World Journal of Pharmaceutical and Life Sciences

www.wjpls.org

Impact Factor: 7.409 Coden USA: WJPLA7

GLOBAL, COMPEHENSIVE, EVIDENCE-BAASED AND MERCHANDISE VIEWPOINTS OF CANCER INCLUDING, MECHANISM, THERAPEUTICS, CHARACTERISTICS, PERSONALITY, OCCUPATION, AND MORPHOLOGY; WITH A HOPE OF WORLD-WIDE HEALTH PROMOTION

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DOI: https://doi.org/10.5281/zenodo.17813624

How to cite this Article: Eiichi Akaho*. (2025). Global, Compehensive, Evidence-Baased And Merchandise Viewpoints Of Cancer Including, Mechanism, Therapeutics, Characteristics, Personality, Occupation, And Morphology; With A Hope Of World-Wide Health Promotion. World Journal of Pharmaceutical and Life Science, 11(12), 291–318.

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Article Received on 06/11/2025 **Article Revised on 27/11/2025** **Article Published on 01/12/2025**

ABSTRACT

Unfortunately, cancer in general is the most awful ailment with high morbidity and mortality. It appears that this trait and phenomenon will be propagating for many years to come. In order to surmount this lethal ailment, it is essential to take a look at it from a standpoint of global and merchandise viewpoints including major aspects such as specification, mechanism, therapeutics, characteristics, and monography. PubMed, Google Scholar, Google Advanced Search are observed and searched. Accumulate evidence-based pieces of information on cancer, BioRender, Google Graphics, and PowerPoint are used to create schematic diagram of cancer. Lycorine, decorin, cinnamaldehyde, unsolid acid, young san, forsythoside A, polysaccharide, protopanaxadiol ginsenoside, Claudin 8, and resveratrol are worth investigating substances for the cancer treatment. Risks of lip cancer and melanoma may be attributed to sun exposure. The reduced alcohol and smoking are attributed to the lessened risk of cancers such as lung cancer, liver cancer, prostate cancer, and larynx cancer. As far as the statically elevated cancer risk for men worker is concerned, waiters, beverage workers, cooks, stewards, and hairdressers show higher SIR(standardized incidence ratio). More severe depression symptoms were associated with reduced likelihood of breast cancer. The immune and nervus systems cooperate to sustain intestinal homeostasis which is conducive to the development of probiotics. Xenoestrogens affects breast cancer cell morphology and motility. The CAR T (Chimeric Antigen Receptor-T) cell in the blood stream kills cancer cell.

KEYWORDS: Cancer, mechanism, characteristics, treatment, immunotherapy, comprehensive viewpoint, chemotherapy, morphology.

INTRODUCTION

Hepatocellular carcinoma (HCC) represents an arduous global health challenge due to its prevalence, marked by high mortality and morbidity.[1] In order to surmount this lethal ailment, I am going to take a look at it from a standpoint of global and merchandise viewpoints including specification, mechanism, therapeutics, environmental, characteristics, personality, occupation, morphology, and so on. At the present time, cancer is wide-spread awful disease, and regrettably it will be so for the many years to come. The difficulty for the cancer rebellion lies in the fact that one suffers from cancer from many angles including genetic, infectious, environmentals, emotionals, and the like. Normal cells follow a set of organized metabolic programs, while cancer cells in our body replicate in an orderly fashion through a process called mitosis. When these cells are thronged during growth, they stop growing through the inhibition caused by mutual contact of cells. In case a defect or mutation occurs in a cell, it is removed by the cellular machinery through programed cell death called apoptosis.[2] Approximately 38 trillion microorganisms have been discovered in human microbiota such as fungi, bacteria, and virus. The number of them roughly

resembles that of human cells.[3] The eukaryote organelles, mitochondria, and the plant organelles, and chloroplasts, have descended, respectively, from proteobacteria and cyanobacteria. These endosymbionts of eukaryote cells act as auxiliary DNA-containing compartments. During the course of evolution, the continual transfer of organelle DNA to the nucleus takes place. These nuclear copies of DNA are called nuclear mtDNA. (NUMTs).[4] Transferred chloroplast DNA fragments are called NUPTs for nuclear chloroplast or plastid DNA.[5]

The importance of the microenvironment is underlined by recent studies showing that supposedly 'normal' stromal cells can influence epithelial tumor-cell behavior in reconstitution experiments, and by the detection of specific somatic genetic changes in the stromal component of some tumors. Tumor architecture mimics many of the features of normal tissues, with a cellular hierarchy that regulates the balance between cell renewal and cell death. Somatic stem cells are responsible for the development and homeostasis of a variety of bodily tissues and organs.[6] Chromosome translocations generate chimeric fusion genes, which provide stable sensitive markers that are unique for each patient's leukemic clone and can be used to track its origins and response to treatment.[7]

Cancer development after middle age usually exhibits genomic instability, multiple mutations and extreme chromosomal instability (CIN) or microsatellite instability (MSI), while pediatric tumors usually develop as a result of specific chromosomal translocations and epigenetic aberrations. MSI occurs in cells that do not have adequate mismatch repair systems[8-10], and CIN occurs in the presence of other types of repair deficiencies. The hereditary versions of myelodysplastic syndrome[11], breast and ovarian cancers[12-15], and skin cancers[16-18] are examples of CIN. CIN can even occur in normal senescent cells.[12-15]

The development of genomic instability is accompanied by mutations that lead to cellular immortalization and transformation. Cancer transpires when cancer-initiating cells (CICs), or cancer stem cell develop as a result of these mutations. Although CIC characteristics are increasingly being well elucidated, a little is known how CICs develop.[21]

Unlike pediatric tumors, which usually develop as a result of very specific chromosomal translocations[22,23] and epigenetic aberrations[24,25], cancers that become more common around the age of 40 years exhibit extreme chromosomal instability (CIN) or microsatellite instability (MSI).[26-29] MSI occurs in cells that do not have adequate mismatch repair systems[30-32], and CIN occurs in the presence of other types of repair deficiencies. The hereditary versions of myelodysplastic syndrome[32], breast and ovarian cancers[33-36], and skin cancers[37-39] are examples of CIN. CIN can even occur in normal senescent cells.[40,41]

CRISP/Cas9 (Clustered regularly interspaced short palindromic repeats associated protein 9 has shown as a puissant gene editing method with vast potential including its application in cancer research and therapy. Cancer is a genome-related disease involving gene alterations and epigenome dysregulation. Understanding the impact of genetic changes, cellular adaptations, and microenvironment changes on cancer treatment is crucial for cancer research technology.[39]

MATERIALS AND METHODS

A) PubMed Advanced Search, Google Scholar, Google Advanced Search are observed and searched to accumulate evidence-based pieces of information on cancer.

