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Impact of Environmental Carcinogens on Africa: A Quick Review of Health Indices in Africa between 2010 and 2024

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Review Article

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ABSTRACT

Cancer is a leading cause of death globally, with Africa experiencing a significant rise in cases over the past decade. This review explores the impact of environmental carcinogens on Africa's health indices from 2010 to 2024. Environmental carcinogens, comprising chemical, physical, and biological agents, contribute significantly to DNA damage, leading to cancer. These carcinogens can act directly, such as UV radiation, or indirectly, like polycyclic aromatic hydrocarbons, which require bioactivation to become harmful. Carcinogens are classified based on their mode of action and their interplay between internal factors (e.g., inherited mutations) and external factors (e.g., chemical exposure) in cancer development. This review reveals a troubling increase in cancer cases across Africa, tied to widespread exposure to carcinogens. This paper calls for urgent public health measures and policies to reduce carcinogen exposure and mitigate cancer's growing impact in Africa.

Keywords: Africa; cancer; DNA damage; environmental carcinogens.

ABBREVIATIONS

ABC : ATP-binding Cassette
CRC : Colorectal Cancer
DNA : Deoxyribonucleic Acid
EASL : European Association for the Study of the Liver
ENM : Engineered Nanomaterials
HBV : Hepatitis B Virus
HCC : Hepatocellular Carcinoma
HER-2 : Human Epidermal Growth Factor Receptor 2
ICGC : International Cancer Genome Consortium
MPNSTs : Malignant Peripheral Nerve Sheath Tumor
MRI : Magnetic Resonance Imaging
NCDs : Non-Communicable Disease
NGS : Next Generation Sequencing
NNAL : NNK-4- (methylnitrosamino)-1-(3-pyridyl)-1-butanol
OS : Overall Survival
PAGE-B : Platelets, Age, Gender, and Encephalopathy B
PET : Positron Emission Tomography
PFS : Progression Free Survival
QOL : Quality of Life
RCTs : Randomized Controlled Trials
RNA : Ribonucleic Acid
SLT : Smokeless Tobacco
SWCNT : Single-Walled Carbon Nanotubes
TCGA : The Cancer Genome Atlas
WES : Whole Exome Sequencing
WGS : Whole Genome Sequencing

1. INTRODUCTION

Around 20% of the global population and roughly 33% of individuals in developed countries will receive a cancer diagnosis at some point during their lives [1]. Humans are frequently exposed to various DNA-damaging chemicals, with some being identified as carcinogens [2]. DNA damage results from both external and internal processes; regardless of their source, carcinogens can lead to DNA damage through multiple mechanisms [3]. These mechanisms include the covalent binding of carcinogens to DNA, or the formation of DNA double-strand breaks (DSBs) triggered by free radicals generated by ionizing radiation (IR) [4]. We can define carcinogens as physical, biological, or chemical agents, which can trigger change or damage on the DNA due to physicochemical properties, which includes deforming molecules in DNA or causing cross-linking in DNA [2]. This review explores to bridge a gap and highlight the disparity in carcinogen-related studies concerned with Africa while collating available data into a singular pool [5].

A carcinogenic substance is any material that stimulates the formation of cancer [6]. These agents can consist of man-made chemicals, natural substances, physical factors like ionizing and non-ionizing radiation, and biological entities like viruses and bacteria [7]. They can lead to mutations that cause uncontrolled cell division, bypassing the body's regulatory mechanisms, which accumulates over time [6]. Each carcinogen and cell type has its unique mode of carcinogenic action. Carcinogens are generally classified as either activation-dependent or activation-independent, indicating whether the agent can directly interact with DNA [2], resulting in DNA damage either directly [8] or indirectly [9].

Direct Acting: These substances are referred to as pro-carcinogens, they need no metabolic

activation [8] or chemical modification to inflict DNA damage [10]. Ultraviolet (UV) radiation, alkylating agents, nitrosamines and infrared radiation are the common examples [11]. The specific type of carcinogen determines how DNA bases are altered [12] leading to the formation of adducts in the DNA disruptions of materials peculiar to the gene [2]. Failure in the mechanisms of repair in the DNA leads to the passing of DNA lesions to daughter cells [8], which in turn leads to DNA damage accumulation and potentially resulting in cancer.

Indirect Acting: Typically includes inactive parent compounds, such as aristolochic acid (AA), mycotoxins, heterocyclic aromatic amines (HAAs), N-nitrosamines, and polycyclic aromatic hydrocarbons (PAHs) [3]. They are frequently induced by bioactivation within host cells to metamorphose into cancer-causing metabolites [13] or an intermediate that is very reactive and can induce genotoxicity [14]. While indirect carcinogens depend on activation, some can also enhance bioactivation by modifying gene expression [2].

Cancer causes are typically classified into two groups: internal factors (such as inherited mutations, hormones, immune responses, and metabolism-induced mutations) and external factors (including infectious agents, chemicals, tobacco, and radiation). These factors can initiate the process of carcinogenesis either independently, in sequence, or in combination [15].

2. ENVIRONMENTAL CARCINOGENS

Environmental carcinogens are defined as outdoor and indoor air pollutants, as well as toxins in soil and drinking water [17]. The growing trend in cancer incidence over the last 50–60 years can be linked to both populations aging and the spread of carcinogenic agents in occupational and general settings [18]. They can be broadly categorized into three groups: chemical (such as asbestos, tobacco smoke, and alcohol) [19], physical (including UV rays and ionizing radiation) [20], and biological (like viruses such as human papillomavirus and hepatitis B and C viruses) [19].

Classification of carcinogens according to the International Agency for Research on Cancer (IARC) [15],[16].

Group 1: There is enough data on substances in this category to prove that they cause cancer in people. When there is strong evidence from human studies that exposure to these agents causes cancer (like lung cancer, leukemia, mesothelioma and others) the IARC classifies the substance in this way [1].

Group 2A: Substances in this group are probably carcinogenic to humans, however, the evidence for this is not as compelling as it is for Group 1. Based on enough information from research on animals and scant evidence from human subjects, the categorization was made, causing cancers like glioma, renal cell carcinoma, and others [21].

Group 2B: This category of substances has less than adequate data or antecedence of their carcinogenicity in animals and little antecedence or data in humans. Although the data is insufficient to establish a clear connection between this categorization and cancer, it does indicate a possible risk of causing cancers like kidney, thyroid, gastrointestinal, and others [15].

