INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA):IJPS00]
Journal Homepage: https://www.ijpsjournal.com

OPEN ACCESS

Review Article

An Overview Of The Most Recent Cancer Treatments, As Well As The Origins And Mechanisms Of Several Cancer Types

Shraddha Srivastava*[1], Priyanka sonker[2]

[1]*Institute of Technology and Management, Gorakhpur*
[2]*Institute of Technology and Management, Gorakhpur*

ARTICLE INFO

Received: 23 May 2024
Accepted: 27 May 2024
Published: 06 June 2024

Keywords:
cancer, antioxidant, chemotherapy, treatment

DOI:
10.5281/zenodo.12589301

ABSTRACT

Globally, cancer is a leading illness with a high death rate. After cardiovascular disease, cancer is regarded as the second most prevalent cause of mortality in many nations. The clinical management of cancer remains a challenging task due to the limitations of traditional therapies like chemotherapy and radiation treatment, as well as their respective toxicity profiles, have limits. The two main risk factors for cancer are an unhealthy lifestyle and poor eating habits; however, risk may be reduced by leading a healthy lifestyle. Because fruits and vegetables are rich in antioxidants and phytochemicals, epidemiological research has shown that include a high fruit and vegetable consumption in a regular diet may effectively lower the chance of getting some forms of cancer. With targeted treatment, there was reduced chance of harm to healthy cells while preventing the development and spread of certain cancer cells. An emerging less invasive method for burning or freezing tumors without open surgery is ablation treatment. Natural antioxidants have shown promise in locating free radicals and reducing their damaging effects, which may help cure or prevent cancer. Clinical trials are now being conducted on a number of novel technologies, some of which have already received approval. An update on current developments and discoveries in cancer treatments was provided by this review.

INTRODUCTION

A illness called cancer includes uncontrollably growing cells that have the ability to spread to other areas of the body. It will account for around 10 million fatalities worldwide in 2020, making it a major cause of mortality [1]. New approaches to managing the illness are being looked for as the global incidence of cancer keeps rising. Radiation exposure, hormonal variables, dietary and lifestyle choices, and other factors may all have a role in the development of cancer, which is a complex illness [2]. Lifestyle variables, including alcohol intake, smoking, and food habits, are the key objectives for primary prevention and are thought to have a

***Corresponding Author:** Shraddha Srivastava
Address: *Institute of Technology and Management, Gorakhpur*
Email : srsraddha00@gmail.com

Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

substantial role in the genesis of cancer. The potential one cannot ignore the link between food and the development of cancer. For instance, diets heavy in red and processed meats have been connected to a higher risk of colon cancer, while diets high in fat have been related to a higher risk of breast cancer [3,4]. Foods that are salted, pickled, or smoke-cured are associated with a higher risk of stomach cancer. Foods rich in fat and/or low in fiber are linked to problems with the colon, prostate, pancreas, breast, endometrial, and ovarian cancers. The kind and severity of the cancer determine how the disease is treated clinically. The majority of patients have several treatments, such as radiation and chemotherapy in addition to surgery. Other techniques including immunotherapy, gene therapy, photodynamic and thermal therapy, and others have also become cutting-edge cancer therapies. Bioactive compounds called phytonutrients are present in plants and are well-known for their anti-inflammatory and antioxidant properties for people. Flavonoids and anthraquinones are two of these phytonutrients that are known to shield the body from different kinds of cancer [5,6].

Mechanism of cancer:

Cancer is a hereditary illness that can result from both internal genetic alterations and the combined impact of several external causes. At the cellular level, somatic mutation in the DNA and subsequent exposure to carcinogenic stimuli are what cause this malignancy to grow [6]. This somatic mutation entails gene translocation and strengthening, which results in a unique expression of the cell type known as reformed genes. Proto-oncogenes are the name given to these altered genes. Numerous cell duplication sequences cause the genetic harm to generally become permanent. A substance is deemed genotoxic if it causes mutations or the breakdown of DNA within a cell. A genotoxin may exert its effects directly or indirectly. Direct-acting genotoxins include ethylene imine and its chloromethyl ether. Aflatoxin and hepatitis B virus are examples of toxins that have been linked to the genesis of hepatocellular carcinoma, whereas tobacco and alcohol use are risk factors for mouth cancer. For indirect-acting genotoxins to cause cancer, their metabolism must be activated. Polycyclic aromatic hydrocarbons are one example Aromatic amines and polycyclic aromatic hydrocarbons have been connected to bladder and lung cancer, respectively [7]. It is often accepted that lifestyle and nutrition have a major role in the development of cancer. Our diet contains a number of ingredients that are mutagenic and carcinogenic [8,9]. Edible mushrooms contain hydrazine, whereas spices and flavorings contain alkenylbenzene and safrole, both of which have been linked to cancer. Furthermore, mycotoxins like aflatoxin that are found in rotten food have been demonstrated to cause cancer and weaken the immune system by fungus [10,11]. Because of their ability to scavenge free radicals, phytochemicals included in our food have the potential to control the genesis of cancer and slow the growth of tumors.They could have a favorable impact on oxidative stress response, inflammation, and cell signaling systems. The protective effects of flavonoids, carotenoids, phenolic acids, and organosulfurs on the downregulation of certain carcinogenic pathways are well-documented [12,13].

Various Forms of Cancer and Their Underlying Causes:

Cancers are categorized either by the type of tissue they originally appeared in or by the organ in which they did. Furthermore, some malignancies have many kinds. Cancer develops slowly, progresses slowly, and occurs seldom. It can take a few of years to materialize. One of the main contributing factors to the development of cancer is eating big amounts of food. In wealthy nations, bad eating habits have been linked to 30% of

cancer cases [11]. Still, only 20% of cancer cases worldwide are attributable to food, which is comparatively lower in underdeveloped nations [14]. Research suggests that 50% of all cases of breast cancer, 70% of cases of colon cancer, and 50% of cases of gallbladder cancer may be caused by poor nutrition [2]. Obesity has been found to have a strong positive correlation with increased death rates from a variety of malignancies, including cervical, kidney, esophageal, breast, and pancreatic cancers. Scientists surmise that this is mostly because of the inflammation brought on by visceral fat [15,16]. Approximately 20% of cancer-related fatalities in women and 14% in men are connected to overweight or obesity [17]. Approximately 20% of cancer-related fatalities in women and 14% in men are connected to overweight or obesity [18,19]. Epidemiological studies have consistently demonstrated a robust correlation between lower rates of cancer and cardiovascular disease mortality and higher consumption of whole grain products, vegetables, and fruits illnesses, the world's two leading causes of mortality [20,21]. A broad understanding of the connection between diet and malignancies is provided by Figure 1.