- Example of PubMed Search

Search logic

a) {"Cancer" and "mechanism" appear in title with the publication date "January 1, 2024 to present}
b) Publication date is assigned from January 1, 2024 to present.

SEARCH RESULT

((cancer[Title]) AND (mechanism[Title])) AND (("2024/01/01"[Date - Publication]: "3000"[Date - Publication])); 1039 articles were hit and obtained.

B) BioRender, Smart Servier, Google Graphics, and PowerPoint are used to create schematic diagram of cancer.

RESUTS AND DISCUSSION

1. Comprehensive Viewpoint Of Cancer

Cancer involves a wide range of aspects. Those consist of molecular domains, DNA, RNA, single nucleotide, polymorphism, gene mutations, transcription, translation, noncoding nucleic acid, metabolite, microorganism and so on.[23] These technological and research progresses led us to the discovery of additional cancer traits by broadening our cancer knowledge bases.[24] Consequently, We intend to observe and investigate cancer-related issues from muti-omics and multangular viewpoints.

1.1 Characteristics and constancy attributed to various cancers

Metabolic reprograming is one of the pivotal characteristics of cancer. Additionally, this metabatic feature is an essential process leading to tumor development, malignant proliferation, and chemotherapeutic resistance. We have been observed various therapeutic drugs targeting metabolic reaction enzymes, special metabolic processes, and transport receptors.[27] Cancer cells maintain redox homeostasis and promote their proliferation, migration and invasion through metabolic reprograming. The preferential use of

large amount of glucose to generate lactate by tumor cell even under aerobic condition in the prominent metabolic alteration among them.[25]

Hepatocellular carcinoma (HCC) represents an arduous global health challenge due to its prevalence, marked by high mortality and morbidity.[[] Subsequently, we picked up ten keywords to observe cancer from muti-omics and multangular viewpoints. The first one is characteristics.

Table 1: Characteristics and constancy attributed to various cancers. Hepatocellular carcinoma (HCC) represents an arduous global health challenge due to its prevalence, marked by high mortality and morbidity.[I]

NO	Nomination	Feature	Function	Treatment, and /or finding		Reference
1	Neutrophil	Prominent leukocyte in peripheral blood that accumulate in tumors.	Play a role in cancer progression and the interactions between neutrophils and the tumor microenvironment.	Platelets ensnared via NET(neutrophil extra type trap) oasis have a potential to obstruct the ingress of circulating tumor cells.	Leukemia, breast cancer, hepatocellular carcinoma, renal cell carcinoma, lung adenocarcinoma, prostate carcinoma, ovarian cancer, colorectal cancer, head and neck carcinoma	[26]
2	Artificial Neural Network	The differentially expressed genes are identified and compared between the normal and cervical cancer tissues.	A large number of deferentially expressed genes can help identify target protein and agents to treat cervical cancer patients.	Tumor-infiltrating immune cells act as important determinant of the tumor microenvironment and cancer treatment response.	Cervical cancer	[47]
3	Cancer stem cell	They have the ability to replicate cells identical to their own	They are deeply involved in the formation, growth, metastasis, and recurrence of cancer.	CD44v was found in cancer stem cell. CD44v needs cysteine to produce glutathione which promotes cancer. Sulfasalazine blocks cysteine resulting in positive cancer treatment.	Liver cancer, breast cancer, head and neck carcinoma, hepatocellular carcinoma, renal cell carcinoma, lung adenocarcinoma, prostate carcinoma, ovarian cancer, colorectal cancer	[28,29]
4	Ginger oleoresin	Ginger oleoresin inhibits HGC-27 human gastric cancer cell proliferation at the rate of 4.05-41.09 % and induced cell apoptosis at the rate of 10.4 –20.9 %	The anticancer potentials of ginger phenolic compounds could be attributed to the presence of hydroxy group in the unbranched 1-alkyl chain and to the length of carbon side chain.	Ginger oleoresin shows substantial anticancer therapeutic potential and can be used for the novel food-drug development.	Gastric cancer	[49]
5	Tomor initiating cells	They are founder cells for metastasis formation.	They are functionally characterized in hematological malignancies as well as in solid tumors.	Application of a broad range of protease inhibitors to TIC reduced anchorage independent growth.	Breast cancer	[9]
6	NPM-ALK	NPM-ALK /βactin	Pediatric ALCL	The cell not expressing	Pediatric ALCL (anaplastic	[7]

	fusion protein	ratio was compared to those determined by electrophoresis in good agreement.	(anaplastic large cell lymphoma) are associated with the t(2;5)(p23;q35)translocation which fuses the Anaplastic Lymphoma kinase gene. This fusion protein is an active tyrosine kinase which plays a major role in tumor pathogenesis.	NPM-ALK fusion protein but lysed with the Cell Lysis Buffer gave only background ELISA signal.	large cell lymphoma)	
7	Prognostic factors as patient characteristic	Age, gender, performance status metastasis's location, and adjuvant chemotherapy are often reported.	It has the potential to determine the survival of patients.	A common set of characteristics and strata will end in trial reporting, interpretation and future meta-analysis.	Colorectal cancer	[11]
8	Plasma from cancer patients	degradation products of antiplasmin and laminin-chain, and elevated levels of acute phase proteins	laminin-chain, and elevated levels of acute phase proteins have been described as anti-apoptotic factors	I found that formation of the CD95 death-inducing signal complex was strongly inhibited in the presence of plasma from cancer patients.	They found that formation of the CD95 death-inducing signal complex was strongly inhibited in the presence of plasma from cancer patients.	[12]
9	Clone dominance	Characteristics of in vivo tumor engraftment	Measurement of cancer cell growth heterogenicity through lentiviral barcoding identifies clone dominance			[13]
10	Machine learning	feature	Function	Treatment/finding	Gallbladder cancer	[141]