Table 1. Classification of carcinogens according to IARC

Group	Meaning	Some examples	No. Of agents
1	Carcinogenic to humans	Cadmium, chromium IV, arsenic, nickel, plutonium, hepatitis B and C, HPV (type 16, 18, 33)	129
2A	Probably carcinogenic to humans	Indium phosphide, lead compound (inorganic), cobalt metal with tungsten carbide	96
2B	Possibly carcinogenic to humans	Hexachlorocyclohexane, goldenseal root powder, gasoline, fuel oils JC polyomavirus	321
3	Not classifiable as to its carcinogenicity to humans	Hypochlorite salts, implanted foreign bodies of metallic chromium or cobalt, jet fuel, isopropyl oils	499

Group 3: These substances are lacking sufficient or have limited antecedence or data of carcinogenicity in people and this category includes animals [1]. The classification means that the possibility of the substance causing cancer is not well supported by the available data. Group 3 substances cover a broad spectrum of chemicals and exposures for which there is insufficient or conflicting research [21].

2.1 Types of Environmental Carcinogens

2.1.1 Physical Carcinogens

Physical carcinogens include ionized radiation from X-rays, sunlight's UV rays and other radioactive elements [22]. Ultraviolet (UV) radiation can cause squamous cell carcinoma, malignant melanoma and basal-cell carcinoma in the skin [23]. The kind of UV radiation one is exposed to (UV-B rays are the most dangerous), the intensity of the exposure, and the amount of protection that the skin cells receive from the natural pigment melanin all affect one's risk of developing UV-induced cancer. Because they have less melanin to protect them from the sun, people with fair skin have the highest incidence of melanoma [24].

UV radiation most certainly acts as a full carcinogen, meaning it can both cause and encourage the growth of tumors [25].

Particulate and electromagnetic ionizing radiation are potent carcinogens, albeit it may take years for a tumor to develop after exposure, when compared to the impact of chemicals, radiation's contribution to the total number of human malignancies is likely negligible; nonetheless, the extended latency of radiation-induced tumors and the cumulative effect of repeated modest doses make it challenging to precisely calculate radiation's relevance [22].

2.1.2 Biological carcinogens

Several viruses and organisms that have been known to be "oncogenic" (are suspected of causing cancer in animals, including humans) are termed biological carcinogens. Examples include hepatitis B virus, the Epstein-Barr virus (EBV), *Helicobacter pylori,* and human papillomaviruses. The development of tumors in people is linked to an RNA virus which is called T-cell leukemia virus type I (HTLV-I) [23]. Epstein-Barr virus (EBV) and Kaposi's sarcoma-associated herpesvirus (KSHV) which are typical large DNA viruses have the potential to cause lymphoid cancers and solid tumors [26]. Infection by these viruses is approximated to cause 15–20% of all known human cancers [27]. EB virus is largely recognized to affect East Asia (Hodgkins's lymphoma), East Africa (Endemic Burkitt lymphoma) and regions of America (gastric carcinoma) [28]. HBV affects Sub-Sahara Africa, Asia and regions of South America (hepatocellular carcinoma) [29]. Human Papilloma Virus (HPV) affects Southern and Central America, Sub-Sahara Africa and regions of Asia (cervical carcinoma, oropharyngeal carcinoma and anogenital carcinoma) [30],[31].

2.1.3 Chemical carcinogens

Chemical compounds are useful to civilization in many ways. For example, the use of pesticides makes large production of food crops possible to meet the requirements of millions of people, which has increased life expectancy. However, there are occasionally drawbacks that outweigh these advantages, most notably the hazardous side effects of the chemical substances used of which can be natural and synthetic [32]. There is a wide variety of chemical carcinogens, as these are some of the most common. The Table 2 highlights some common chemical carcinogens, cancers caused, and possible uses and sources [33].

2.2 Mechanisms of Carcinogenesis

Carcinogenesis, the multistep process by which normal cells turn into cancer cells, is fueled by a complex interaction of genetic and epigenetic changes, combined with the influence of extrinsic factors [84]. This intricate procedure can be generally classified as three separate stages initiation, promotion, and progression [85].

2.3 Mechanisms of Damage by Carcinogens

2.3.1 Chemically induced bulky DNA lesions

Bulky production of chemical adducts when a water-loving (electrophilic) cancer-causing agent is generated by metabolism of an indirect carcinogen, this then binds to a specific nucleophilic site in the DNA [2]. The strength of the carcinogen's electrophile determines this binding [34]. Attaching to the N and O positions of guanine or the N position of adenine exposes the DNA to replication errors [2].

Table 2. Classification of common chemical carcinogens.

S/N	Chemical	Cancer Caused	Uses/Sources
1	Arsenic	Lung skin, Hemangiosarcoma	Components in alloys, herbicides, pesticides, fungicides etc.
2	Benzene	Leukemia (acute myeloid)	In paints, petrol, shoe repair places.
3	Chromium	Lung	Paints and in chromium alloy foundries.
4	Vinyl chloride	Liver (angiosarcoma hepatocellular carcinoma - HCC)	In the manufacture of polyvinyl chloride.
5	Sulphur mustard	Lung	Manufacture of mustard gas (WW1).
6	Shale Oils	Skin	Mining and production of shales oil and cotton textiles.
7	Pentachlorophenol (PCP)	Non-Hodgkin's lymphoma	Manufacture of PCP and other chemicals in agricultural settings, wood treatment and waste incineration.
8	Nickel compounds	Lungs, nasal cavity and paranasal sinuses	Mining, refining and smelting of nickel; production of alloys, stainless steel, nickel and batteries; electroplating; paint production and use

2.3.2 Oxidative damage

External or internal agents, such as UV radiation, ionizing radiation (IR), or naturally produced oxygen molecules, can trigger intracellular oxidative stress, leading to DNA damage induced by reactive oxygen species (ROS) like hydroxyl radicals (OH) and singlet oxygen (peroxynitrite) [35]. These ROS primarily target guanine bases by inactivating critical proteins and repair enzymes [36].

2.3.3 Cross-linking damage

DNA cross-links can be created by a variety of chemotherapeutic drugs, such as cisplatin [2], as well as by the body's natural production of acetaldehyde and malondialdehyde, which can occur alone or in combination [37]. Cross-linking agents are often mutagenic or carcinogenic, and as such, they can result in chromosomal aberrations through fragments of broken chromosomes [38].

2.3.4 Single- and double-strand breaks (SSBs and DSBs)

During base excision repair, the activity of endonuclease enzymes leads to the formation of single-strand breaks (SSBs) [39]. These SSBs can be caused by various factors, including UV radiation, ionizing radiation (IR), mycotoxins, and different intracellular reactions. If double-strand breaks (DSBs) are not properly repaired or if errors occur during repair, it can lead to cell death, genomic instability, and carcinogenesis [40]. DSBs may also be caused by IR or chemical carcinogens, either directly or indirectly [41]. Additionally, other types of damage can also induce DSBs [2].

