into the dermis skin layers. The top layer of the skin is the epidermis, while the middle layer is the dermis(Professional, n.d.). In the skin, UVA causes increased levels of reactive oxygen species, which can cause oxidative stress and lead to cell damage. UVB has slightly shorter wavelengths (280-320 nm) radiation, generating 1-10% of the UV radiation reaching Earth. Unlike UVA, UVB is partially filtered by the stratospheric ozone layer. Primarily damaging the epidermis, UVB has shorter wavelengths with higher energy. UVB forms lesions such as cyclobutane pyrimidine dimers and pyrimidine-pyrimidone photoproducts (Narayanan et al., 2010). Lesions are breaks of changes in the nucleotide structure of the helix (Alhmoud et al., 2020). This ultimately affects transcription, which then affects translation. Cyclobutane pyrimidine dimers are lesions with thymine or cytosine in DNA or uracil in RNA covalently linked by a cyclobutane ring, which blocks DNA replication and transcription(PubChem, n.d.). Pyrimidine-pyrimidine photoproducts are a covalent bond that links carbon at position 6 of one pyrimidine ring and carbon at the four positions of the other base's ring. These lesions both interfere with the nucleotide sequences and, hence, interfere with replication and transcription, which causes mutations in cells(Yokoyama & Mizutani, 2014). The shortest wavelength of UV is UVC (100-280 nm), which is entirely absorbed by the stratospheric ozone layer and does not reach the Earth's surface. UVC is entirely absorbed and will not be further discussed as it does not reach the Earth's surface. Overall, each U.V. subtype has a different effect on the body. Although UV-A is the lowest energy level, it still affects the skin's middle layer and can cause skin cancer. UV-B, with shorter wavelengths, affects the epidermis and is a main cause of skin cancer. (Narayanan et al., 2010)

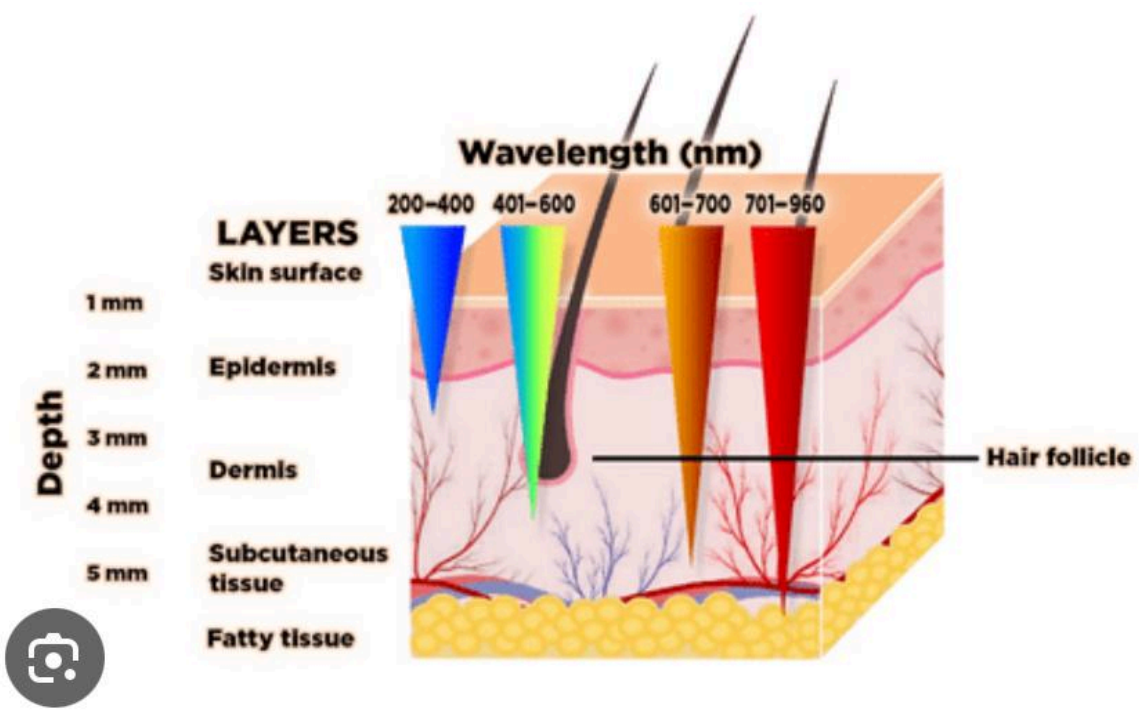

Figure 1

(*Figure 2. Light Skin Penetration With Different Wavelengths (Nm).*, n.d.)

This diagram shows the different layers of the skin and the penetration levels of UV compared to a hair follicle. The skin layers include the skin surface, epidermis, dermis, subcutaneous tissue, and fatty tissue. The wavelengths included in the diagram range from 200 to 960 nm.