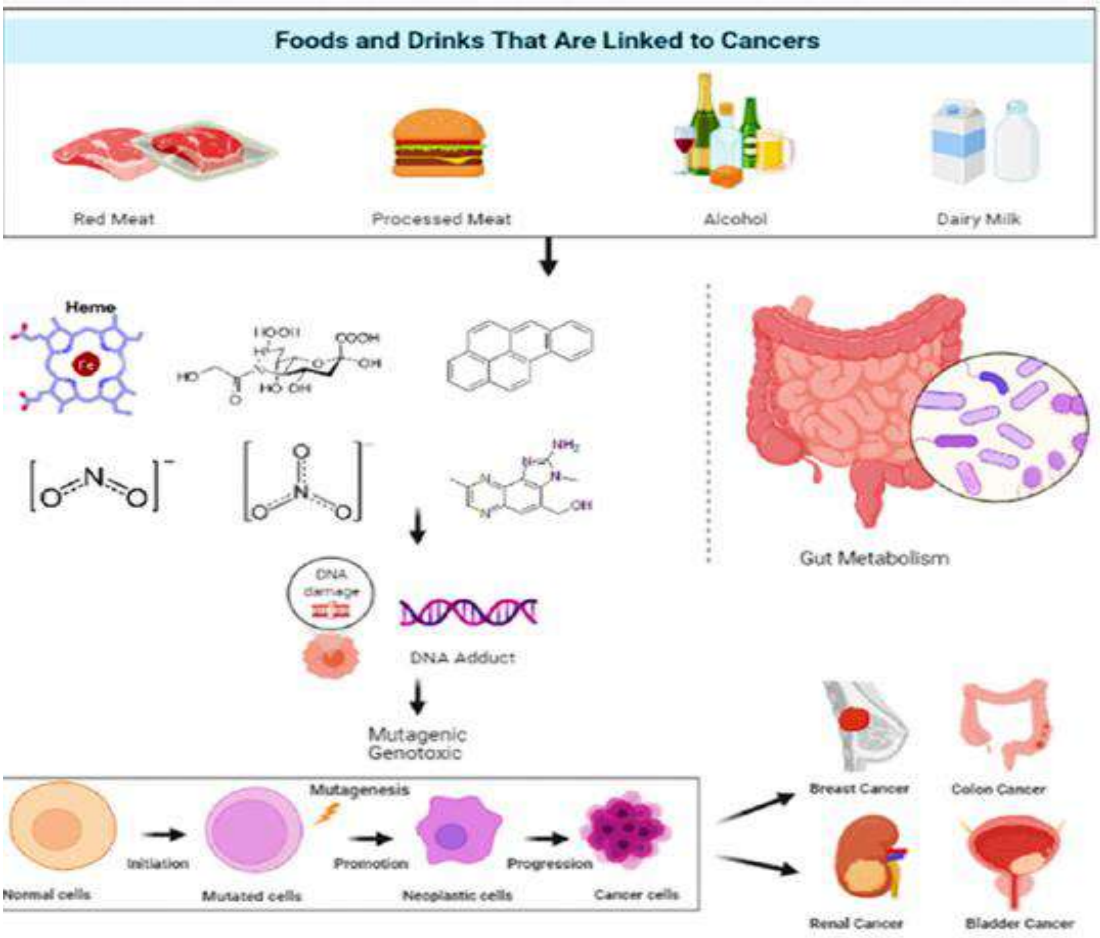

Figure 1: A Diagram Showing The Different Dietary Categories And How They Affect Certain Forms Of Cancer

Colon Cancer:

For men, colorectal cancer ranks third in terms of cancer-related deaths; for women, it is second [22], and is in fourth place worldwide for cancer-related mortality [19,22]. Numerous investigations have demonstrated that the Western diet pattern is associated with an increased risk of colorectal cancer, while diets high in fiber and whole grains are associated with a decreased risk of colorectal cancer [23-27]. Rats fed a diet high in fried meat

had higher levels of mutagenic activity in their urine and feces than germ-free rats, according to in vivo research investigating the role of gut bacteria in mutagenic activation. A connection between colon and rectal cancer and a diet high in meat was thus established [28,29]. lactic acid bacteria Lactobacillales, which are present in fermented dairy products, decrease the amount of pro-carcinogens in the gut by reducing the concentration of the enzymes -glucosidase, nitroreductase, glucuronidase, and azoreductase, which are responsible for converting pro-carcinogens into carcinogens [30]. It was also shown that consuming curd on occasion provided protection against colon cancer. Probiotics have been studied in animal experiments for their potential to reduce cancer incidence during a 20-week period. For example, rats fed a diet consisting only of beef saw a 77% incidence of colon cancer, whereas rats fed a diet consisting of beef plus Lactobacillus acidophilus exhibited a 40% incidence [31].

Bladder Cancer:

Bladder cancer ranks tenth among all cancers in males and is the most prevalent malignancy of the urinary system [32]. Males are far more likely than females to have this malignancy [32]. Approximately half of all bladder cancer cases in both men and women are caused by smoking, making it the most significant risk factor for the disease. Bladder cancer has also been connected to occupational exposure to aromatic amines used in the dye industry. Sectors that printing firms and leather, rubber, textiles, and paint pose more dangers. Aristolochic acid-containing dietary supplements may increase the risk of urothelial malignancies, such as bladder cancer [33]. No one food by itself can prevent cancer. However, research shows a healthy diet filled with various fruits, vegetables, whole grains and other plant foods could inhibit carcinogenic development, consequently preventing bladder cancer incidence. People who drink a lot of fluids, especially water, each day tend to have lower rates of bladder cancer [34]. Research on animals has demonstrated an inverse relationship between the frequency of urine and the concentration of possible carcinogens in the urothelium. By diluting uriney metabolites, an increase in total fluid intake tends to shorten the duration of interaction between carcinogens and the urothelium and increasing how often you urinate. In a randomized trial, 65 smokers who increased their water intake for fifty days had a significant reduction in urine mutagenicity [35]. A research showed that the T24 cell line was significantly affected by the apoptotic effects of the oligomers of epicatechin, resveratrol, and catechin. Conversely, catechin monomers, resveratrol, and Epicatechin did not exhibit anticancer effects on T24 cells, as evidenced by the cell viability being relatively unchanged when compared to the control group [36].