2.3.5 Environmental and diet induced DNA damage

Lung cancer has been associated with tobacco combustion [42]. Studies on migrant communities have also documented that exposure to DNA-damaging carcinogens leads to a generational increase in susceptibility to diseases like stomach, colon, prostate, and breast cancer [43]. Compelling research supports the role of carcinogenic compounds as contributing factors [2], which include:

2.3.5.1 Lung cancer and tobacco smoking

Thousands of chemicals are produced when cigarette burns during smoking, many of which have the potential to damage DNA [44]. It is estimated that 90% of patients suffering from lung cancer are associated to smoking in nations where tobacco use is widespread [2]. Nevertheless, it is also widely known that carcinogens from smoking cause malignancies at other tissue sites [45]. PAH carcinogens are the major and common nitrosamines found in tobacco.

2.3.5.2 Prostate cancer and carcinogenic PAH exposure

Prostate cancer has historically been less common in citizens of the UK than in citizens of

countries like India, China, and Japan; however, within these ethnic groups, the risk increases in the grandchildren of migrants from these countries and these risk span towards the Northern and Western part of Europe, suggesting that environmental together with dietary exposures to PAH may be causal factors [46].The normal human prostate is recognized for its ability to express phase I and II enzymes, which can bioactivate a variety of pro-carcinogens [47].

2.3.5.3 Urothelial carcinoma and AA

Compelling evidence indicates that a carcinogen causing DNA damage is responsible for the disorders commonly referred to as Balkan endemic nephropathy also famously called Chinese herb nephropathy (CHN) and aristolochic acid nephropathy (AAN) [2]. The consumption of aristolochic acid (AA) is linked to the rapid rise of renal failure and upper tract urothelial cancer in both BEN and CHN. Formation of Aristo-lactam AL-DNA adducts in urothelial tissues due to persistent exposure to AA and metabolism, or "bioactivation" subsequently [48]. Research strongly supports that AA is not only a potent carcinogen but also a highly effective nephrotoxin, with a short latency period observed in both humans and animals [49].

3. IMPACT OF ENVIRONMENTAL CARCINOGENS ON HEALTH

The major and most notable impact of environmental carcinogens is that they can cause cancer, which drastically decreases the quality of life as well as poses great financial, economic, and psychological implications. Cancer treatments are extremely expensive or, in some cases, not even accessible in Low-and-Middle-Income Countries (LMICs). This has led to many deaths associated with a lack of proper treatment more than the severity of the cancer.

Although the relative risks of cancer from environmental carcinogen exposure may seem modest, the potential attributable risks can be significant due to the prevalence and frequency of such exposure. It was estimated by the World Health Organization (WHO) that about 20% of the cancer cases globally are linked to environmental factors, with chemical management, air pollution, workplace safety and radiation being the primary contributors. The impact of environmental carcinogens on cancer rates varies depending on a country's level of industrialization. In short, the higher the industrialization, the more the diagnosis of cancer, there are reports of fewer deaths when compared with countries with little industrialization but a higher death toll due to a lack of facilities and funds to be able to tackle this disease [50].

The duration between exposure to a cancer-causing agent and the initiation of cancerous cells of cancer is referred to as the period of latency or the latency period. Typically, it is estimated that solid tumors have a latency period of 10 to 40 years, with variation depending on the type of cancer. However, the latency period for blood cancers is sadly estimated to be within two years. Prolonged periods of latency make it difficult to identify carcinogens [51].

3.1 Prevalence, Epidemiology and Case Studies

Throughout most of the past century, infectious diseases have been the primary health concern in Africa. However, the continent is now undergoing a significant shift, marked by rapid population growth, increasing urbanization, and improvements in life expectancy, among the highest globally [52]. This transition is leading to a changing disease profile, with non-communicable diseases (NCDs) becoming more common. Data has it that NCD-related deaths are secondarily caused by cancer in Africa with cancer contributing to 17% of the continent's mortality burden from NCDs [50].

Exposure to e-waste in Nigeria due to her population has caused a high generation of it (461.3 kg-tonnes) with no adequate recycling methods [53] has been linked to its toxicity. The increased release of heavy metals has been linked to cancer [54]. The toxicity of these metals, such as lead and chromium, contributes to DNA damage and impairs the function of inflammatory cells. Research indicates that in Nigeria, exposure to toxic metals occurs through various routes, including significant leaching from e-waste into the soil [55], uptake by plants and ingestion of vegetables, seeds, fruits, and edible roots contaminated with metals [54]. Additional exposure comes from inhaling metallic fumes from the air and dust [56], and drinking metal-contaminated groundwater. Moreover, exposure can also happen through skin contact, particularly among e-waste scavengers engaged

in the primitive extraction of copper, silver, and gold, as well as at e-waste dumpsites [57].

In North Africa and Middle East Africa, it was revealed that high tobacco consumption, diet, and additional elements contributed to an increased risk of cancer. Since smoking tobacco is the primary cause of lung, bladder, squamous cell carcinoma, and colorectal cancer, be it direct or indirect exposure to the carcinogenic compound, even without being an avid smoker, being in an environment with avid smokers and constantly inhaling the fume could lead to the above [58].

In Congo, there have been reports of inhalation of radionucleotides (radon, thoron); however, though cancer-inducing, there is a low-risk potential for it; less than 2% of the population under 55 years of age were diagnosed, which could also be due to a lack of diagnosis due to the development of the nation [59].

In Nigeria, a meta-analysis of radon-risk exposure through contaminated drinking water posed a risk of cancer among non-smokers. Radon 222 concentration was estimated to be 25.01 with a 95% confidence interval. With levels at Kundiga, Ekiti, Abeokuta and Edbe exceeding the WHO recommended limit [60].

An analysis of the constituents of smokeless tobacco (SLT) products in Africa revealed the presence of carcinogenic substances. Samples were collected from five African countries, which are South Africa, Nigeria, Mauritania, Uganda, and Zambia. Analysis of the SLT was carried out, which showed the presence of nicotine, tobacco-specific N-nitrosamines (TSNA), 10 polycyclic aromatic hydrocarbons (PAHs), nitrites, and metals and metalloids (Arsenic, Cadmium, Chromium, Nickel, and Lead), all of which are classified by the IARC as being carcinogenic [61].

The occurrence of breast cancer is often linked to regional differences, suggesting that an individual's environment may play a crucial role in the development of cancer [62], which is predisposed by environmental factors such as exposure to wastewater or sewage, which is composed of a variety of carcinogenic, mutagenic, or pathogenic compounds. This wastewater has been known to be a primary source of mammary carcinogens, which promotes tumorigenesis through various processes [63]. These agents have also been found to be present in breast milk, meaning that they can accumulate in the breast and adipose tissue [64].

In 2018, there were 608,616 and 446,556 new diagnoses of cancer in females and males, respectively, in Africa. Most of its burden is associated with breast cancer, and in affected children, survival rates are as low as 20% in Africa and as high as 80% in other regions [65].