UVB exposure causes basal cell carcinoma through damage to DNA and its repair system. This causes the immune system to alter, resulting in more mutations and neoplasms. Neoplasm is classified as an abnormal tissue growth in a part of the body. The start of abnormal cell growth is due to mutation of the Hedgehog pathways. H.H. pathways are involved in the development of this type of cancer. The normal activation of the H.H. pathway promotes fetal development, cell proliferation, and tissue patterning. Activation of the mutated H.H. pathway promotes cell proliferation, inhibits apoptosis, enhances cell metastasis, and promotes blood vessel formation and tumor inflammation. For adults, the H.H. pathway is crucial for tissue maintenance, renewal, and regeneration and is active in the stem cells of the central nervous system, skin, and intestine. In these areas, it maintains homeostasis and regeneration. In humans, three secreted ligands control the activation of the H.H. pathway: Sonic Hedgehog, SHH; Desert Hedgehog, DHH; and Indian Hedgehog, IHH. There is also the transmembrane receptor Patched 1(PTCH1), the 7-pass transmembrane G protein-coupled receptor (GPCR), the main transducer Smoothened (SMO), and three zinc finger GLI transcription factors (GLI1, GLI2, GLI3). When H.H. ligands are absent, PTCH1 stops SMO by preventing it from entering the Primary cilium. The GLI is locked into the cytoplasm by SUFU and phosphorylated by the protein kinases(PKA, CK1, and GSK3β). GLI undergoes a post-translational modification. This leads to the formation of repressor proteins (GLI3/2) that move into the nucleus and inhibit GL1 target genes. In the presence of H.H. ligands, PTCH1 attaches to a ligand and moves through

P.C. Now SMO can move to the P.C., which promotes detachment of G.L. to SUFU. Then, the GLI ACT can move into the nucleus and transcribe GLI target genes. (Giammona et al., 2023)

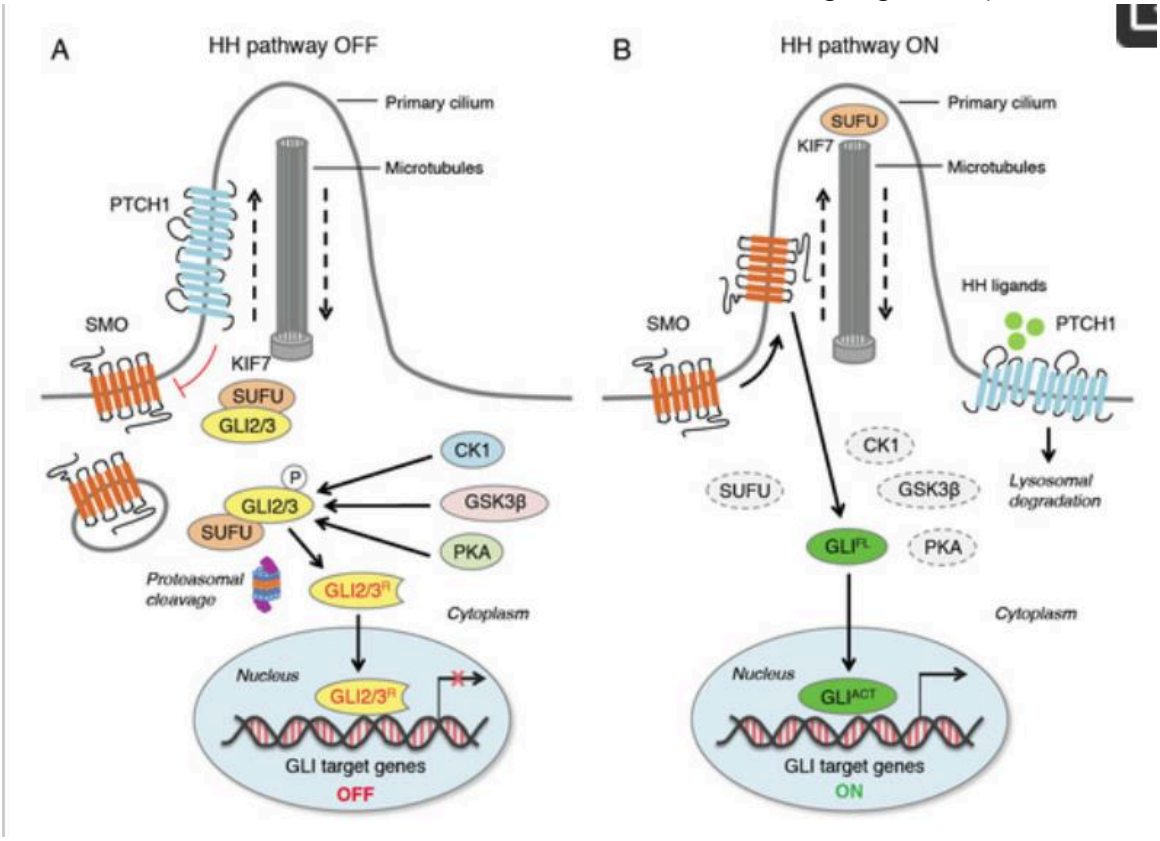

Figure 2 (Giammona et al., 2023)
The Hedgehog pathway inactivated (A) and when activated (B). The HH pathway is activated when the HH ligand attaches to PTCH1, enabling PTCH 1 to move across the cell membrane, and SMO can move up to the primary cilium. This leads to detached GL1 which translocates down to the nucleus and transcribes target genes.