Breast Cancer:

Hormonal imbalance has been linked to the development of cancer in some sensitive tissues, including the breast, according to observations [37]. When some processes that regulate cell development and division are interfered with by mutations in the DNA of breast cells, breast cancer results. Breast cancer risk factors include early or late menstruation menopause. A higher risk of postmenopausal breast cancer might probably be caused by increased endogenous estrogen levels in postmenopausal women. Additional risk factors include obesity, a sedentary lifestyle, hormonal imbalances, late reproductive age, and alcohol consumption [38,39]. Breast cancer development has been demonstrated to be positively correlated with increased consumption of total and saturated fat. Nonetheless, a another study including 90,000 nurses did not discover a link between dietary fat intake and the prevalence of breast cancer [40-42]. Glycemic load and glycemic index diets have been associated with a higher risk of breast cancer.

Nevertheless, a meta-analysis revealed no link between the incidence of postmenopausal breast cancer and the amount of glucose ingested [43].

Renal Cancer:

Renal cancer is a rare cancer that makes up around 2% of all malignancies, although its incidence rate has been rising globally recently. Although the exact cause of this kind of cancer is unknown, the most well-established risk factors—smoking, hypertension, and obesity—are thought to be responsible for about 40% of all causes, along with certain dietary variables [44,45]. A meta-analysis research was carried out in Europe to investigate the relationship between the incidence of kidney cancer and dietary intake of carbs, protein, fat, fiber, and several other components. The findings demonstrated that there was no relationship between the risk of kidney carcinoma and macronutrient intakes. The findings validated the hypothesis that there is a negative correlation between the risk of kidney carcinoma and dietary fiber intake. According to an American cohort research, eating more fiber overall significantly reduced the chance of developing kidney cancer by 15–20% [46]. as renal cell carcinoma is a cancer linked to obesity, eating fiber may be beneficial as it increases satiety and causes weight loss by lengthening the transit time of the stool [47]. People with high GI diets who are obese and hypertensive have a 2.7-fold increased risk of kidney cancer compared to people without these conditions. The same study found that the incidence of kidney cancer was associated with GI but not GL, indicating that the quality rather than the amount of carbs may be more important. Certain meals, such processed and sugary foods, include chemicals that might cause or exacerbate inflammation [48]. Another study found a correlation between low intakes of magnesium or vitamin E and eating fried or sautéed beef, which raised the risk of kidney cancer. The data suggests a potential link between nutrition and the development of renal cancer, despite the fact that there are conflicting and definitive findings about food and renal cancer. The dietary categories that are thought to be protective include whole grains, nuts, butter, fried foods, and alcohol. The food groups that showed the biggest rise were butter, fried foods, and whole leafy greens [49,50].

Traditional Oncology Therapy:

In addition to more modern methods like immunotherapy and interventional radiology, effective cancer therapies also include surgery, chemotherapy, and radiation. The kind of cancer and its stage of progression determine the kind of treatment a patient gets. Furthermore, the greatest outcomes typically need a mix of therapy [51].Below is a summary of a quick talk on traditional cancer therapy. The traditional therapy used in may be seen in Figure 2. Table 1 outlines the many cancer therapy approaches.

Chemotherapy:

Chemotherapy is the term for applying chemical agents to either regulate or eradicate cancer cells. These substances usually, but not always, cause cytotoxicity by inducing apoptosis, a non-immunogenic kind of cell death [56]. Chemotherapy medicines come in a wide variety and can be used either as a stand-alone treatment or in conjunction with other medications. Popular chemotherapeutic drug paclitaxel is efficient against a variety of malignancies, such as multiple myelomas, breast and ovarian cancers, colon cancer, small-cell and non-small-cell lung cancer, melanoma, and head and neck cancer [57].