In a study in 2018, it was found that due to the high consumption of nicotine by African men, when compared to other regions, showed a high up take contained polycyclic aromatic and nitrosamine 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) hydrocarbons [66] when paired with other exomic mutations gave differences in the onset and progression of lung cancer.

Across much of sub-Saharan Africa, access to healthcare services crucial for early cancer detection, diagnosis, treatment, and care is severely limited. Additionally, the financial outlook for addressing these needs continues to worsen, leading to increasing debt burdens [52].
Heavy metals occur naturally in the environment [15]. These heavy metals include arsenic, cadmium, calcium, lead, silver, mercury, zinc and others. Human exposure to heavy metals had become a significant health risk [16], which has been linked to rapid population growth, industrialization as well as slums and poor planning has led to a very high increase in waste production [67]. In recent decades, heavy metal pollution has reached unprecedented levels due to various factors, including the expansion of mining activities, the use of leaded gasoline, the release of fugitive dust, the improper disposal and burning of hazardous waste, the placement of industrial facilities in residential areas, and insufficient pollution regulations [15].

In Pretoria, South Africa, there have been documented cases of arsenic-related skin cancers, like those reported in Zimbabwe. Additionally, a study of miners in Lesotho, Mozambique [68], and South Africa found a strong link between mining dust exposure and cancers of the lung, liver, esophagus, and lymphatic system [15].

While exposure to arsenic has been connected to cancers of the bladder, skin, lung, liver, and kidney, asbestos exposure has been associated with mesothelioma and lung cancer [69]. Indoor air pollution puts women at the highest risk for

lung cancer; however, the carcinogenic influence on children's cancer risk in these nations has not yet been investigated. Low- and middle-income countries (LMICs) have researched ambient air pollution hazards less; some relationships have been documented, but they are either weak or can be misclassified. Respiratory conditions and tobacco use have a beneficial synergistic effect on cancer chances [70].

4. PREVENTION AND SAFETY

Preventive measures offer dual benefits by not only reducing the risk of cancer but also helping to prevent other chronic diseases with similar risk factors. In developing regions, infectious agents contribute to nearly 25% of cancer deaths, compared to 6% in developed countries. In regions with elevated rates of cancer attributed to biological agents, targeted interventions will be useful in addressing these types of infections [71]. For instance, in regions where cancer of the liver is prevalent, integration of hepatitis B vaccination alongside other immunization programs, serves as a primary prevention method. Effective vaccines have been developed and shown to prevent cervical cancer in humans. Similarly, preventing HIV infection disease will lead to a significant reduction in the prevalence of HIV/AIDS-related cancers, which includes Kaposi lymphoma and sarcoma. Additionally, specific actions to control or avoid environmental and occupational carcinogens, such as excessive sun exposure among albinos in Africa, will significantly lower the rates of cancers like lung, bladder, and skin cancers [2].

4.1 Maintaining Healthy Lifestyle

Embracing a healthy lifestyle and steering clear of risk factors for Hepatocellular Carcinoma (HCC) are essential for its prevention. Avoiding excessive alcohol consumption and a calorie-rich diet that contributes to obesity and metabolic syndrome can help reduce liver damage associated with fatty liver, cirrhosis, and consequently, the risk of Hepatocellular Carcinoma [72]. Smoking is a largely agreed to be a risk factor for Hepatocellular carcinoma, and relevant data from 14 different studies conducted in U.S. show that the risk of Hepatocellular Carcinoma in people who stopped smoking over three decades ago is at equivalence to that of Individuals who have never smoked had a hazard ratio of 1.09, with a 95% confidence interval ranging from 0.74 to 1.61. This data suggests that quitting smoking significantly decreases the risk of developing Hepatocellular carcinoma [72].

Minimizing the exposure to dietary aflatoxin is essential for decreasing the incidence of Hepatocellular carcinoma in areas with high prevalence. A meta-analysis of 19 studies found that the risk of Hepatocellular carcinoma related to aflatoxin was estimated to be 17%. This risk was notably higher among Hepatitis B Virus (HBV) carriers, ranging from 21% to 23%, compared to noncarriers, where the risk ranged from 8% to 9% [72]. The analysis made use of biomarkers and dietary history to assess exposure. The International Agency for Research on Cancer in 2014 evaluated possible intervention strategies like selecting resistant seeds, improving postharvest processing, and using mycotoxin-trapping enterosorbents. These measures significantly reduced aflatoxin biomarkers in participants [72].

About 33% of cancer cases worldwide could be avoided or prevented each year [73], however, cancer progression risk depends on factors like exposure type, duration, and intensity. Reducing or avoiding exposure to carcinogens lowers cancer risk [73]. Establishing a coordinated network for primary prevention involving experts, NGOs, academic, governmental institutions, and media is essential. Avoiding occupational and environmental carcinogens, along with detoxification strategies, is crucial for individualized cancer prevention, particularly as oxidative damage from reactive species plays a key role in the pathogenesis of many carcinogens [73].

4.2 Surveillance and Early Detection

Early detection through screening asymptomatic populations and raising awareness of early cancer signs significantly improves cure rates when followed by prompt treatment [71]. This strategy is particularly crucial for cancers such as those of the cervix, breast, larynx, skin, rectum, stomach, colon, mouth, and endometrium. Currently, routine screening of the general population is recommended for breast, cervical, colon, and rectal cancers in countries that have the required resources for comprehensive coverage and adequate diagnostic and treatment capabilities. Africa faces challenges in these areas, but studies are exploring low-cost screening methods like acetic acid (VIA) for cervical cancer and alternatives to

mammography, such as clinical breast exams [71].

Hepatocellular carcinoma surveillance, considered as a secondary strategy used in the prevention which seeks the mitigation of the impact of Hepatocellular carcinoma by identifying tumors at an early stage and managing them effectively. It is advised for individuals with liver cirrhosis or chronic HBV infection, including Asian males over the age of 40, Asian females over 50 and individuals who carry HBV and have a family history of Hepatocellular carcinoma, as well as those of African or North American Black ancestry with chronic hepatitis [72]. According to European Association for the Study of the Liver (EASL) guidelines, HCC surveillance should be implemented for high-risk chronic HBV patients using the PAGE-B classification system, which estimates the period of five years risk of Hepatocellular carcinoma based on factors such as platelet count, age, and gender [72].

4.3 Cancer Prevention Agents

From a keynote address delivered by Dr. Elizabeth Jaffee, a presiding lecturer at Johns Hopkins University called 'Intercepting Pancreatic Cancer Development with Oncogene-Targeted Immunotherapy.' She discussed the challenges pancreatic tumors encounter in responding to immune therapy and stressed the advantages of early intervention, before tumors establish an immunosuppressive microenvironment. Her preclinical studies, which show the feasibility and safety of a vaccine targeting the Kirsten rat sarcoma viral oncogene homolog with six common mutations, have prompted the start of a cancer prevention trial for individuals at high risk for pancreatic cancer [74].