1.2 Relevant issues attributed to cancer development

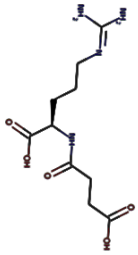
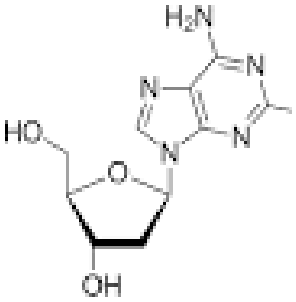
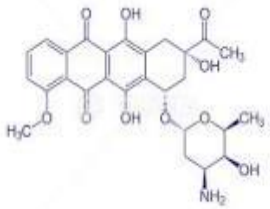
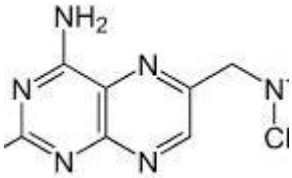
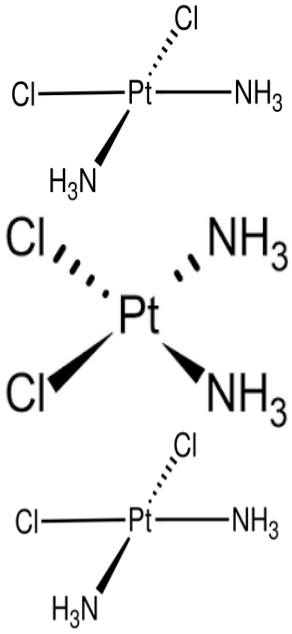
Signal induced proliferation associated protein 1 (SIPA1), a mitogen-inducible gene, has been involved in tumor metastases. The SIPIPA1 polymorphisms can be associated with several different types of cancers and interaction between SIPA1 and binding molecules encompass a series of cellular functions, which may promote the development of cancer metastasis.[25] After the middle age of our life the development of cancer usually exhibit genomic instabilities and multiple mutations. The genomic instability is associated with mutations that contribute cellular immortalization and transformation. Cancer develops when cancer-initiating cells, called stem cells, develop as a result of these mutations.[31] Under Evolian from cytoplasmic organelles to the nucleus, the stem cell has been associated with aging, and human pathologies such as cancer, neurogenesis., hematogenesis and the like. The transformed mitochondrial DNA (mDNA) fragment in the nuclear genome is called nuclear NUMTs. Keshov et al. called this process numtogenesis and they reported article claiming numtogenesis as a mechanism for development of cancer.[32]

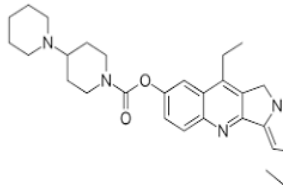
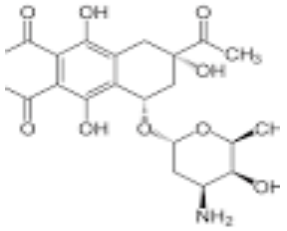
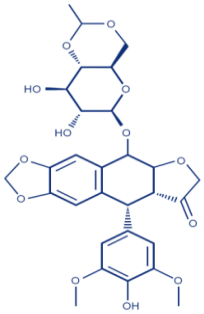
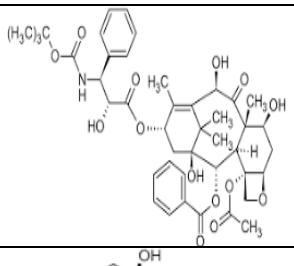
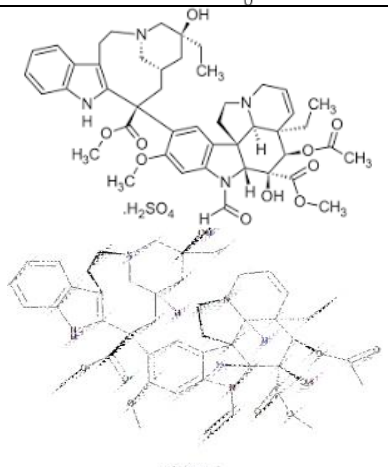
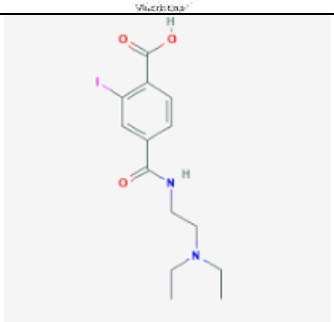
Table 2: Relevant issues attributed to caner development.