Squamous cell carcinoma, the tumor progression depends on the mitogen-activated protein kinase (MAPK) signaling pathway. MAPKs are crucial to cell life and are involved in gene expression, mitosis, cell survival, and apoptosis. The RAF protein family mediates through the pathway and regulates cell cycle progression. This pathway is split into a three-stage enzymatic cascade: (MAPKKK→MAPKK→MAPK). This is a phosphorylation cascade controlled by kinases. ERK1 and ERK2 pathways play a role in cell survival, while the JNK and p38 MAPK pathways play roles in regulating apoptosis. The RAS/MAPK pathway begins with activating the receptor tyrosine kinase. Once active, the receptor phosphorylates the tyrosine residue in the cytoplasm. The Grb2 abductor protein then recognizes the phosphotyrosine residues and attaches to the phosphorylated protein. SOS is a guanine nucleotide exchange factor that helps during GTP exchange. When connected to RAS, RAS can exchange a GDP with a GTP. Overall, this activates the RAS protein and starts the phosphorylation cascade of downstream proteins RAF, MEK, and ERK. Once activated, RAS is able to move around the membrane to interact with other proteins. RAS then interacts and phosphorylates RAF. RAF is then able to phosphorylate the MEK, and MEK phosphorylates ERK. ERK then moves into the nucleus and

interacts with transcription factors and RNA polymerase to allow gene expression. Transcribed genes that inhibit survival and proliferation are Cc MYC, Bc12, and BCl-XL. One target gene is Cyclin D. Cyclins and CDKs are known to be the drivers of the cell cycle. Through the disruption, the cells have an overactivation of growth, survival, and proliferation (Braicu et al, 2019) (Animated Biology With Arpan, 2022).
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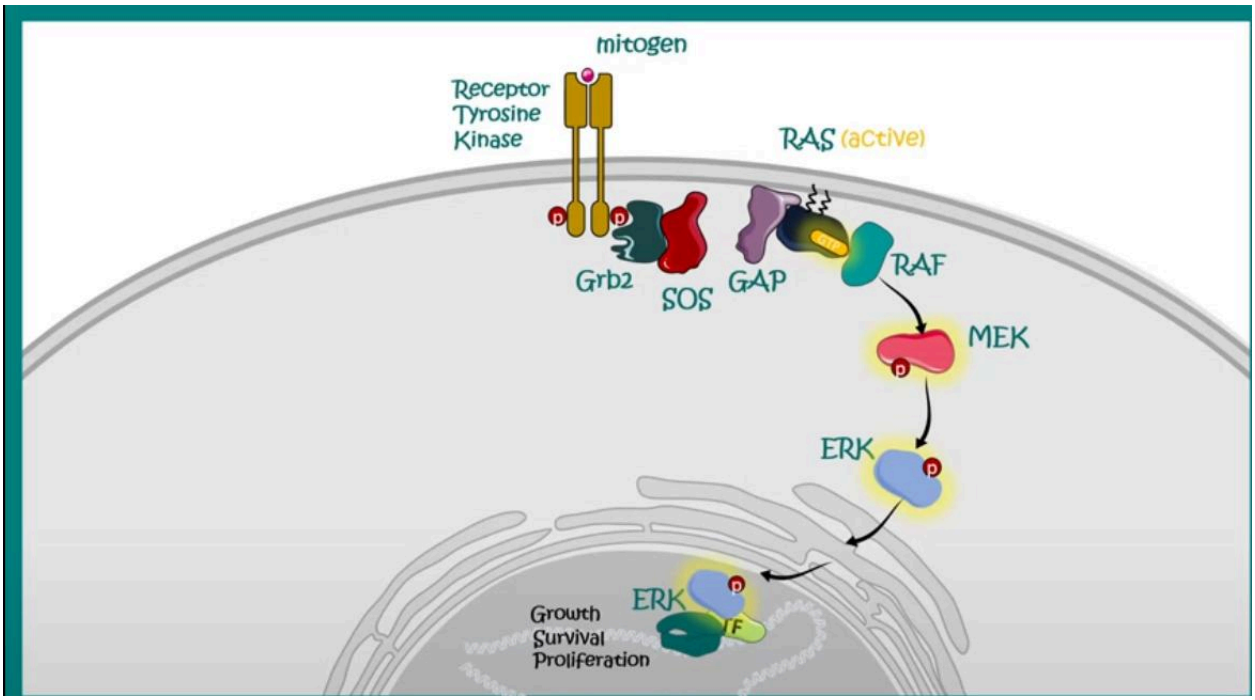

Figure 3 (Animated Biology With Arpan, 2022)
This is an illustration of the MAPK pathway when activated and the subsequent phosphorylation cascade. When the mitogen is attached to the transmembrane receptor protein, then autophosphorylation occurs within the cell. Grb2 then attaches to phosphorylates tyrosine residue on the receptor. Then the Grb2 recruits the SOS which catalyzes the activation of the Ras signaling pathway. A phosphorylation cascade occurs and results in downstream protein activation leading to phosphorylated Erk in the nucleus.