A presentation named "Innovative Vaccines for Cancer Prevention" concentrated on cutting-edge vaccination approaches for high-risk cancer groups. Dr. David Largaespada from the University of Minnesota showcased his research on a preventive vaccine aimed at individuals with Neurofibromatosis type 1, who are at a lifetime risk of 1 in 6 of developing malignant peripheral nerve sheath tumors [73]. He showed how RNA sequencing and mass spectrometry were employed to detect frameshift mutations and cryptic neoantigens in MPNSTs, revealing new potential targets for vaccine development [71].

During the "Cancer Prevention Clinical Trials" session, the focus was on advancing vaccine strategies for preventing cancer. Dr. Robert Keith from the University of Colorado presented findings from clinical trials involving iloprost, administered in both oral and inhaled forms. The oral version of iloprost showed promise in improving endobronchial dysplasia, a condition that can precede invasive squamous cell carcinoma in former smokers. Research into airway progenitor cell dysfunction, which is critical for sustaining normal airway epithelium, has been used to predict the progression of bronchial dysplasia. The impact of iloprost on dysplastic epithelium has been identified as a key indicator of patient response. Ongoing studies are investigating the mechanisms through which iloprost may prevent cancer, including its effects on progenitor cells and relevant biomarkers [74].

Numerous observational studies suggest that coffee, statins, metformin, and aspirin might offer protection against the onset of HCC [2]. Although these protective benefits have not been validated through randomized controlled trials (RCTs), the 2018 clinical practice recommendations from the European Association for the Study of the Liver now advocate for coffee consumption as a preventive measure against HCC [72].

4.4 Cancer Prevention with Natural Compounds

Cancer prevention is increasingly emphasized by the public, media, government, and cancer research community due to its cost-effectiveness and ability to avoid the toxic side effects of cancer treatment [75]. Cancer treatment expenses can vary widely, with some interventions being particularly costly. For example, contrast-enhanced MRI screening can cost $1,000, which is ten times more expensive than mammography. Positron Emission Tomography (PET) scans are priced at $1,800, while intensity-modulated radiation therapy for prostate cancer costs around $48,000 annually. The medication trastuzumab for HER-2 positive breast cancer amounts to $50,000 per year. Additionally, gefitinib for lung cancer is $1,800 per month, and a six-day treatment course of palifermin for oral mucositis exceeds $8,000 [5]. This cost has led to the natural use of different fruits to prevent cancer.

Consuming natural antioxidants from vegetables and fruits plays a crucial role in cancer prevention [76]. Various mechanisms suggest that phytochemicals in plants can reduce cancer

risk. According to George et al., many plant compounds protect DNA from damage caused by carcinogens, thus preventing cancer formation [76]. Green tea, the second most popular beverage after water, contains catechins, which have been shown to inhibit carcinogenesis in various animal studies [77]. The Zingiberaceae family, prevalent in Southeast Asia, includes curcumin, known for its anti-carcinogenic effects in animal and cell models. Resveratrol, found in grapes, red wine, and mulberries, has protective effects against carcinogenesis, though its molecular mechanisms are still not fully understood [78].

Plumbagin, a promising compound for cancer treatment, promotes cell death, influences key features of cancer, interacts with specific cancer targets, inhibits ABC transporters, and has good absorption properties. Its extensive use in traditional medicine and occurrence in various plants contribute to its extensive research [75].

4.5 Controlled Exposure to Nanomaterials

Findings from laboratory studies or the discovery of engineered nanomaterials (ENMs) in human tissues frequently give rise to public concerns over ENMs and health. Nevertheless, before drawing the conclusion that ENMs are dangerous, these reports should be carefully analyzed. The chance of exposure and a chemical's potential for harm determine risk, which is related to the dose-response principle. Research on lung cell cultures and inhaled ENM has raised questions regarding potential health hazards. For instance, rats exposed to Degussa P-25 titania for two years developed lung tumors as a result of the inhalation trial, and SWCNTs containing residual metals significantly harmed their lungs. It is advised to use protective garments to stop ENMs from getting inside organs [79].

4.6 Advancement to Mitigating the Effects of Carcinogens

There has been substantial improvement in the number of cancer survivors thanks to advancements in and the expansion of treatment options [76]. Initially, effective cancer treatments were primarily cytotoxic agents, which continue to play a crucial role in many therapy plans. Surgery and chemotherapy are widely used and have successfully treated millions of patients. Nonetheless, complications can occur depending on the patient's overall health, and these methods may not be appropriate for cases of advanced cancer [76].

The development of innovative technologies that apply genetic therapy for the treatment of chronic illnesses including cancer has proved very essential, motivated by the necessity to overcome the constraints of conventional treatments [77]. Recent advancements, particularly in bioinformatics and high-throughput genomic approaches, have enabled the identification of specific mutations for targeted gene delivery. The idea of gene therapy was initially introduced by Rogers and Pfuderer in the 1960s. Since its inception, it has demonstrated significant potential as a treatment for cancer and various other conditions. In 1971, Carl R. made groundbreaking advances by introducing foreign DNA into human fibroblast cells [80]. Over the years, extensive research and clinical trials have marked the evolution and success of gene therapy, especially in recent times, which are considered its golden age [80].

The potential of various gene therapy techniques to treat cancers is now widely recognized, as evidenced by the growing number of clinical trials each year [76]. Various approaches have been utilized to address different cancer types, such as naked DNA therapy, microRNA targeting, oncolytic virotherapy, therapies targeting telomerase, introduction of tumor suppressor genes, and gene-directed enzyme prodrug therapy, among others [74].

The comprehensive sequencing projects, Whole Exome Sequencing (WES) and Whole Genome Sequencing (WGS), have identified cancer as a genome-driven, multifactorial disease, with most cases having a non-Mendelian (somatic) origin, while inherited cancers follow a Mendelian pattern. Thanks to initiatives like TCGA (The Cancer Genome Atlas) and ICGC (International Cancer Genome Consortium), extensive data on gene alterations in protein-coding regions for various cancers are now accessible [81]. Although not all alterations identified by NGS (Next Generation Sequencing) have immediate applications in translational medicine, they contribute to our understanding of cancer pathways and enhance the cancer genomics database. This has facilitated the development of lung cancer biomarkers in the last ten years, resulting in a commercial NGS panel designed for precision oncology. This panel targets 15–21 genes and enables the identification of different

structural variants all within a particular platform [82]. This breakthrough in lung cancer precision oncology paved the way for using NGS panels in other solid and liquid tumors, such as CRC, breast, ovarian, and hematological malignancies, with minimal sample requirements and specialized expertise [81]. Thorough gene testing in cancer improves treatment effectiveness, slows disease advancement, and boosts patients' quality of life (QOL), progression-free survival (PFS), and overall survival (OS) [83]