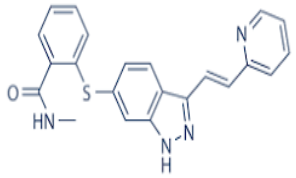
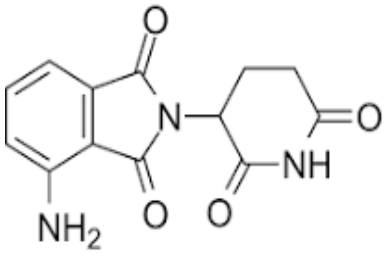
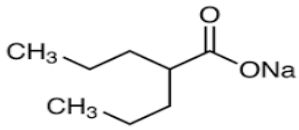
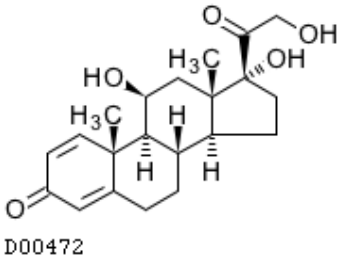
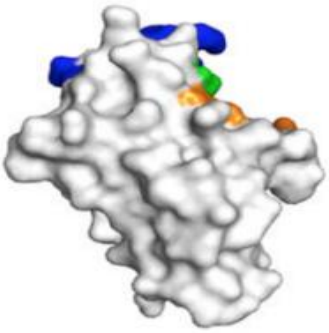
NNO	Relevant developmental factor	Role of developmental factor	Types of related cancer	Therapeutic remarks	Reference
1	NF-κB (nuclear factor-κ B) pathway and angiogenesis	NF-κB is involved in regulation of different pathways including apoptosis, cell proliferation, inflammation, and tumor migration	Colorectal cancer		[31]
2	Microbiome	Cause dysbiosis mucosal barrier disruption and treatment-related toxicity			[32]
3	Keap1-Nrf2	In some critical disease such as cancer Keap1 is found somatically mutated, leading to cancer initiation and malignant progression. Nrf2 can enhance survival and proliferation of cancer cells.	Lung cancer, liver cancer, endometrial cancer, gallbladder adenocarcinoma, breast cancer, adenocarcinoma-of the appendix, gastric adenocarcinoma, kidney cancer, colorectal cancer, ovarian cancer, esophageal cancer, pancreatic cancer, prostate cancer, malignant melanoma, carcinoma of the urinary tract.	Stabilize he redox system (e.g. eliminating reactive oxygens)	[33]
4	PRMT(protein antigen methyl transferase-1)	PRMT1-mediated changes in chromatin accessibility and transcription control the development of pancreatic cancer.	pancreatic cancer	Gemcitabine (Gem) is a first-line therapeutic option for pancreatic cancer and pharmacological inhibition of PRMT1 synergizes with gem treatment.	[34]
5	microbiota	Microbiota could colonize tumor tissues through mucosal destruction, adjacent tissue migration, and hematogenic invasion	Lung cancer, liver cancer, colorectal cancer, gastric cancer, breast cancer, pancreatic cancer, oral cancer, head-and-neck carcinoma, esophageal cancer, nasopharyngeal carcinoma, ovarian carcinoma, endometrial cancer, cervical cancer, prostate cancer, bladder cancer, pituitary neuroendocrine tumor, Birkitt's lymphoma, NK cell and T cell lymphomas, lymphocytic leukemia	Certain medications such as antibiotics, and antiviral drugs have been used to eradicate microorgan-ism. In addition, biopsy, and immunotherapy have been used,	[36]
6	Alcohol	Modulation of estrogen levels, generation of reactive oxygen species, production of acetaldehyde, promotion of chronic inflammation and induction of epigenetic changes.	Breast cancer, and ovarian cancer	Avoid alcohol consumption	[37]
7	Collagen	Collagen plays a critical role in regulating tissue homeostasis whose	Breast carcinoma, gastric cancer, papillary thyroid carcinoma,	Typical inhibiters of collagen include	[38]

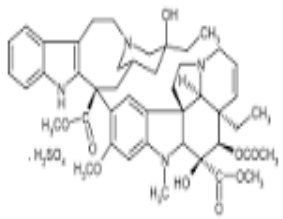
		balance can be disrupted due to an abundance of collagen affecting tumor growth, cell invasion, and metastasis.	colorectal carcinoma, esophageal cancer, pancreatic cancer, lung cancer, ovarian cancer, melanoma, glioblastoma, oral squamous cell carcinoma, hepatocellular carcinoma, glioblastoma, prostate cancer, clear cell, renal cell carcinoma, cervical cancer, hepato-carcinoma,	budesonide, catechol, coumaric acid, ganetespib,[222]	
8	Sorkin	Aberrant Sorkin expression and/or gene amplification was found related to epithelial-mesenchymal-transition, tumor, immune microenvironment, and drug resistance.	Neuroblastoma, adenocarcinoma, glioma, ovarian cancer, central nervous system cancer, gallbladder cancer, hepatocellular cancer, and leukemia.	Sorkin's involvement in tumorigenesis and multidrug resistance lines suggest that lowering its effect could be a viable strategy by using microRNAs, siRNA, growth factor (TGF-β1, TGFα), small molecules (Tetrandrine, PH-H7, dihydromyricetin, Ondansetron, Calcitriol, Palmitate, Triptolide) and protein extracts (Harshens)	[6,33]
9	Adipose tissue	The role of adipose tissue is the initiation and progression of breast and prostate cancers. Obesity dramatically modifies adipose tissue microenvironment in numerous ways including induction of fibrosis, and angiogenesis, increased stem cell abundances, and expansion of proinflammatory immune cells.	breast and prostate cancers	As many of obesity modifications also resemble the shift observed within tumor microenvironment, proximity of adipose tissue may present a hospital environment in developing tumors .	[6,39]
10	ER(endoplasmic-reticulum)-phage	When physiological or pathological stimuli induce disorders of ER function, misfolded protein trigger ER-phage which is beneficial for restoring cell homeostasis or promoting cell apoptosis.	Cancers in general	Mild ER stress is one defense mechanisms of tumor cells that can help to maintain cell homeostasis. However, strong stimuli can induce ER phase and even lead to apoptosis	[18]

Table 3: Major Anticancer drugs, their example, mechanism, and structure.[42,46-49]

NO	Category of anticancer drugs	Example of the relevant anticancer drug	Mechanism	structure	remarks
1	Alkylating agents	Busulfan	Alkylate DNA and keep the cancer cell from reproducing by damaging its DNA.		
2	Nucleoside analog (Antimetabolites)	Cladribine	Antimetabolites interfere with DNA and RNA by acting as a substitute for the binding block of RNA and. When this happens, DNA cannot make copies of itself and cell cannot reproduce.		
3	Ant-tumor antibiotics	Daunorubicin	Ant-tumor antibiotics work by changing the DNA inside the cancer cell to keep them from growing and multiplying.		
4	Antifolate	Methotrexate	Dihydrofolate reductase inhibitor Enzymes		
5	Platinum formulation	Cisplatin	React with DNA, inducing apoptosis, non-cell cyclic specific.		

6	Topoisomerase I inhibitor	Irinotecan	Topoisomerase I inhibitors interfere with enzymes called topoisomerases, which separate the strands of DNA so that they can be copied.		
7	Anthracycline	Idarubicin	Inhibit DNA and RNA synthesis by intercalating DNA base pairs by inhibiting topoisomerase II.		
8	Epipodophyllotoxins	Etoposide,	epipodophyllotoxins inhibit topoisomerase II.		
9	Texans	Docetaxel	Texans disassembly inhibit microtubule arrests cells in late G2 phase and Inhibit phase.***		
10+	Mitotic inhibitor (taaxanes and Vinca alkaloids)	Vincristine	Mitotic inhibitors work by stopping cells from deviding to form new cells. They can damage cells in all phases by keeping enzyme from making proteins needed for cell reproduction		
11	Monoclonal antibodies	Bevacizumab	Monoclonal antibodies are lab-made proteins that target specific proteins on cancer cells often blocking growth or signaling pathways.		