B. Diagnosis and treatment

BCC skin cancer begins with a mutation in basal cells. Basal cells produce new skin cells as old ones die off. Similar to SCC, lighter skin usually develops BCC in sun-exposed areas. Darker skin shades usually get BCC in sun-exposed areas as well. BCC starts as a skin lesion in the form of a sore. The four main types of BCC include (1) nodular, which looks like a pimple surrounded by blood vessels; (2) superficial spreading, which appears as small lighter areas of skin; (3) sclerosing, which forms as scars that expand over time; and (4) pigmentation which makes the area become darker than the skin. After examining the lesion, the doctor will do a physical exam to complete the diagnosis. You are then assigned a stage of cancer: stage 0, where the cancer is only in the epidermis; stage 1, where the cancer is on the epidermis and dermis; stage 2, where cancer is in the epidermis, dermis, and subcutis; stage 3 where cancer

has spread to lymph nodes, and stage 4 where cancer has spread to other organs of your body. Treatment for SCC varies depending on size and shape. Some of the options include cryosurgery, which freezes the cancer cells; photodynamic therapy, which uses blue light and light agents to remove cancer, curettage and electrodesiccation which uses a curette to scratch off the cancerous lump, then burning the area with a needle, excision where cancer is cut out of your skin, Mohs surgery which removes layers of the skin that has cancer, and systemic chemotherapy which uses powerful medicines to kill cancer cells(Professional, n.d.-c). To confirm, the doctor may do a skin biopsy or imaging tests. After being provided with the cancer stage, your doctor will start removing cancer from your body. Like SCC, the options include curettage and electrodesiccation, excision, Mohs surgery, cryotherapy, and photodynamic therapy (Professional, n.d.-a).

SCC skin cancer starts in the skin's squamous cells, middle and outer layers. It occurs on sun-exposed skin: the scalp, the backs of hands, ears, and lips. It can happen in places not exposed to the sun for darker skin. A nodule, or a firm bump on the skin, is a common symptom of SCC. Other symptoms include sores, rough patches inside the mouth, and raised patches on the genitals. The diagnosis process consists of a physical exam. A healthcare professional asks about your history with SCC and looks at symptoms. After the physical exam, the doctor performs a skin biopsy, where a small skin sample is examined under a microscope. Then, imaging tests are taken to identify the size of the cancer and to see if it is malignant. You are then assigned a stage of cancer: stage 0, where the cancer is only in the epidermis; stage 1, where the cancer is on the epidermis and dermis; stage 2, where cancer is in the epidermis, dermis, and subcutis; stage 3 where cancer has spread to lymph nodes, and stage 4 where cancer has spread to other organs of your body. Treatment for SCC varies depending on size and shape. Some of the options include cryosurgery, which freezes the cancer cells; photodynamic therapy, which uses blue light and light agents to remove cancer, curettage and electrodesiccation which uses a curette to scratch off the cancerous lump, then burning the area with a needle, excision where cancer is cut out of your skin, Mohs surgery which removes layers of the skin that has cancer, and systemic chemotherapy which uses powerful medicines to kill cancer cells(Professional, n.d.-c).

C. Climate change

Climate change is a shift in weather patterns over time, including rising temperatures, depletion of the ozone layers, and increased sun exposure. The ozone layer filters all UV-C, some UV-B, and none of UV-A. The ozone layer, O_3, is regulated by levels of O_2, which is a free radical also known as a reactive oxygen species. O_2 is produced by free radicals such as chlorine and bromine. If there is too much O_2, the ozone layer depleted. Chlorofluorocarbons (CFCs) produce free radicals; the more CFCs, the more ozone layer depletion. CFCs are found in aerosol sprays and coolants in refrigerators. The Montreal Protocol was signed to substitute CFC for hydrochlorofluorocarbons (HFCs) to eliminate CFC usage. HCFs cause much less ozone depletion, but they are still greenhouse gasses. Hence, they absorb UV radiation, causing climate change. In the past century, the Earth has warmed up 0.74 degrees Celsius, but over half of the increase occurred since the 1970s. This means there has been an increase in temperature changes recently. This occurs because of an increase in the use of greenhouse gasses. The greenhouse gasses trap radiation from the sun and keep them in the atmosphere. The anthropogenic increases in greenhouse gasses include the burning of fossil fuels and deforestation. Hence, ozone depletion and temperature changes are linked to an increased incidence of skin cancer, but they also affect human behavior (Bharath & Turner, 2009b).