5. CONCLUSION

Key concerns in Africa should address not only the immediate adverse effects of environmental factors, whether natural or human-made, but also their long-term carcinogenic consequences. By reaffirming the Asturias Declaration of March 2011 concerning the global primary prevention of environmental and occupational cancers, we aim to enhance international cooperation and research in this area. Apart from aspects related to lifestyle, the environmental and occupational domains as well as to bridge the gap in the paucity of information available on studies pertaining to carcinogens and cancer in Africa. More research needs to be conducted across several African countries to fully understand health indices that impacts carcinogens and cancer development in Africa.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Authors declare that NO generative AI technologies (ChatGPT, COPILOT) and text-to-image generators have been used during the writing of this manuscript

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. IARC. World Cancer Report. International Agency for Research on Cancer (IARC); Lyon, France; 2014.
2. Jessica L. Barnes, Maria Zubair, Kaarthik John, Miriam C. Poirier, Francis L. Martin; Carcinogens and DNA damage. Biochem Soc Trans 19 October. 2018;46(5):1213–1224.
Available:https://doi.org/10.1042/BST20180519
3. Smith MT, Guyton KZ, Gibbons CF, Fritz JM, Portier CJ, Rusyn I, DeMarini DM, Caldwell JC, Kavlock RJ, Lambert PF, Hecht SS. Key characteristics of carcinogens as a basis for organizing data on mechanisms of carcinogenesis. Environmental Health Perspectives. 2016;124(6):713-21.
4. Chakarov S, Petkova R, Russev GC, Zhelev N. DNA damage and mutation. Types of DNA damage. BioDiscovery. 2014;11:e8957.
5. Desouky O, Ding N, Zhou G. Targeted and non-targeted effects of ionizing radiation. Journal of Radiation Research and Applied Sciences. 2015;8(2):247-54.
6. "Carcinogen". www.genome.gov. Retrieved 2024-08-12
7. "Carcinogenesis". McGraw Hill Medical. Retrieved 2024-08-12.
8. Cohen SM, Arnold LL. Chemical carcinogenesis. Toxicological Sciences. 2011;120(suppl_1):S76-92.
9. Wohak LE, Krais AM, Kucab JE, Stertmann J, Øvrebø S, Seidel A, Phillips DH, Arlt VM. Carcinogenic polycyclic aromatic hydrocarbons induce CYP1A1 in human cells via a p53-dependent mechanism. Archives of Toxicology. 2016;90:291-304.
10. Ravanat JL, Douki T. UV and ionizing radiations induced DNA damage, differences and similarities. Radiation Physics and Chemistry. 2016;128:92-102.
11. Hebels DG, Briedé JJ, Khampang R, Kleinjans JC, de Kok TM. Radical mechanisms in nitrosamine-and Nitrosamide-induced whole-genome gene expression modulations in Caco-2 cells. Toxicological Sciences. 2010;116(1):194-205.
12. Kondo N, Takahashi A, Ono K, Ohnishi T. DNA damage induced by alkylating agents and repair pathways. Journal of Nucleic Acids. 2010;2010(1):543531.
13. Wohak LE, Krais AM, Kucab JE, Stertmann J, Øvrebø S, Seidel A, Phillips DH, Arlt VM. Carcinogenic polycyclic aromatic hydrocarbons induce CYP1A1 in human cells via a p53-dependent

mechanism. Archives of Toxicology. 2016;90:291-304.
14. Walsh AA, Szklarz GD, Scott EE. Human cytochrome P450 1A1 structure and utility in understanding drug and xenobiotic metabolism. Journal of Biological Chemistry. 2013;288(18):12932-43.
15. Fasinu PS, Orisakwe OE. Heavy metal pollution in sub-Saharan Africa and possible implications for cancer epidemiology. Asian Pac J Cancer Prev. 2013;14(6):3393-402.
DOI: 10.7314/APJCP.2013.14.6.3393
16. Malarkey DE, Hoenerhoff M, Maronpot RR. Carcinogenesis: Mechanisms and manifestations. In: Haschek WM, Rousseaux CG, Wallig MA, Bolon B, Haschek-Hock WM, editors. Haschek and Rousseaux's Handbook of Toxicologic Pathology. 3rd ed. Amsterdam: Elsevier. 2013;107-46.
DOI: 10.1016/B978-0-12-415759-0.00005-4
17. Boffetta P, Nyberg F. Contribution of environmental factors to cancer risk. Br Med Bull. 2003;68(1):71-94.
DOI: 10.1093/bmp/ldg023
18. Tebourbi O, Sakly M, Rhouma KB. Molecular mechanisms of pesticide toxicity. Pesticides in the Modern World-Pests Control and Pesticides Exposure and Toxicity Assessment. 2011:297-332.
19. Blake M. What you should know about carcinogens; 2022.
Available:https://www.medicalnewstoday.com/articles/what-is-a-carcinogen
20. Cancer; 2022.
Available:https://www.who.int/news-room/fact-sheets/detail/cancer
21. International Agency for Research on Cancer (IARC). IARC Monographs on the Identification of Carcinogenic Hazards to Humans. World Health Organization; 2020.
22. Blake M. What you should know about carcinogens; 2022.
Available:https://www.medicalnewstoday.com/articles/what-is-a-carcinogen#Types-of-carcinogens.
23. Britannica T. Editors of Encyclopaedia. "Carcinogen." Encyclopedia Britannica; 2024.
Available:https://www.britannica.com/science/carcinogen.
24. Melanin | Biological Pigment, Skin Color, Sun Protection. Encyclopedia Britannica; 2024.
Available:https://www.britannica.com/science/melanin.
25. Costa, J. "cancer." Encyclopedia Britannica; 2024.
Available:https://www.britannica.com/science/cancer-disease.
26. Krump NA, You J. Molecular mechanisms of viral Oncogenesis in humans. Nat Rev Microbiol. 2018;16(11):684-698.
DOI: 10.1038/s41579-018-0064-6
27. Zur Hausen H, De Villiers EM. Cancer "causation" by infections—individual contributions and synergistic networks. In Seminars in Oncology. WB Saunders. 2014;41(6):860-875.
28. Raab-Traub N. Novel mechanisms of EBV-induced oncogenesis. Current Opinion in Virology. 2012;2(4):453-8.
29. Levrero M, Zucman-Rossi J. Mechanisms of HBV-induced hepatocellular carcinoma. Journal of hepatology. 2016;64(1):S84-101.
30. Schiffman M, Doorbar J, Wentzensen N, De Sanjose S, Fakhry C, Monk BJ. Carcinogenic human papillomavirus infection. Nature Reviews Disease Primers. 2016;2:16086. Epub 2016/12/03. Available:https://doi. org/10.1038/nrdp. 2016.86 PMID: 27905473; 2016.
31. Harper DM, DeMars LR. HPV vaccines–a review of the first decade. Gynecologic Oncology. 2017;146(1):196-204.
32. Oliveira PA, Colaço A, Chaves R, Guedes-Pinto H, P LFD-L-C, Lopes C. Chemical carcinogenesis. Anais Da Academia Brasileira De Ciências. 2007;79:593–616.
Available:vhttps://doi.org/10.1590/s0001-37652007000400004
33. Loomis D, Guha N, Hall AL, Straif K. Identifying occupational carcinogens: An update from the IARC Monographs. Occup Environ Med. 2018;75(8):593-603.
DOI: 10.1136/oemed-2017-104944.
34. Rajalakshmi TR, Aravindha Babu N, Shanmugam KT, Masthan KM. DNA adducts-chemical addons. Journal of Pharmacy and Bioallied Sciences. 2015;7(Suppl 1):S197-9.
35. Kryston TB, Georgiev AB, Pissis P, Georgakilas AG. Role of oxidative stress and DNA damage in human carcinogenesis. Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis. 2011;711(1-2):193-201.
36. Evans MD, Dizdaroglu M, Cooke MS. Oxidative DNA damage and disease:

Induction, repair and significance. Mutation Research/Reviews in Mutation Research. 2004;567(1):1-61.
37. Huang Y, Li L. DNA crosslinking damage and cancer-a tale of friend and foe. Translational cancer Research. 2013;2(3):144.
38. Noll DM, Mason TM, Miller PS. Formation and repair of interstrand cross-links in DNA. Chemical Reviews. 2006;106(2):277-301.
39. Horváthová E, Slameňová D, Hlinčíková L, Mandal TK, Gábelová A, Collins AR. The nature and origin of DNA single-strand breaks determined with the comet assay. Mutation Research/DNA Repair. 1998;409(3):163-71.
40. Jeggo PA, Lobrich M. DNA double-strand breaks: their cellular and clinical impact? Oncogene. 2007;26(56):7717-20.
41. Cannan WJ, Pederson DS. Mechanisms and consequences of double-strand DNA break formation in chromatin. Journal of Cellular Physiology. 2016;231(1):3-14.
42. Hecht SS. Lung carcinogenesis by tobacco smoke. International Journal of Cancer. 2012;131(12):2724-32.
43. Martin FL. Epigenetic influences in the Aetiology of cancers arising from breast and prostate: A hypothesised transgenerational evolution in chromatin accessibility. International Scholarly Research Notices. 2013;2013(1):624794.
44. ARC. Tobacco Smoke and Involuntary Smoking. International Agency for Research on Cancer (IARC) Monographs on the Evaluation of Carcinogenic Risks to Humans Volume 83, IARC, Lyon, France; 2004.
45. Vargas AJ, Harris CC. Biomarker development in the precision medicine era: lung cancer as a case study. Nature Reviews Cancer. 2016;16(8):525-37.
46. Singh PB, Ragavan N, Ashton KM, Basu P, Nadeem SM, Nicholson CM, Krishna RG, Matanhelia SS, Martin FL. Quantified gene expression levels for phase I/II metabolizing enzyme and estrogen receptor levels in benign prostate from cohorts designated as high-risk (UK) versus low-risk (India) for adenocarcinoma at this organ site: a preliminary study. Asian Journal of Andrology. 2010; 12(2):203.
47. Martin FL, Patel II, Sozeri O, Singh PB, Ragavan N, Nicholson CM, Frei E, Meinl W, Glatt H, Phillips DH, Arlt VM. Constitutive expression of bioactivating enzymes in normal human prostate suggests a capability to activate pro-carcinogens to DNA-damaging metabolites. The Prostate. 2010; 70(14):1586-99.
48. Yang HY, Chen PC, Wang JD. Chinese herbs containing aristolochic acid associated with renal failure and urothelial carcinoma: A review from epidemiologic observations to causal inference. BioMed Research International. 2014;2014(1): 569325.
49. Arlt VM, Stiborova M, Schmeiser HH. Aristolochic acid as a probable human cancer hazard in herbal remedies: A review. Mutagenesis. 2002;17(4):265-77.
50. World Health Organization (WHO). Cancer Prevention and Control. WHO; 2020. Available:https://www.who.int
51. Ladou Harrison. Current Diagnosis and Treatment Occupational and Environmental Medicine (6th ed.). McGraw Hill Lange. ISBN 978-1-260-14343-0. 2014;389–418.
52. McCormack VA, Schüz J. Africa's growing cancer burden: Environmental and occupational contributions. Cancer Epidemiol. 2012;36(1):1-7. DOI: 10.1016/j.canep.2011.09.005
53. Andeobu L, Wibowo S, Grandhi S. An assessment of e-waste generation and environmental management of selected countries in Africa, Europe and North America: A systematic review. Science of the Total Environment. 2021;792: 148078.
54. Owonikoko WM, Alimba CG. Systematic literature review of heavy metal contamination of the Nigerian environment from e-waste management: Associated health and carcinogenic risk assessment. Toxicology. 2024;505:153811. DOI: 10.1016/j.tox.2024.153811
55. Adeyi AA, Olayanju B, Fatade Y. Distribution and potential risk of metals and metalloids in soil of informal E-waste recycling sites in Lagos, Nigeria. Ife Journal of Science. 2019;21(3):213-33.
56. Adaramodu AA, Osuntogun AO, Ehi-Eromosele CO. Heavy metal concentration of surface dust present in e-waste components: The Westminister Electronic Market, Lagos case study. Resources and Environment. 2012;2(2):9-13.
57. Alabi OA, Adeoluwa YM, Bakare AA. Elevated serum Pb, Ni, Cd, and Cr levels

and DNA damage in exfoliated buccal cells of teenage scavengers at a major electronic waste dumpsite in Lagos, Nigeria. Biological Trace Element Research. 2020;194:24-33.