12	Tyrosine kinase inhibitors	Axitinib	Tyrosine kinase inhibitors (TKIs) work by blocking specific enzymes called tyrosine kinases that act as chemical messengers, sending growth and division signals in cells.		
13	Immunomodulatory	Pomalidomide	This surface protein acts as a “don't eat me!” signal that protects cancer from being consumed by certain immune cells; blocking CD47 can improve anti-tumor function of these immune cells.		
14	Histone deacetylase inhibitor	Valproate	Histone deacetylase (HDAC) inhibitors work by blocking the enzymes, HDACs, which remove acetyl groups from histones acetyl groups		
15	Corticosteroids	Pedonisone	Corticosteroids, or steroids, treat cancer by directly killing some cancer cells (especially in blood cancers), reducing inflammation and swelling caused by the tumor or cancer treatments, alleviating symptoms like nausea, pain, and fatigue, and managing complications such as brain swelling from brain tumors.		
16	Immunotherapy	Pembrolizumab	Cancer immunotherapy works by boosting your own immune system's ability to recognize and fight cancer cells. Unlike chemotherapy, which kills both cancerous and healthy cells, immunotherapy helps the immune system target and destroy tumors more specifically.		
17	Cancer vaccine	HepatitisB vaccine	Vaccines can stimulate the immune system to recognize and fight cancer cells.	—	

18	Vinca alkaloide	vinblastine	Vinca alkaloids work by binding to and inhibiting tubulin, a protein essential for forming microtubules during cell division. By disrupting microtubule formation, these drugs halt the progression of the cell cycle, particularly during the M-phase, leading to cancer cell death through apoptosis.		-

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1.4 A critical subject and its relevant mechanism and treatment

Pancreatic cancer, which exert its strong drug resistance, is an enormously malignant digestive cancer. Lycorine which is a natural plant origin alkaloid, exhibits anticancer effects on various cancers. X Zhou investigated the therapeutic effect of lycopine on pancreatic cancer to elucidate its molecular mechanism. The conversion of fatty aldehydes to aldehydes is inhibited by lycopine in pancreatic cancer with an aid of the inhibition of the expression of ALDH3A1 and oxidation of fattof ALDH3A1 and the oxidation of fatty acid due to the control of the expression of key enzymes involved in fatty acid oxidation resulting in the accumulation of lipids in cells, thereby inhibiting cell proliferation by bloching cell cycle progression and insusing apoptosis.[40]

Decorin, which is expressed by stromal cells and an archetypical small leucine-rich proteoglycan, affects cancer growth in its soluble form by interacting with several receptor tyrosine kinase (RTK). D. Mondal et al. identified that the soluble decorin inhibitd IV sprouting in an 3D model of lymphangiogenic, and in terms of mechanism they found that decorin interact with vasculat endserial growth factor receptor 3(VEGFR3), the main lymphatic RTK, and its action was required for decorin-mediated block of lymphangiogenesisc[40] X. -L. Huang et al. verified by in vitro experiment that ursolic acid (UA) can induce bladder cancer cell apoptosis by regulating the P13K/Akt signaling pathway.

Tale 4: Critical subject and its relevant mechanism and treatment.

NO	Valuable subject	Property of the subject	mechanism of the subject	Therapeutic aspect	Cancer type	Reference
1	Lycorine	Alkaloid of natural plant origin.	Lycorine inhibits the conversion of fatty aldehyde in fatty acids by inhibiting the expression key enzymes involving in fatty enzymes involving in fatty acid oxidation, leading to the accumulation of lipids in cells.	Two human pancreatic cell lines were treated with different concentrations of lycorine for different time periods.	Pancreatic cancer	[2]
2	Decorin	Decorin is archetypical small leucine-rich proteoglycan primary expressed by stromal cells.	It is implicated that decorin, as a biological factor with antilymphangiogenic activity and provide a potential therapeutic agent for curtailing breast cancer growth and metastasis.	The ccmpromized autophagy correrate with a reduction in the expression of these merkers. At the time the accumulation of Leve1in the presence of an inhibitor suggest s a potential nexus between autophagy and the intricate process of lymphangrogene-sis. This dual activity of	Breast cancer	[95]

				decorin ciould prevent systematic spread of breast cancer.		
3	Cinamalde-Hide	Cinamal-dehide is derived from cinnamon..	OncoTargets and Therapy 2020:13 9135–9145	Cinamaldehide can inhibit tumor growth, metastasis by suppressing angiogenesis. inhibiting the recruitment and polarization of immune suppressive cells, reducing the production of pro-inflammatory factors.	Clinical studies emphasize the possible translational significance of cinamaldehide in managing various types of cnacer.	[94]
4	Ursolic Acid	Pentacyclic triterpenoid compound	Ursolic acid can induce apoptosis in T24 cells by down regulating the P/13KAkt signaling patay	The low solubility of Ursoic acid hinder the clinical use. Nanotechnology may be a promising strategy to overcome its low solubility.	Bladder cancer	[52]
5	Yigong San	Yigong San is a representative prescription for the treatment of digestive disorders	The five hub genes of Yingong San are closely related to the biological processes of apoptosis, proliferation and pathogenesis of tumors cells, and may exert anti-tumor effect.	Yigong San has the effect of gastric cancer and immune regulation.	Gastric cancer	[53]
6	Forsythoside A	Forsythoside A is extracted from suspension of Forsythoside A, traditional Chinese medicine.	The effect of Forsythoside A in esophageal carcinoma is mediated through proteins such as BCL3 and BAX, influencing REGG pathways associated with apoptosis.	Forsythoside A contribute to the development of novel anti-tumor drugs for esophageal carcinoma and provide personalized treatment approaches.	esophageal carcinoma	[55]
7	Polysaccha-Rides	Polysaccharides in this study includes ginseng polysaccharide, astrogus polysaccharide, and Lyclum polysaccharide	Investigation of polysaccharide centers on the phenotypic alterations which encompass the regulation of cell cycle arrest, function of cell apoptosis, initiation of cell autophagy, control of cell migration and invasion, and modulation of the immune function	specific fungi polysaccharide, lentinan, has gained significant attention for food and pharmaceutical industries due to the wide-ranging applications and promising prospects	Gastric carcinoma	[56+]
8	Protopanaxadiol Ginsenoside	Protopanaxadiol Ginsenoside is the primary component of GRR which is roots of Panax gensen.	Protopanaxadiol Ginsenoside could inhibit proliferation, invation, and metastasis of hepatocellular carcinoma.	hepatocellular carcinoma have been found to inhibit the expression of matrix metaroprotenase, thereby suppressing hepatocellular carcinoma invation and metastasis.	hepatocellular carcinoma.	[54]