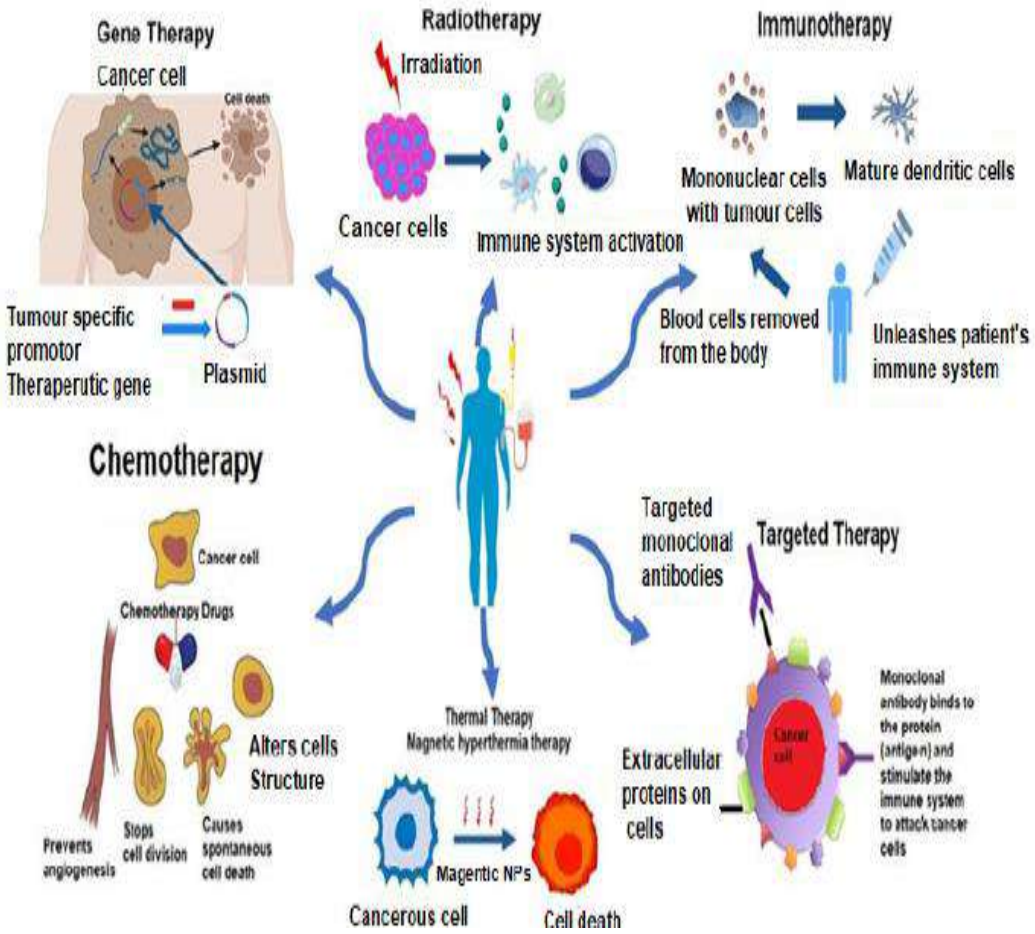

Figure 2 Traditional Oncology Therapy

Table 1: Enumeration of cancer therapy options

Sr. No.	Oncology therapy	Details	References
1.	Radiation therapy		
	Individualization of radiation treatment according to hypoxia indicators	increases the blood's oxygen content by breathing in large amounts of oxygen both before and during radiation exposure in order to radiosensitize or kill hypoxic cells with oxygen-mimicking medications.	52
	Personalized radiotherapy based on FDG-PET	On a positron emission tomography (PET) scan, the intensity of fludeoxyglucose (18F-FDG) indicates the extent of glucose absorption by malignant cells that are actively dividing.	

	DNA repair marker	Histone protein gH2AX, which is present upon the production of double-strand breaks, is one of the best indicators for tumor radioresponse of DNA double-strand breaks.	
2.	Gene Therapy		
	Genedicine (Adenovirus Ad5RSV-P53, Recombinant Human P53))	Gendicine is a non-replicative vector in which the p53 cDNA gene is inserted in place of the E1 gene. Tumor cell p53 expression promotes the anticancer impact by starting the apoptotic pathway and preventing damaged DNA repair.	53
	Recombinant oncolytic ad5 (rAd5-H101)	Oncorine is an ad5 virus with a deletion in the E1B 55K gene; host cell p53 gene inactivation is necessary for wild-type to stop the activation of the apoptotic pathway. It has been demonstrated to cure resistant nasopharyngeal cancer.	
3.	Thermotherapy		
	Alternatives for Thermal Ablation	It employs temperatures ranging from 55 to 100 degrees Celsius as an external stimulation to overheat the tumor, causing destruction and its elimination. It can treat a variety of cancers, including prostate, kidney, liver, lung, and rectum.	
	Ablation using Microwaves (MWA)	It generates a direct hyperthermic injury by heating the chosen location with an electro-magnetic (EM) signal. Starting at 915 MHz, the frequency range extends to 2.45 GHz. It produces coagulation necrosis by directing an ultrasonic (US) beam toward overheating a specific tissue. It kills tumors with extreme precision and treats some of the	

	High Focused Ultrasound Intensity (HIFU) Cryoablation	associated health problems. Low temperatures, between 30 and 40 degrees Celsius, are used in cryotherapy to produce a freezing zone and cause the targeted region to be destroyed. To chill the tissue to 100 °C, argon or nitrogen is supplied to the probe tip.	
4.	Chemotherapy		
	Dacarbazine, temozolomide Ethyleneimines: thiotepa, mechlorethamine, chlorambucil, cyclophosphamide streptozocin, Daunorubin, Doxorubixin Epirubicin, Actinomycin-D Bleomycin, Mitomycin-C 5-fluorouracil (5-FU) 6-mercaptopurine (6-MP) Capecitabine, Cladribine Cloafarabine, Cytarabine Floxuridine, Fludarabine Gemicitabine, Hydroxyurea Methotrexate, Pemetrexed Pentostatin, Thioguanine Topotecan, Irinotecan Etoposide, Teniposide	At different stages of the cell cycle, damage DNA. G1 phase, S phase, G2 phase, M phase, and GO phase (resting phase). Hodgkin's illness, lymphoma, ovarian cancer, breast cancer, and lung cancer, sarcoma, many, and myeloma. disrupt the enzymes necessary for DNA replication at every stage of the cell cycle. digestive tract cancer, ovarian cancer, breast cancer, leukemia, and other cancers interfere with the synthesis of RNA and DNA in cells. gastrointestinal tracts, ovarian, breast, and other malignancies. interfere with topoisomerase by preventing DNA strand breaks during replication, such as topoisomerase inhibitor I and II. colon, ovarian, lung, and other gastrointestinal malignancies.	54
5.	Targeted Therapy		
	HER2 inhibitors: Herceptin, Herceptin Hylecta, Margenza, Perjeta	In order to stop the activity, HER2 inhibitors bind to the HER2 cell surface receptor.	55

	SMALL MOLECULES Tyrosine Kinase Inhibitors: Imatinib, gefitinib, erlotinib, sorafenib, sunitinib, dasatinib diphosphate-ribose polymerase inhibitors (PARP): Olaparib, niraparib, rucaparib, talazoparib.	Tyrosine kinase inhibitors function by preventing tyrosine from acting on receptors. Enzymes called kinases aid in the growth signaling of cancer cells. PARP protein helps to repair damaged DNA in cancer cells. In order to prevent PARP proteins from mending DNA in cancer cells, PARP inhibitors work.	

Immunotherapy:

The immune system is crucial in controlling the development of tumors. While an immune response unique to the tumor may be able to limit its growth, certain forms of inflammatory reactions have a tendency to promote tumor growth [58]. Immuno-oncology, another name for cancer immunotherapy, is a type of cancer treatment that stimulates the patient's immune system to fight, manage, and eradicate cancer. Immunotherapy aims to overcome the processes by which tumors stifle the immune response in order to strengthen or restore the immune system's capacity to recognize and eliminate cancer cells [58]. Adoptive cell immunotherapy, immune checkpoint inhibitors, cytokines, cancer vaccines, oncolytic viruses, and targeted antibodies are examples of immunotherapeutic techniques. The conventional method of immunotherapy involves adoptive cell transfer, cytokines, and cancer vaccines to boost the frequency of tumor-specific T lymphocytes. Using interferons and Toll-like receptor agonists to cause inflammation and innate immune activation in the tumor microenvironment is an additional strategy. Targeting several immune cell activation checkpoints, such as cytotoxic T lymphocyte-associated protein 4 (CTLA4), monoclonal antibodies (mAbs), regulatory T-cells, and programmed cell death protein 1 (PD1), is the most efficient way to initiate antitumor immune responses [59]. The majority of solid tumors are characterized by vascular anomalies, which raise proangiogenic factors such vascular endothelial growth factors (VEGF) and angiopoietin 2. Utilizing medications that target these components sensibly aids in boosting the immune system's reaction and the aberrant vasculature to normal. They cause immune effector cells to infiltrate cancer cells and change the immunosuppressive tumor microenvironment into an immune-stimulating one. Immune responses and vascular normalization are mutually controlled. Thus, combining immunotherapies with antiangiogenic medicines may improve immunotherapy's potential and lower the chance of immune-related adverse effects [60].