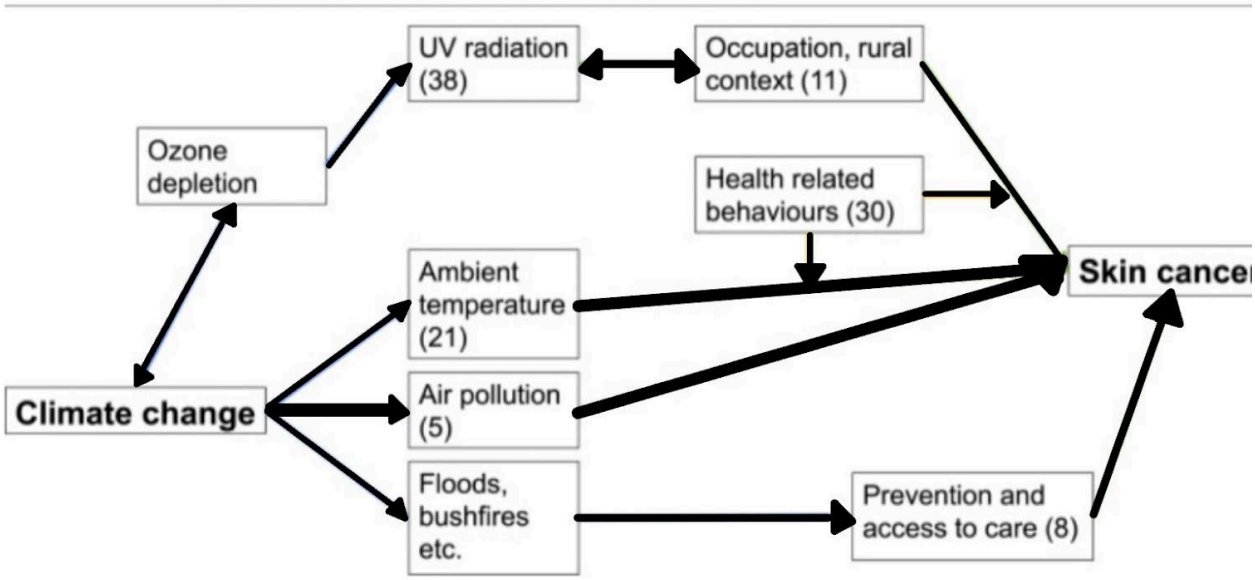

Figure 4 (modified from Bharath & Turner, 2009b)
An illustration of the relationship between climate change and risk factors of skin cancer. The directionality of the arrow indicates the effect each component has on the next. This diagram shows climate change causing ambient temperature, air pollution, floods, bushfires, etc., and ozone depletion. These factors lead to a chain reaction of an increase in UV radiation and prevention of access to care, as well as skin cancer.

D. Risk factors and Statistics

Skin cancer statistics vary across countries due to environmental, behavioral, and healthcare factors. Using disability-adjusted life years (DALYs), scientists can analyze trends in DALYs per 100,000 cases of keratinocyte carcinoma of 7 Global Burden of Disease(GBD) super regions from 1990 to 2017. The GBD study divides the world into seven regions to analyze and compare health data worldwide. Each line shows an increase in DALYs per 100,000 (Fig 4). This indicates that there is a more significant burden of disease in the population because DALYs are a combination of a measure of years of life lost and years lived with disability. Central Europe, Eastern Europe, Central Asia, and High-income regions have had the highest DALYs per 100,000 cases throughout the period. High-income regions include the US, Canada, Western European countries, Australia, and Japan. This means there is a more significant burden of disease in the population. A greater DALY means a more substantial challenge for the healthcare system with higher demand and increased healthcare costs. The sizable global growth indicates behaviors that increase diagnosis. Part of the wide geographic variance in skin cancer rates is due to geography. This affects ozone depletion, urbanization, altitude, and latitude variations. At higher altitudes, such as in Central Asia, they have higher UV exposure due to a thinner atmosphere through which the light passes. Regions closer to the equator are also at risk of higher skin cancer rates. Though Sub-Saharan Africa is the closest GBD region to the equator, they have relatively low skin cancer rates. This can be attributed to the higher melanin levels of the population, and melanin acts as a protective barrier against UVR. This can also be attributed to the limited access to healthcare and lack of awareness. Hence, this leads

to less diagnosis. Though there are fewer diagnoses, the death rates could be higher. In addition to health care and geographic factors, there is also variance due to the behaviors of each region. This includes sun exposure habits, access to protective measures, cultural practices, and healthcare access. In high-income and Latin America and Caribbean regions, with more access to protective measures, people tend to take more vacations and get involved in outdoor activities, hence prolonged sun exposure. In the Sub-Saharan Africa region, though there is less access to use of sunscreens, there is less sunbathing, and their clothes are protective from the sun. In cultural practices in high-income regions, tanning is normalized and a sign of health, but there is greater healthcare access than in other regions (Urban et al., 2021).