9	Claudin 8	Claudin is a protein associated with the loss of cell adhesion.	Claudin 8 could serve as an independent prognostic factor in kidney renal clear carcinoma。	Claudin 8 may suppress initiation and progression of kidney renal clear carcinoma.	kidney renal clear carcinoma	[83]
10	Resveratrol	A natural phytoestr-ogen	Resveratrol accelerates ferroptosis and inhibit malignant behaviors in oral cell carcinoma by regulating p53protein/SLC7A11messe nger RNA.	Resveratrol can provide a certain perspective for the treatment of oral cell carcinoma	Oral cell carcinoma	[84]

Pancratic cancer is Pyroptosis is a form of programmed cell death associated with antimicrobial responses during inflammation. In contrast to apoptosis, pyroptosis requires the function of caspase-1, and has been studied in the context of salmonella-infected macrophages. Recently, it was shown that Caspase-1 is activated during pyroptosis by a large supramolecular complex termed the pyroptosome. Only one large pyroptosome is formed in each macrophage, within minutes after infection. Biochemical and Mass Spectroscopic analysis revealed that this pyroptosomeis largely composed of dimers of the adaptor protein ASC.

1.5 Influential traits between occupation and cancer

Cancer survival and incidence are affected by socioeconomic disparities such aspects as ethnicity, gender, education, residence, and occupation. Temporal tendencies between occupation and cancer rates appears to be dynamic reflecting evolutions and progressive changes of society and the environment. The promotion of the use of personal protective equipment and the continuous surveillance protocols have been lead to the improvement of knowledge regarding cancer hazards.[27]

Table 5: Relationship between occupation and cancer.

		Characteristics of cancer in subject occupation		
1	a)Hematopoietic cancer b)Lip and melanoma	1	a) The exposure to pesticides may be contributed to b) increase risks to Hematopoietic cancer c) Risks of lip and melanoma may be attributed sun d) exposure. e) The reduced alcohol and smoking are attributed f) to the decreased risk of the ray of decreased risk of cancers such as lung cancer, liver cancer, prostate cancer, and larynx cancer.	[65]
2	Thyroid cancer	US Connecticut residents (462 thyroid cancer patient and *** controls)	a)general and operations managers (OR=5.36, 95% CI: 1.05–27.25) b) cooks and food preparation workers (OR=4.13, 95% CI: 1.04–16.39) c) retail salesperson (OR=3.13, 95% CI: 1.27–7.67) d) building cleaning and pest control worker (OR=2.36, 95% CI: 1.02–5.50) e)building and grounds cleaning and, maintenance workers (OR=2.12,95% CI: 0.99–4.54)	[66]
3	Tongue cancer	Tongue cancer patient in Nodic counties [9020 patients based on Nordic Occupational Study containing 14.9 million people]	***for men, statically elevated cases are as follows; a)waiters (SIR 4.36, 95% confidence interval (CI) 3.13-–5.92) b) beverage workers (SIR 3.42, 95% CI 2.02-5.40)	[67]

			c) cooks and stewards (SIR 2.55, 95% CI 1.82-3.48) d)seamen (SIR 1.66, 95% CI 1.36-2.00) e) journalists (SIR 1.85, 95% CI 1.18-2.75) f)artistic workers (SIR 2.05, 95% CI 1.54-2.66) g) hairdressers (SIR 2.17, 95% CI 1.39-3.22) h)d economically inactive persons (SIR 1.57, 95% CI 1.42-1.73) ***for women, statistically elevated only for; in waitresses (SIR 1.39, 95% CI 1.05-1.81)	
4	Beast cancer	Ontario workers (total of 17865 and 495 cases) observed from the Occupational Disease Surveillance Systemaa	Elevated risks were observed in a)management (Women(W): HR=1.54, 95%CI =1.40-1.70; Men (M): HR=2.79,95%CI=1.44-5.39, b)administration and clerical W:HR=1.16, 95% CI=1.11-1.21 c)teaching (W:HR=1.54, 95%CI=1.44-1.63; M:HR=3.00, 95%CI=1.49-6.03). Other elevated risks were U in nursing/health, social science, and janitor/cleaning services for both genders.	[71]
5	Lung, Gastric and colorectal cancers	Utilized the data of "Report of Vital Statistics of Occupational and Industrial Aspects" from the Japanese Ministry of Health, Laboure and Welfare.	*Lung cancer death rate (%) a) Unemployed (59.7) b) administration and managerial (50) c) service (4.9) . . h) sales (4.5) i) cerk(3.5) j) carrying, cleaning packaging (1.1) *Gastric cancer death rate (%) a) unemployment (57.5) b) service (4.9) c) administration and managerial (4.3) . . h) sale (4.2) i)clerk (3.5) j)carrying, clean- ing, package(1.0) ing (1.1)	[97]
6	Cancers among employed workers (n=1023)	White color workers and blue color workers (AOR)	White color & blue color; n(%) Stomach 25(12.4) 61(12.7) Colon 23 (11.4) 30(8.4) Breast 42 (20.9) 72(20.2) Cervix 37 (18.4) 68(19.2) Other 75(37.3) 128(36.5)	[73]
7	Thyroid cancer and radiaton-exposed workers aswell as. health care occupation			
8	Gastric cancer and occupations in coal and tin mining, metal, processing, particularly steel iron and rubber manufacturing industries. Wood processing and work in high temperature	.	a) Effort to access the benefit in occupation groups such as miners through identification of precursor lesions in dyspepsia clinics may provide a model for screening. b) If serological methods of screening	[96]

	environment have soso implicated, but the evidence is not strong		are developed occupational screening could come a steep closer.	
9	Breast cancer and lowest and highest occupations	8,678 survival Swissl patients in the non-parametric and parametric net survival ratios in.	Professionals with the highest skill level and belonging to top management and independent category had the highest breast cancer survival (BCS). Women in elementary occupations, with low skill level, and paid employ level had the lowest BCS.	[25]
10	Prostate cancer and occupations coded using ISCO 68	EPICAP (French population-based Case-control study) 819 prostate cancer patients and 879 controls	An increased risk of aggressive Prostate cancer for more than 10years	[26]