Radiotherapy:

Conventionally, radiotherapy is used to treat cancer together with surgery or chemotherapy. It's the most significant non-surgical cancer therapy option available. Combining radiotherapy and immunotherapy, known as radiation-immunotherapy, has showed promising outcomes in the treatment of cancer [61,62]. In addition to curing cancer, radiation therapy can lessen the symptoms of incurable cancer. It can be used alone or in combination with other therapies. Optimizing radiation to cancer cells while limiting damage to nearby healthy cells is a major challenge [63]. Technological developments in radiology, including image-guided radiation therapy,

stereotactic body radiotherapy, and intensity-modulated radiation therapy, have contributed to the preservation of a therapeutic-toxin balance.

By precisely delivering radiation to the targeted tumor cells, these methods lessen the harm done to the nearby healthy cells [64].

Targeted Therapy:

Drugs used in targeted treatment are made specifically to target cancer cells, sparing surrounding normal cells. They are divided into two categories: tiny and big molecules. Since small-molecule medications may fit into cancer cells, they function via identifying and inhibiting a certain chemical present in the cell to kill the cancerous cell. Imatinib, for instance, blocks tumor-activating impulses to treat chronic myelogenous leukemia and other malignancies [65,66]. Big molecules, like certain mAbs, are too large to fit inside a cell. They function by targeting and eliminating proteins or enzymes on the cell's surface and preventing interactions that might otherwise promote tumor development between receptors and ligands. Examples are cetuximab for lung, head, and neck malignancies, trastuzumab for breast tumors, and alemtuzumab for persistent leukemias [67,68]. Antibody-targeted therapy: It has been demonstrated that one of the most effective therapeutic approaches for both solid tumors and hematologic malignancies is mAbs-based treatment. mAbs function in a variety of ways, including antibody-dependent cellular phagocytosis, apoptosis, complement-dependent cytotoxicity, antibody-dependent cellular cytotoxicity, and signal transduction blocking. These mAbs are being loaded with cytotoxic medicines in an attempt to optimize their effectiveness and reduce their toxicity Known as antibody-drug conjugates (ADC), these molecules are thought to be tumor-specific. These ADCs combine the powerful cytotoxic potential of small-molecule medicines with the specificity and advantageous pharmacokinetics of mAbs. The following list of examples of targeted cancer therapies is arranged according to how they work [69-72]. High-precision targeted administration of anticancer medications is made possible by molecular targeted treatment. Typically, a smallmolecule medication that targets markers inside the cell or an antibody that binds to certain targets on the cell surface is utilized as the therapeutic medicine. Growth factor receptors, cell surface antigens, and signal transduction pathways that control angiogenesis, metastasis, and cell death are all impacted by molecularly targeted therapeutics [72].

Thermal Therapy:

In thermal ablation of cancer, neoplastic tissues are killed by applying heat (hyperthermia) or cold (hypothermia). Cell necrosis has been seen to occur at temperatures more than 60 °C or lower than 40 °C. It follows that long-term exposure to temperatures between 41 ~C and 55 ~C is efficient in killing tumor cells. There are five therapeutic strategies that can be used in thermal therapy: cryotherapy (~508 ~C for >10 min), moderate cooling (0–108 ~C for 10 min), low-temperature hyperthermia (39–418 ~C for up to 72 h), moderate-temperature hyperthermia (42–458 ~C for 15–60 min), and high-temperature thermal ablation therapy (>508 ~C for >4-6 min). These therapeutic approaches can affect the tissues through an increase or decrease in oxygenation, blood perfusion, and cellular metabolisms, which can lead to protein denaturation, tissue necrosis, and cellular coagulation [73]. Radiation sensitivity of tumor cells is increased with hyperthermia. Numerous clinical trials have demonstrated the considerable reduction in tumor size of several cancer types, such as melanoma, sarcoma, lung, breast, esophageal, brain, and others, when paired with hyperthermia and radiation and/or chemotherapy. It was shown that the combination of chemotherapy with hyperthermia allowed

medications to enter tumor tissues more deeply, improving the effectiveness of treatment [74-76].

Gene Therapy:

In order to stop the proliferation of malignant cells, genetic elements like DNA or RNA are injected into the cells using gene therapy. This can be accomplished by suppressing tumor angiogenesis, blocking the expression of the mutant tumor suppressor gene, and blocking the appearance of an oncogene [77]. Retroviruses and adenoviruses are the most significant viral vectors; non-viral vectors are particle-based, chemical-based, and based on naked DNA. Viral vectors are useful for gene delivery and cell transfection, but their use is restricted because of their immunogenicity and toxicity; non-viral vectors are less toxic, but they need delivery vehicles to infiltrate various cell types [71].

CONCLUSION:

This review delved into the mechanisms underlying specific types of cancer, along with potential avenues for prevention and treatment. It suggested incorporating certain anticancer foods into the diet, which have shown effectiveness in controlling cell growth and enhancing survival rates across different cancer types. The extensive research on the impact of nutrition on cancer has made it evident that what we eat plays a significant role in cancer development. Diet is just one aspect of lifestyle that affects cancer risk; others include alcohol consumption, smoking, physical activity levels, and obesity.Phytonutrients present in fruits and vegetables work together synergistically to reduce the risk of cancer through various mechanisms such as hormone regulation, suppressing carcinogenic pathways, and reducing inflammation. The ideal dietary approach appears to be a well-rounded one, emphasizing lean proteins, whole grains, fruits, and vegetables, while limiting intake of red meat, sugar, coffee, and alcohol. It's important to note that there's no scientific basis for claiming that a specific diet can cure or treat cancer. However, there's substantial evidence indicating that adopting a healthy diet and lifestyle can lower the risk of developing certain types of cancer.