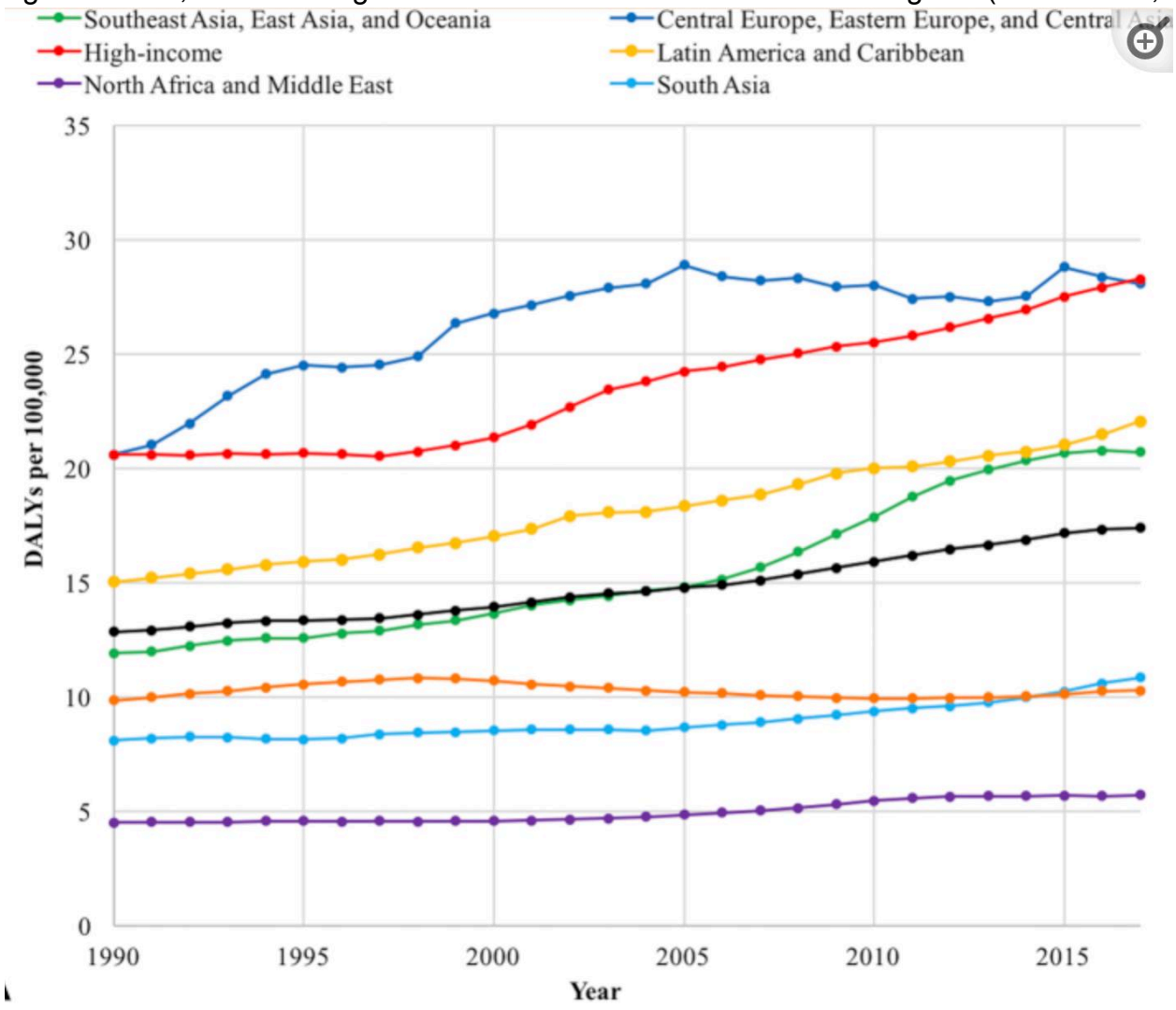

Figure 5 (Urban et al., 2021)
This chart shows the Trends in DALYs per 100,000 cases of keratinocyte carcinoma in 7 GBD super regions from 1990 to 2017. Each GBD region displays upward trends over the 27-year period, with each color representing a different GBD region. Central Europe, Eastern Europe, Central Asia, and High-income regions have had the highest DALYs per 100,000 cases throughout the period. High-income regions include the US, Canada, Western European countries, Australia, and Japan.

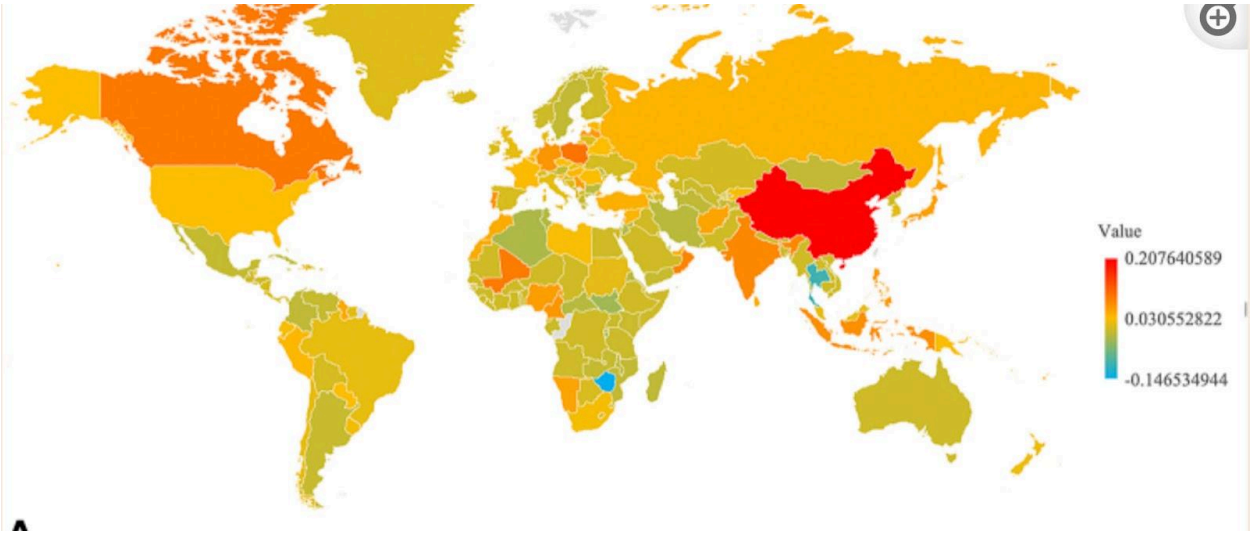

Figure 6 (Urban et al., 2021)

The map shown above shows the percent changes in the age standardized prevalence rate of skin cancer from 1990 to 2017. The red color shows the highest increase in skin cancer levels, while the blue shows the lowest decrease in skin cancer levels. The chart shows that the top 10 countries with the most significant increase were China, Trinidad and Tobago, Poland, Canada, Mali, Oman, Lebanon, India, Indonesia, and Portugal. The top 10 countries with the most significant decrease were Zimbabwe, Thailand, Burundi, South Sudan, Algeria, Jordan, Tunisia, Central African Republic, Iran, and Brunei.