58. Mansour R, Al-Ani A, Al-Hussaini M, Abdel-Razeq H, Al-Ibraheem A, Mansour AH. Modifiable risk factors for cancer in the middle East and North Africa: A scoping review. BMC Public Health. 2024;24(1):223. Published 2024 Jan 18.
DOI: 10.1186/s12889-024-17787-5
59. Sondzo JS, Dallou GB, Meye PO, et al. Simultaneous measurements of radon, thoron and thoron progeny and induced cancer risk assessment in Djeno, Pointe-Noire, Republic of Congo. Radiat Prot Dosimetry. 2024;200(5):437-447.
DOI: 10.1093/rpd/ncad314
60. Adamu UM, Ahmad ZA, Mohammad FR, Noorain MI, Abdullahi SA, Muyiwa MO. A systematic review and meta-analysis of radon risk exposure from drinking water resources in Nigeria. Journal of Environmental Science Health Toxicology. 2023;41(3-4):150-174.
DOI: 10.1080/26896583.2023.2278957
61. Gomez F, Ayo-Yusuf O, Yershova K. Heterogeneity of harmful constituent profiles in smokeless tobacco products from five African countries. Chem Res Toxicol. 2023;36(12):1901-1911.
DOI: 10.1021/acs.chemrestox.3c00181
62. Du Plessis M, Fourie C, Stone W, Engelbrecht AM. The impact of endocrine disrupting compounds and carcinogens in wastewater: Implications for breast cancer. Biochimie. 2023;209:103-115.
DOI: 10.1016/j.biochi.2023.02.006
63. Pandey S. A comprehensive review on recent developments in bentonite-based materials used as adsorbents for wastewater treatment. J Mol Liq. 2017;241:1091-113.
DOI: 10.1016/j.molliq.2017.06.115
64. Siegel R, Ma J, Zou Z, Jemal A. Cancer statistics, 2014. CA: A cancer Journal for Clinicians. 2014;64(1).
65. Patel YM, Park SL, Carmella SG, Paiano V, Olvera N, Stram DO, et al. Metabolites of the polycyclic aromatic hydrocarbon phenanthrene in the urine of cigarette smokers from five ethnic groups with differing risks for lung cancer. Plos One. 2016;11(6):e0156203.
DOI: 10.1371/journal.pone.01562033
66. Murphy SE, Park SL, Balbo S, Haiman CA, Hatsukami DK, Patel Y, et al. Tobacco biomarkers and genetic/epigenetic analysis to investigate ethnic/racial differences in lung cancer risk among smokers. NPJ Precis Oncol. 2018;2:17.
DOI: 10.1038/s41698-018-0057-y
67. Scheinberg A, Spies S, Simpson MH, Mol AP. Assessing urban recycling in low-and middle-income countries: Building on modernised mixtures. Habitat International. 2011;35:188-98
68. Mc Glashan ND, Harington JS, Chelkowska E. Changes in the geographical and temporal patterns of cancer incidence among black gold miners working in South Africa 1964 1996Br. J. Cancer. 2003;88:1361-9.
69. Lacourt A, Gramond C, Audignon S, Ducamp S, Févotte J, Soit Ilg AG, Goldberg M, Imbernon E, Brochard P. Pleural mesothelioma and occupational coexposure to asbestos, mineral wool, and silica. American Journal of Respiratory and Critical Care Medicine. 2013;187(9):977-82.
70. Hashim D, Boffetta P. Occupational and environmental exposures and cancers in developing countries. Ann Glob Health. 2014;80(5):393-411.
DOI: 10.1016/j.aogh.2014.10.002
71. World health Organisation. Key Prevention and Control Key Prevention and Control Interventions for Reducing Cancer Burden in the Who African Region. WHO Reg. Off. Africa; 2012.
Available:http://www.who.int.
72. Yang JD, Hainaut P, Gores GJ, Amadou A, Plymoth A, Roberts LR. A global view of hepatocellular carcinoma: Trends, risk, prevention and management. Nat Rev Gastroenterol Hepatol. 2019;16(10):589-604.
DOI: 10.1038/s41575-019-0186-y
73. Yang M. A current global view of environmental and occupational cancers. J. Environ. Sci. Heal. - Part C Environ. Carcinog. Ecotoxicol. Rev. 2011;29(3):223–249.
DOI: 10.1080/10590501.2011.601848.
74. Miller MS, Mohammed A. Emerging Trends in Cancer Prevention Agent Development. J. Cancer Prev. 2023;28(1):24–28.
DOI: 10.15430/jcp.2023.28.1.24
75. Gaascht F, Dicato M, Diederich M. Venus Flytrap (Dionaea muscipula Solander ex

Ellis) Contains Powerful Compounds that Prevent and Cure Cancer. Front Oncol. 2013;3:202. Published 2013 Aug 20. DOI: 10.3389/fonc.2013.00202

76. Yahya EB, Alqadhi AM. Recent trends in cancer therapy: A review on the current state of gene delivery. Life Sci. 2021;269(2020):119087. DOI: 10.1016/j.lfs.2021.119087
77. Baguley BC. Multiple drug resistance mechanisms in cancer. Molecular Biotechnology. 2010;46:308-16.
78. Gullett NP, Amin AR, Bayraktar S, Pezzuto JM, Shin DM, Khuri FR, Aggarwal BB, Surh YJ, Kucuk O. Cancer prevention with natural compounds. In Seminars in Oncology. WB Saunders. 2010;37(3):258-281.
79. Yokel RA, MacPhail RC. Engineered nanomaterials: Exposures, hazards, and risk prevention. J. Occup. Med. Toxicol. 2011;6(1):1–27. DOI: 10.1186/1745-6673-6-7.
80. Malech HL, Garabedian EK, Hsieh MM. Evolution of gene therapy, historical perspective. Hematol. Oncol. Clin. North Am. 2022;36(4):627–645. DOI: 10.1016/j.hoc.2022.05.001
81. Satam H, Joshi K, Mangrolia U, Waghoo S, Zaidi G, Rawool S, Thakare RP, Banday S, Mishra AK, Das G, Malonia SK. Next-generation sequencing technology: Current trends and advancements. Biology. 2023;12(7):997.
82. Aramini B, Masciale V, Banchelli F, D'amico R, Dominici M, Haider KH. Precision medicine in lung cancer: Challenges and opportunities in diagnostic and therapeutic purposes. In Lung Cancer; Intech Open: Rijeka, Croatia; 2021.
83. Lee CS, Song IH, Lee A, Kang J, Lee YS, Lee IK, Song YS, Lee SH. Enhancing the landscape of colorectal cancer using targeted deep sequencing. Sci. Rep. 2021;11:8154.
84. Bhat AS, Ahmed M, Abbas K, Mustafa M, Alam M, Salem MAS, Islam S, Tantry IQ, Singh Y, Fatima M, Usmani N, Faraz M, Afeef M, Habib S. Cancer initiation and progression: A comprehensive review of carcinogenic substances, anti-cancer therapies, and regulatory frameworks. Asian Journal of Research in Biochemistry; 2024. Available:https://doi.org/10.9734/ajrb/2024/v14i4300
85. Ciriello G, Magnani L, Aitken SJ, Akkari L, Behjati S, Hanahan D, Landau DA, Lopez-Bigas N, Lupiáñez DG, Marine JC, Martin-Villalba A, Natoli G, Obenauf AC, Oricchio E, Scaffidi P, Sottoriva A, Swarbrick A, Tonon G, Vanharanta S, Zuber J. Cancer evolution: A multifaceted affair. Cancer. 2024;36-48.

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