REFERENCES:

1. World Health Organization Cancer. Available online: https://www.who.int/news-room/fact-sheets/detail/cancer (accessed on 8 August 2021).
2. Anand, P.; Kunnumakkara, A.B.; Sundaram, C.; Harikumar, K.B.; Tharakan, S.T.; Lai, O.S.; Sung, B.; Aggarwal, B.B. Cancer is a preventable disease that requires major lifestyle changes. Pharm. Res. 2008, 25, 2097–2116.
3. Santarelli, R.L.; Pierre, F.; Corpet, D.E. Processed meat and colorectal cancer: A review of epidemiologic and experimental evidence. Nutr. Cancer 2008, 60, 131–144
4. Rather, I.A.; Koh, W.Y.; Paek, W.K.; Lim, J. The Sources of Chemical Contaminants in Food and Their Health Implications. Front. Pharmacol. 2017, 8, 830
5. Liskova, A.; Samec, M.; Koklesova, L.; Brockmueller, A.; Zhai, K.; Abdellatif, B.; Siddiqui, M.; Biringer, K.; Kudela, E.; Pec, M.; et al. Flavonoids as an effective sensitizer for anti-cancer therapy: Insights into multi-faceted mechanisms and applicability towards individualized patient profiles. EPMA J. 2021, 17, 1–22
6. Gupta, C.; Prakash, D. Phytonutrients as Therapeutic Agents. J. Complement. Integr. Med. 2014, 11, 151–169
7. Moorthy, B.; Chu, C.; Carlin, D.J. Polycyclic aromatic hydrocarbons: From metabolism to lung cancer. Toxicol. Sci. 2015, 145, 5–15
8. Rivlin, N.; Brosh, R.; Oren, M.; Rotter, V. Mutations in the p53 Tumor Suppressor Gene: Important Milestones at the Various Steps of Tumorigenesis. Genes Cancer 2011, 2, 466–474.

9. Nguyen, T.T.; Ung, T.T.; Kim, N.H.; Jung, Y. Do Role of bile acids in colon carcinogenesis. World J. Clin. cases 2018, 6, 577–588
10. Chariyarangsitham, W.; Krungchanuchat, S.; Khuemjun, P.; Pilapong, C. Effect of advanced oxidation and amino acid addition on antioxidant capability, iron chelating property and anti-cancer activity of tannic acid. Arab. J. Chem. 2021, 14, 103312.
11. Waladkhani, A.R.; Clemens, M.R. Effect of dietary phytochemicals on cancer development (review). Int. J. Mol. Med. 1998,1, 747–800.
12. Garcia, M.; Jenber, A.; Ward, E.; Center, M.; Hao, Y.; Siegel, R.; Thun, M. Global Cancer Facts & Figures 2007; American Cancer Society: Atlanta, GA, USA, 2007.
13. World Cancer Research Fund; American Institute for Cancer Research. Diet, Nutrition, Physical Activity and Cancer: A Global Perspective A Summary of the Third Expert Report; Continuous Update Project Expert Report;World Cancer Research Fund: London, UK; American Institute for Cancer Research: Boston, MA, USA, 2018.
14. Key, T.J.; Schatzkin, A.;Willett,W.C.; Allen, N.E.; Spencer, E.A.; Travis, R.C. Diet, nutrition and the prevention of cancer. Public Health Nutr. 2004, 7, 187–200.
15. Pan, P.; Yu, J.;Wang, L.S. Colon Cancer: What We Eat. Surg. Oncol. Clin. North Am. 2018, 27, 243–267.
16. Jones, P.; Cade, J.E.; Evans, C.E.L.; Hancock, N.; Greenwood, D.C. The Mediterranean diet and risk of colorectal cancer in the UK Women's Cohort Study. Int. J. Epidemiol. 2017, 46, 1786–1796.
17. Yusof, A.S.; Md Isa, Z.; Shah, S.A. Dietary patterns and risk of colorectal cancer: Asystematic review of cohort studies (2000-2011). Asian Pacific J. Cancer Prev. 2012, 13, 4713–4717.
18. Haslam, A.; Robb, S.W.; Hébert, J.R.; Huang, H.; Ebell, M.H. Association between dietary pattern scores and the prevalence of colorectal adenoma considering population subgroups. Nutr. Diet. 2018, 75, 167–175.
19. Mehta, R.S.; Song, M.; Nishihara, R.; Drew, D.A.; Wu, K.; Qian, Z.R.; Fung, T.T.; Hamada, T.; Masugi, Y.; da Silva, A.; et al.Dietary Patterns and Risk of Colorectal Cancer: Analysis by Tumor Location and Molecular Subtypes. Gastroenterology 2017, 152, 1944–1953.e1.
20. Castelló, A.; Amiano, P.; Fernández de Larrea, N.; Martín, V.; Alonso, M.H.; Castaño-Vinyals, G.; Pérez-Gómez, B.;Olmedo-Requena, R.; Guevara, M.; Fernandez-Tardon, G.; et al. Low adherence to the western and high adherence to the mediterranean dietary patterns could prevent colorectal cancer. Eur. J. Nutr. 2018, 58, 1495–1505.
21. Kim, M.; Park, K. Dietary Fat Intake and Risk of Colorectal Cancer: A Systematic Review and Meta-Analysis of Prospective Studies. Nutrients 2018, 10, 1963.
22. Hodge, A.M.;Williamson, E.J.; Bassett, J.K.; MacInnis, R.J.; Giles, G.G.; English, D.R. Dietary and biomarker estimates of fatty acids and risk of colorectal cancer. Int. J. Cancer 2015, 137, 1224–1234.
23. Bernstein, C.; Holubec, H.; Bhattacharyya, A.K.; Nguyen, H.; Payne, C.M.; Zaitlin, B.; Bernstein, H. Carcinogenicity of deoxycholate, a secondary bile acid. Arch. Toxicol. 2011, 85, 863–871.
24. Franceschi, S.; Dal Maso, L.; Augustin, L.; Negri, E.; Parpinel, M.; Boyle, P.; Jenkins, D.J.A.; Lavecchia, C. Dietary glycemic load and colorectal cancer risk. Ann. Oncol. 2001, 12, 173–178.
25. Van Blarigan, E.L.; Fuchs, C.S.; Niedzwiecki, D.; Ye, X.; Zhang, S.; Song, M.; Saltz, L.B.;

Mayer, R.J.; Mowat, R.B.; Whittom, R.; et al.Marine !-3 Polyunsaturated Fatty Acid and Fish Intake after Colon Cancer Diagnosis and Survival: CALGB 89803 (Alliance).Cancer Epidemiol. Prev. Biomark. 2018, 27, 438–445.