The percent change demonstrates an essential public health opportunity for prevention. While U.V. is the most significant risk factor, other factors contribute to these percentage changes. The chart shows that the top 10 countries with the most significant increase were China, Trinidad and Tobago, Poland, Canada, Mali, Oman, Lebanon, India, Indonesia, and Portugal. The top 10 countries with the most significant decrease were Zimbabwe, Thailand, Burundi, South Sudan, Algeria, Jordan, Tunisia, Central African Republic, Iran, and Brunei (Urban et al., 2021). With the highest percent change, China has most people with fair skin along with changing temperatures, which cause increased UVB exposure. The increase in skin cancer rates can also be because of the technological advancements in China, causing more diagnoses in earlier stages. The number of people taking physical examinations has increased, as has the diagnosis rate. This indicates that an increase in the percent change in the age-standardized prevalence rate does not necessarily mean skin cancer rates are worsening (Bai et al., 2021). It's important to note that in Australia, skin cancer incidence rates have always remained the highest yet not the highest percent change age-standardized prevalence rate. This is due to Australia's latitude gradient, high exposure to UV, predominantly fair-skinned people, and an aging population (Reyes-Marcelino et al., 2023).

E. Common preventative measures

Each country takes preventive measures to prevent skin cancer, the most common being sunscreen, which is used as a spray or lotion. Increased incidence of skin cancer has helped

evolve the formulations and appeal of sunscreen. The affordability and accessibility of healthcare vary between countries, so proactive and preventative measures are critical to preventing adverse health conditions. The use of sunscreen is one such measure. For example, Australia's financial annual burden of SCC and BCC was projected to surpass 700 million dollars. Sunscreen acts as a UV filter that interferes with solar radiation through absorption, reflection, or energy dispersion. Based on their mechanism of action, there are two categories of sunscreens: chemical and mineral-based. Chemical sunscreens act as an absorbent, releasing the UVR as heat energy from the skin. Mineral sunscreens reflect the UV light to protect the skin (Geoffrey et al., 2019).

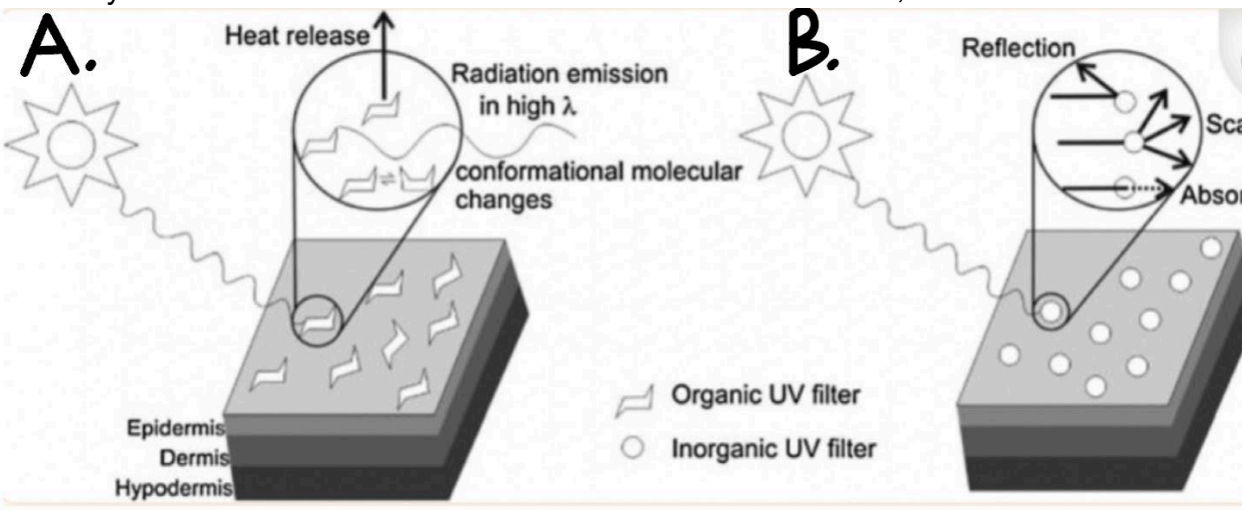

Figure 7
(Geoffrey et al., 2019)
The diagram above shows the different mechanisms of action between organic and inorganic filters. Organic sunscreen is on the left on the skin(A), and inorganic is on the right (B) applied to the skin.Organic sunscreen is the same as chemical sunscreen, and inorganic sunscreen is the same as mineral sunscreen. Organic sunscreen (A) absorbs the UV and releases heat energy. Inorganic (B) reflects the light.