1.6 Association between personality and cancer

Aarstad et al. accomplished a study on personality traits in head and neck squamous cell carcinoma patients. Their aim was to investigate whether personality scores, as measured using the Eyseneck Personality Inventory (EPI,) are associated with the risk and prognosis of head and neck squamous cell carcinoma.[28]

Daniel et al. reported a study on personality disorders in patients with cancer. Patients with personality disorders exhibit character rigidity resulting from enduring patterns of inner experience and behavior. Therefore, they may experience some level of interpersonal conflict among medical staff caring for them. These conditions become exacerbated under stressful cancer-related situations, and it is likely that they may lead to adverse conditions and consequences. When they observe those patents carefully they noticed that patients tend to be chaotic, fearful, and scary,[27] Table 6 lists relevant reports attributed to the association between personality and cancer.

Table 6: A list of relevant investigation attributed to the association between personality and cancer.

	Type of personality and Cancer type	Test subject	Conclusion and/or findings	
1	Colorectal cancer (CRC) and various personalities		weak positive association between object-dependence/nappiness and CRC negative association histerical personality and CRC	[52]
2	Sever depressive symptoms with breast and/or cervical cancers.	Women in the UK biobank cohort who are eligible for breast cancer screening (age 50-70, n=143 460) and/or cervical cancer screening (<65 years, n=141 753)	A)more severe depression symptoms were associated with reduced likelihood of breast cancer (OR = 0.960, 95%: 0.950-0.970) and cervical cancer (OR = 0.58, 95% CI : 9.50-9.66).	[78]
3	Depressive symptoms, neuroticism and participation in breast and cervical caners	Women in the UK Biobank cohort who were eligible for breast cancer screening (aged 50 – 70, N = 143 461) and/or cervical cancer screening (>65 age, N = 141 753) at baseline recruitment (2006-2010) and those with follow-up data (2014 -2019) were identified (N=11 050 and N=9780) for breast and cervical cancer screening	Increased neuroticism is significantly associated with breast cancer screening (OR=1.01, 95%CI (1.00-1.02) and cervical screening (OR=0.99, 95%CI (0.98-0.99)	[51]
4	Logistic extraversion and participation in breast and gastric caner	Miyagi cohort study in Japan) N=21,911	The highest extraversion score had higher odds of gastric cancer screening attendance.	[79]
5	Demographic and socioeconomic covaries with breast, cervical, prostate, colorectal cancers	Health and retirement study in USA(N=14,394)	II. Increased conscientiousness was related with a)breast(OR=1.13),95%,CI:1.07-1.20, c) cervical(OR=1.14,95%,CI:1.07-1.20, d) prostate cancer (OR=1.08),95%,CI:1.01-1.16,. II. Higher neuroticism was associated with a) colorectal cancer (OR=1.05),95%,95%,CI:1.01-1.09. III. Increased extraversion was related with breast (OR=1.14),95%,CI:116Negative -1.32, cervical(OR=1.17,95%,CI:1.10-1.25. colorectal cancer (OR=1.06,95%,CI:1.01-1.02	[80]

6	Personality factors in colorectal cancer (CRC)	Based on Cochrane Collaboration and PRISMA	a) Positive association between cognitive orientation and CRC (p <0.001) b) Positive association between negative affect and presence of CRC c) Positive association between anger subscale and CRC) (p,0.001 d) Strong positive association between denial of anger and CRC in women (p=0.002) e) positive association between commitment to social norms and CRC in women (p=0.002)	[49]
7	Personality characteristics and cancer screening	Data from German National Cohon (N=132,298)	a)extraversion with breast cancer (OR = 1.12, 95% CI : 1.14-1.22) b)agreeableness with skin cancer (OR=1.02.95%1.01-1.04) c) neuroticism with cervical cancer(OR=1.13,95%CI:1.11-1.15)	[81]
8	Personality trait and risk of cancer	Cohort identified in Swedish Twin registry	a) Neuroticism (low, 0-4), age-adjusted (1.02, 95% CI:0.90-1.17) for all cancers b) Extraversion (high, 5-9), age-adjusted (1.33, 95% CI:0.80-2.21) for respiratory cancer Extraversion (high, 5-9), age-adjusted (1.38, 95% CI:0.92-2.06) for virus-related and immune-related cancers cancers	[82]
9	Personality and breast cancer screening	The target population consists of the employees of the French gas and electricity company, (44,992 men aged 40-50, and 13,511 women aged 35-50.)	a) Type A personality traits (sense of time urgency, high job involvement competitiveness) independently predicted use among postmenoposal women. b) For women with low level of these traits clinicians may help those with higher levels to better consider the risks of false positive and overdiagnosis.	[21]
10	Personality traits of cancer patients	Author's cancer patients	a) Patients with personality disorder will test physician's limitations of interpersonal skills. b) They treat physicians to provide organization, structure, and a blueprint for acceptable behavior. c) Although their oncological management may require interdisciplinary care, addressing these personality issue is an integral part of their comprehensive personal care.	[22]

1.7 Monography-Related Issue

The cell-cell interaction and dysregulation of cell morphology brings about cancer cell growth, migration, invasion, and metastasis. In addition, a balance between the extracellular matrix (ECM) and matrix metalloprotease (MMP) are required for cancer cell morphology and angiogenesis. The comprehensive balance of morphology and microenvironment of primary central nervous system lymphoma enabled prognosis prediction by a combinatorial expression of 8 representative genes, including KRT17, CDH10, CDH18, COL8A2, ADAM22, ADAM28, MMP11, and MMP24.[33] Each of pancreatic ductal adenocarcinoma (PDAC) precursor lesions, invasive primary PDAC, and metastatic PDAC displays distinct morphologies representing unique biology. This 'biomorphology' is determined by a complex carcinogenetic history, geographic locations, external environmental exposures, intrinsic metabolic demands, and tissue migration patterns. Understanding is not only of academic interest but also of great practical value. Applying this bio-morphological evolution of PDAC progression knowledge to surgical pathology practice facilitates the correct diagnosis on routine two histological stains including hematoxylin and eosin stains Most biopsy and resection specimens are currently obtained prior to treatment making sure that understanding of untreated PDAC bio-morphology is mature. The bio-morphology of treated PDAC is less defined but will assume greater importance as the frequency of neoadjuvant therapy increases.[34]

Table 7: Morphology-relatded issues.