26. Fadelu, T.; Zhang, S.; Niedzwiecki, D.; Ye, X.; Saltz, L.B.; Mayer, R.J.; Mowat, R.B.; Whittom, R.; Hantel, A.; Benson, A.B.; et al.Nut Consumption and Survival in Patients with Stage III Colon Cancer: Results from CALGB 89803 (Alliance). J. Clin. Oncol. 2018, 36, 1112–1120.
27. Miller, G.D.; Jarvis, J.K.; McBean, L.D. Handbook of Dairy Foods and Nutrition, 2nd ed.; CRC Press: Rosemont, IL, USA, 2000, ISBN 0849387310.
28. Brinton, L.A.; Gaudet, M.M.; Gierach, G.L. Breast cancer. In Cancer Epidemiology and Prevention; Michael Thun, M., Linet, M.S.,Cerhan, J.R., Haiman, C.A., Schottenfeld, D., Eds.; Oxford University Press: New York, NY, USA, 2017; pp. 861–888.
29. Dong, J.Y.; Qin, L.Q. Soy isoflavones consumption and risk of breast cancer incidence or recurrence: A meta-analysis of prospective studies. Breast Cancer Res. Treat. 2010, 125, 315–323.
30. Smith, R.A.; Andrews, K.S.; Brooks, D.; Fedewa, S.A.; Manassaram-Baptiste, D.; Saslow, D.; Brawley, O.W.; Wender, R.C. Cancer screening in the United States, 2017: A review of current American Cancer Society guidelines and current issues in cancer screening. CA Cancer J. Clin. 2017, 67, 100–121.
31. Kwan, M.L.; Garren, B.; Nielsen, M.E.; Tang, L. Lifestyle and nutritional modifiable factors in the prevention and treatment of bladder cancer. Urol. Oncol. Semin. Orig. Investig. 2019, 37, 380–386.
32. Philips, B.J.; Coyle, C.H.; Morrisroe, S.N.; Chancellor, M.B.; Yoshimura, N. Induction of apoptosis in human bladder cancer cells by green tea catechins. Biomed. Res. 2009, 30, 207–215.
33. Meneses-Gutiérrez, C.L.; Hernández-Damián, J.; Pedraza-Chaverri, J.; Guerrero-Legarreta, I.; Téllez, D.I.; Jaramillo-Flores, M.E. Antioxidant Capacity and Cytotoxic Effects of Catechins and Resveratrol Oligomers Produced by Enzymatic Oxidation against T24 Human Urinary Bladder Cancer Cells. Antioxidants 2019, 8, 214.
34. Silva, M.M.; Santos, M.R.; Caroço, G.; Rocha, R.; Justino, G.; Mira, L. Structure-antioxidant Activity Relationships of Flavonoids: A Re-examination. Free Radic. Res. 2002, 36, 1219–1227.
35. Xue, Y.Q.; Di, J.M.; Luo, Y.; Cheng, K.J.; Wei, X.; Shi, Z. Resveratrol Oligomers for the Prevention and Treatment of Cancers. Oxid. Med. Cell. Longev. 2014, 2014, 765832.
36. Wu, J.; Yu, Y.; Huang, L.; Li, Z.; Guo, P.; Xu, Y.W. Dairy Product Consumption and Bladder Cancer Risk: A Meta-Analysis. Nutr.Cancer 2020, 72, 377–385.
37. Buendia Jimenez, I.; Richardot, P.; Picard, P.; Lepicard, E.M.; De Meo, M.; Talaska, G. Effect of Increased Water Intake on Urinary DNA Adduct Levels and Mutagenicity in Smokers: A Randomized Study. Dis. Markers 2015, 2015, 478150.
38. Wang, J.;Wu, X.; Kamat, A.; Barton Grossman, H.; Dinney, C.P.; Lin, J. Fluid intake, genetic variants of UDP-glucuronosyltransferases,and bladder cancer risk. Br. J. Cancer 2013, 108, 2372–2380.
39. Bai, Y.; Yuan, H.; Li, J.; Tang, Y.; Pu, C.; Han, P. Relationship between bladder cancer and total fluid intake: A meta-analysis of epidemiological evidence. World J. Surg. Oncol. 2014, 12, 223.

40. Ali, A.; Waly, M.I.; Devarajan, S. Chapter 11 Impact of Processing Meat on the Formation of Heterocyclic Amines and Risk of Cancer. In Biogenic Amines in Food: Analysis, Occurrence and Toxicity; The Royal Society of Chemistry: London, UK, 2020; pp. 187–211; ISBN 978-1-78801-436-6.
41. Siegel, R.L.; Miller, K.D.; Jemal, A. Cancer statistics, 2018. CA. Cancer J. Clin. 2018, 68, 7–30.
42. Winters, S.; Martin, C.; Murphy, D.; Shokar, N.K. Breast Cancer Epidemiology, Prevention, and Screening. Prog. Mol. Biol. Transl. Sci. 2017, 151, 1–32.
43. Van Ryswyk, K.; Villeneuve, P.J.; Johnson, K.C.; The Canadian Cancer Registries Epidemiology Research Group. Dietary patterns and the risk of female breast cancer among participants of the Canadian National Enhanced Cancer Surveillance System. Can. J. Public Health Rev. Can. St. Publique 2016, 107, e49–e55.
44. Maino Vieytes, C.A.; Taha, H.M.; Burton-Obanla, A.A.; Douglas, K.G.; Arthur, A.E. Carbohydrate Nutrition and the Risk of Cancer. Curr. Nutr. Rep. 2019, 8, 230–239.
45. Vigneri, R.; Goldfine, I.D.; Frittitta, L. Insulin, insulin receptors, and cancer. J. Endocrinol. Invest. 2016, 39, 1365–1376.
46. Belfiore, A.; Malaguarnera, R. Insulin receptor and cancer. Endocr. Relat. Cancer 2011, 18, R125–R147.
47. Yao, Y.; Suo, T.; Andersson, R.; Cao, Y.; Wang, C.; Lu, J.; Chui, E. Dietary fibre for the prevention of recurrent colorectal adenomas and carcinomas. Cochrane Database Syst. Rev. 2017, 1.
48. Ayala, A.; Muñoz, M.F.; Argüelles, S. Lipid peroxidation: Production, metabolism, and signaling mechanisms of malondialdehyde and 4-hydroxy-2-nonenal. Oxid. Med. Cell. Longev. 2014, 2014, 360438.
49. Liu, Y.; Nguyen, N.; Colditz, G.A. Links between Alcohol Consumption and Breast Cancer: A Look at the Evidence. Women's Health 2015, 11, 65–77.
50. Crippa, A.; Larsson, S.C.; Discacciati, A.; Wolk, A.; Orsini, N. Red and processed meat consumption and risk of bladder cancer: A dose-response meta-analysis of epidemiological studies. Eur. J. Nutr. 2018, 57, 689–701.
51. Feng, S.S.; Chien, S. Chemotherapeutic engineering: Application and further development of chemical engineering principles for chemotherapy of cancer and other diseases. Chem. Eng. Sci. 2003, 58, 4087–4114.
52. Aminuddin, A.; Ng, P.Y. Promising Druggable Target in Head and Neck Squamous Cell Carcinoma: Wnt Signaling. Front. Pharmacol. 2016, 7, 244.
53. Ong, C.P.; Lee, W.L.; Tang, Y.Q.; Yap, W.H. Honokiol: A Review of Its Anticancer Potential and Mechanisms. Cancers 2020, 12, 48.
54. Li, X.; Yao, Z.; Jiang, X.; Sun, J.; Ran, G.; Yang, X.; Zhao, Y.; Yan, Y.; Chen, Z.; Tian, L.; et al. Bioactive compounds from Cudrania tricuspidata: A natural anticancer source. Crit. Rev. Food Sci. Nutr. 2020, 60, 494–514.
55. Islam, R.; Lam, K.W. Recent progress in small molecule agents for the targeted therapy of triple-negative breast cancer. Eur. J. Med. Chem. 2020, 207, 112812.
56. Zitvogel, L.; Apetoh, L.; Ghiringhelli, F.; Kroemer, G. Immunological aspects of cancer chemotherapy. Nat. Rev. Immunol. 2008, 8, 59–73.
57. Feng, S.S.; Chien, S. Chemotherapeutic engineering: Application and further development of chemical engineering principles for chemotherapy of cancer and