In countries such as Japan, South Korea, and France, sunscreens have newer chemical filters that better protect against UV rays. Due to the Federal Food, Drug, and Cosmetic Act (FD&C Act) passed in 1938, sunscreens in the United States must be tested on animals and are considered drugs instead of cosmetics. In many other regions, like Europe, sunscreens are classified as cosmetics. This classification means that sunscreens and their chemical ingredients are not required to undergo the same rigorous testing as pharmaceuticals, allowing them to be released to the public more quickly—the U.S. The Food and Drug Administration has only approved 16 active ingredients for sunscreens, of which 10 are adequate broad-spectrum U.V. protection. Broad Spectrum U.V. protection refers to a sunscreen that protects from UVA and UVB rays (Aswell, 2019). Countries such as Japan, South Korea, and France have approved 30 or more U.V. filters for sunscreen products (Scaturro, 2024). To protect against UVA light rays, the U.S. has only three approved chemicals, while Europe has seven. The FDA process is slow and expensive, so there is little incentive to create newer and better products if it takes 20 years and tens of millions of dollars. Few products pass the rigorous process. Meanwhile, other countries can get faster approvals because U.V. filters are considered

cosmetics. The U.S. sunscreen lets in three times as many UVA rays as European countries with the same SPF. The FDA approval of Bemotrizonal, an ingredient in nearly all European and Asian sunscreens, would become the safest sunscreen ingredient on the U.S. market (Aswell, 2019). This is due to the broad spectrum protection, and it is highly photostable, meaning it does not degrade in the sun, causing long-lasting protection. It also stays on top of the surface rather than penetrating the skin; this makes it safer for long-term usage. This ingredient is also known for its success with working with other U.V. filters and is versatile with different ingredients. Bemotrizonal is soluble in cosmetics oils to aid in preparing better sunscreens (*Bemotrizinol: A New Sunscreen Active Ingredient Considered by FDA_Chemicalbook*, n.d.). The clinical trial by D'Ruiz and other colleagues titled "Preliminary clinical pharmacokinetic evaluation of bemotrizinol" evaluates the active ingredient Bemotrizonal for inclusion under the FDA's over-the-counter sunscreen standards and regulations. The active ingredient, Bemotrizonal, is responsible for blocking the UVA sun rays. This trial aimed to determine whether 6% Bemotrizinol in a sunscreen oil formulation containing 10% ethanol to enhance skin absorption would result in significant systemic plasma concentrations. If it reaches systemic plasma concentrations, enough of the substance has been in the body's circulation to impact humans. An in vitro Skin Permeation Test (IVTP) uses skin from human donors to assess absorption. The skin samples are placed in diffusion cells, allowing controlled application of substances. Bemotrizinol is applied to these skin samples, and the amount of the substance penetrating a receptor fluid beneath the skin is measured. Following this, a Maximum Usage Trial (MUsT) is conducted. In this trial, healthy participants apply the formula to up to 75% of their body surface multiple times a day over several days. Blood samples are then collected to measure the concentration of the substance in the bloodstream. The concentrations of Bemotrizinol in the plasma did not meet the threshold set by the FDA for systemic exposure. This means the Bemotrizonal does not enter the bloodstream in large amounts when applied to the skin. Overall, the study suggests that Bemotrizonal does not exceed the FDA's threshold for systemic exposure, that there is no accumulation in the bloodstream, and that it is safe for use (D'Ruiz et al., 2023). Bemotrizonal is a broad spectrum SPF that blocks both UVA and UVB filters and is versatile in forms that it can come in.

Other preventative measures include shade umbrellas, UPF clothing, limiting time outdoors, avoiding tanning beds etc, avoiding going outside at peak UV hours, checking UV index, however, these will not be discussed in depth in this review.

Conclusion

This review examined the relationship between climate change and skin cancer and what we can do to better protect ourselves. UVA and UVB play a key role in SCC and BCC development. BCC is caused by UVB but more specifically through mutations in the Hedgehog pathway. This pathway regulates cell growth and tissue regeneration; when it is mutated, there is uncontrolled cell proliferation, leading to cancer. Mutations in the MAPK signaling pathway cause SCC. This pathway regulates cell survival, proliferation, and apoptosis, and mutations lead to constant activation of RAF, MEK, and ERK, causing uncontrolled cell division. BCC and SCC are diagnosed through physical exams, biopsies, and imaging tests to determine the cancer stage; treatment options include Mohs surgery, excision, cryotherapy, and photodynamic therapy.

Climate change increases the diagnosis rate and overall risk of skin cancer. Climate change continues to elevate skin cancer through increased UV radiation if not acted upon anytime soon. Skin cancer rates vary globally due to geographic location, healthcare, and

cultural behaviors. Sunscreen is a crucial preventative measure that is taken all around the world in different ways. In the U.S., sunscreen is considered a drug, making it harder to pass new chemicals, leaving them less advanced than other countries where sunscreen chemicals are classified asa cosmetics rather than drugs. Significant strides have been taken to lower skin cancer incidence rates, but not enough to completely curb skin cancer diagnosis and mortality rates. Skin cancer remains an important public health challenge, and there is still much to be discovered about prevention, diagnosis, and treatment when it comes to these malignancies. Increasing funding research and public awareness is necessary to address these concerns and this significant public health problem. Approval of new sunscreen ingredients in the U.S. is crucial to better-preventing skin cancer. Collaborative research can accelerate discoveries in skin cancer. Protective legislation, whether it is an accelerated approval process, negating the need for animal testing, or banning sunbeds, are options that should and have been considered. The battle against skin cancer is not one we can win alone; with the help of other countries, we can fight this together. Spreading preventative ways from skin cancer and awareness to others is our biggest tool to combat cancer.

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