other diseases. Chem. Eng. Sci. 2003, 58, 4087–4114.
58. Mellman, I.; Coukos, G.; Dranoff, G. Cancer immunotherapy comes of age. Nature 2011, 480, 480–489.
59. Farkona, S.; Diamandis, E.P.; Blasutig, I.M. Cancer immunotherapy: The beginning of the end of cancer? BMC Med. 2016, 14, 73.
60. Fukumura, D.; Kloepper, J.; Amoozgar, Z.; Duda, D.G.; Jain, R.K. Enhancing cancer immunotherapy using antiangiogenics: Opportunities and challenges. Nat. Rev. Clin. Oncol. 2018, 15, 325–340.
61. Zitvogel, L.; Apetoh, L.; Ghiringhelli, F.; Kroemer, G. Immunological aspects of cancer chemotherapy. Nat. Rev. Immunol. 2008, 8, 59–73.
62. Chen, H.H.W.; Kuo, M.T. Improving radiotherapy in cancer treatment: Promises and challenges. Oncotarget 2017, 8, 62742–62758.
63. Frey, B.; Rubner, Y.; Kulzer, L.;Werthmöller, N.;Weiss, E.M.; Fietkau, R.; Gaipl, U.S. Antitumor immune responses induced by ionizing irradiation and further immune stimulation. Cancer Immunol. Immunother. 2014, 63, 29–36.
64. Baumann, M.; Krause, M.; Overgaard, J.; Debus, J.; Bentzen, S.M.; Daartz, J.; Richter, C.; Zips, D.; Bortfeld, T. Radiation oncology in the era of precision medicine. Nat. Rev. Cancer 2016, 16, 234–249.
65. Seidel, J.A.; Otsuka, A.; Kabashima, K. Anti-PD-1 and Anti-CTLA-4 Therapies in Cancer: Mechanisms of Action, Efficacy, and Limitations. Front. Oncol. 2018, 8, 86.
66. Falzone, L.; Salomone, S.; Libra, M. Evolution of Cancer Pharmacological Treatments at the Turn of the Third Millennium. Front. Pharmacol. 2018, 9, 1300.
67. Baudino, T.A. Targeted Cancer Therapy: The Next Generation of Cancer Treatment. Curr. Drug Discov. Technol. 2015, 12, 3–20.
68. Lee, Y.T.; Tan, Y.J.; Oon, C.E. Molecular targeted therapy: Treating cancer with specificity. Eur. J. Pharmacol. 2018, 834, 188–196.
69. Lee, Y.T.; Tan, Y.J.; Oon, C.E. Molecular targeted therapy: Treating cancer with specificity. Eur. J. Pharmacol. 2018, 834, 188–196.
70. Cerny, J.; Raffel, G. DeVita, Hellman, and Rosenberg's Cancer: Principles & Practice of Oncology, 9th ed.; LippincottWilliams &Wilkins:Philadelphia, PA, USA, 2011; ISBN 10: 1-4511-0545-2.
71. Bidram, E.; Esmaeili, Y.; Ranji-Burachaloo, H.; Al-Zaubai, N.; Zarrabi, A.; Stewart, A.; Dunstan, D.E. A concise review on cancer treatment methods and delivery systems. J. Drug Deliv. Sci. Technol. 2019, 54, 101350.
72. Ke, X.; Shen, L. Molecular targeted therapy of cancer: The progress and future prospect. Front. Lab. Med. 2017, 1, 69–75.
73. Stauffer, P.R. Evolving technology for thermal therapy of cancer. Int. J. Hyperth. 2005, 21, 731–744.
74. Mondal, S.; Sur, A.; Kanoria, M. A graded spherical tissue under thermal therapy: The treatment of cancer cells. Waves Random Complex Media 2020, 32, 488–507.
75. Lee, C.T.; Mace, T.; Repasky, E.A. Hypoxia-driven immunosuppression: A new reason to use thermal therapy in the treatment of cancer? Int. J. Hyperth. 2010, 26, 232–246.
76. Pucci, C.; Martinelli, C.; Ciofani, G. Innovative approaches for cancer treatment: Current perspectives and new challenges. Ecancermedicalscience 2019, 13, 961–987.
77. Belete, T.M. The Current Status of Gene Therapy for the Treatment of Cancer. Biol. Targets Ther. 2021, 15, 67–77.

HOW TO CITE: Shraddha Srivastava, Priyanka sonker, An Overview Of The Most Recent Cancer Treatments, As Well As The Origins And Mechanisms Of Several Cancer Types, Int. J. of Pharm. Sci., 2024, Vol 2, Issue 6, 347-362. https://doi.org/10.5281/zenodo.12589301