	Morphological item	Test subject	Cancer type	Conclusion and/or findings in	Ref
1	cachexia	Weight-stable cancer patients (n=12;6/6[male/female]) and cachetic cancer patients (n=10;6/4[male/female])		Cachexia compromises CNS(central nervous system) morphorogy mostly causing change in the GM in the chachetic patients	[6]
2	Xenoestrogen	MCF-7 breast cancer cells and MDA MB-231 (both ATCC, Manassas, VA, USA) invasive phenotype breast cancer cells were used.	Breast cancer	Xenoestrogens Affects Breast Cancer Cell Morphology and Motility	[7]
3	plasminogen activator inhibitor-1 (PAI1)	HCC1954 (ATCCCat#CRL2338) and SKBR3 (ATCCCat#HTB30), which are HER2(human epidermal growth factor receptor -2) positive breast cancer cell lines, were used.	Breast cancer	Cell morphology and migration capabilities were influenced by PAI1 overexpression or silencing.	[8]
4	vocal folds	Group A; 79 leukoplakia patients (21 female, 58 male) Group B; 21 malignant lesion patients (4 female and 17male)	Leukoplakia, and malignant lesion	In combination with further standardized morphologic endoscopy findings should be undertaken	[9]
5	exercise dose	Twelve women with stage III or IV ovarian cancer completed a 12-week supervised resistance exercise intervention	ovarian cancer	Resistance exercise dose effects on muscle morphology, muscle function and quality of life in advanced‐stage ovarian cancer survivors	[10]
6	Spheroid	Five lung cancer cell lines, H1975, A549, H1299, PC6, and PC9, were used.	Lung cancer	MCTS(multicellular tumor Spheroid) morphology could be used in basic oncology research and as a clinical prognostic tool.	[11]
7	Foodborne Xenoestrogens	MCF-7 breast cancer cells and MDA MB-231 invasive phenotype breast cancer cells were used.	Breast Cancer Cell	Short-Term Exposure to Foodborne Xenoestrogens Affects Breast Cancer Cell Morphology.	[12]
8	keratin 19 (K19)	The MDA-MB-231 cell line was used	breast cancer	A new quantitative method of single cell analysis from DHM (digital holographic microscopy), facilitated evaluation of K19–substrate interactive effects on cell morphology.	[13]
9	S 100 family proteins	PdxCre ; RosaYFP (CY) and PdxCre ; KrasG12V; p53R172H; RosaYFP (CKPY) mice were maintained on a C57BL/6 background and bred and maintained in a specific pathogen-free animal facility.	pancreatic cancer	exogenous TGFβ1 induces solid organoid morphology that is associated with changes in the S100 family,	[14]
10	Lumican	Breast cancer, low-metastatic and ERa-positive MCF-7/c cells (#HTB-22TM), and highly metastatic, ERb-positive MDA-MB-231 cells (#HTB-26TM) were obtained from	breast cancer	Downstream signaling pathways for integrins, were found to be downregulated by lumican	[15]

2. CANCER OVERVIEW BASED ON GRAPHOLOGY

The interest in graphic medicine has been growing recently, and health communicators started engaging readers with compelling visual and schematic accounts of health and medicine. One context where comics have been shown is cancer commutation.[43] Institutions around world are in the constant struggle to improve science communication. From calls for journal papers to be simpler and more accessible to encouraging scientists to take a more active role through community engagement, there is drive to demystify and improve public understanding of and engagement with science.[44] Based on the above-mentioned argument I will present schematic views for various cancer issues.

2.1 Cancer Cell

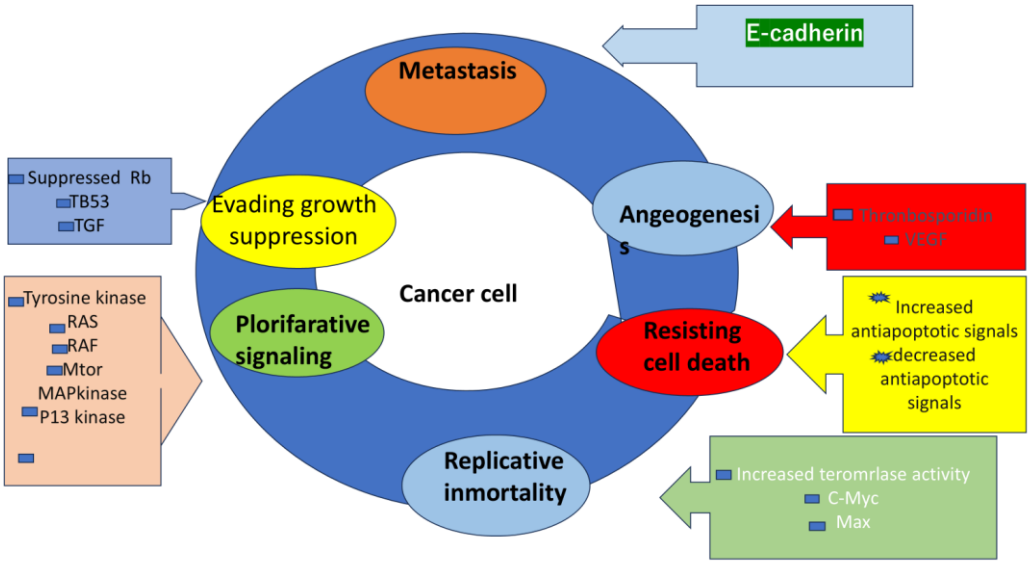

Figure 1: Increase in anti-apoptotic signals and decrease in pro-apoptotic factors denote resistance of cell death. Increase in telomerase creates replicative immortality. The increase in VGES and thrombospondin results in angiogenesis. E-cadherin instigate metastasis. The evading growth suppression mediate suppression of Rb TB53 and TGF.

2.2 Apoptosis-Related Feature

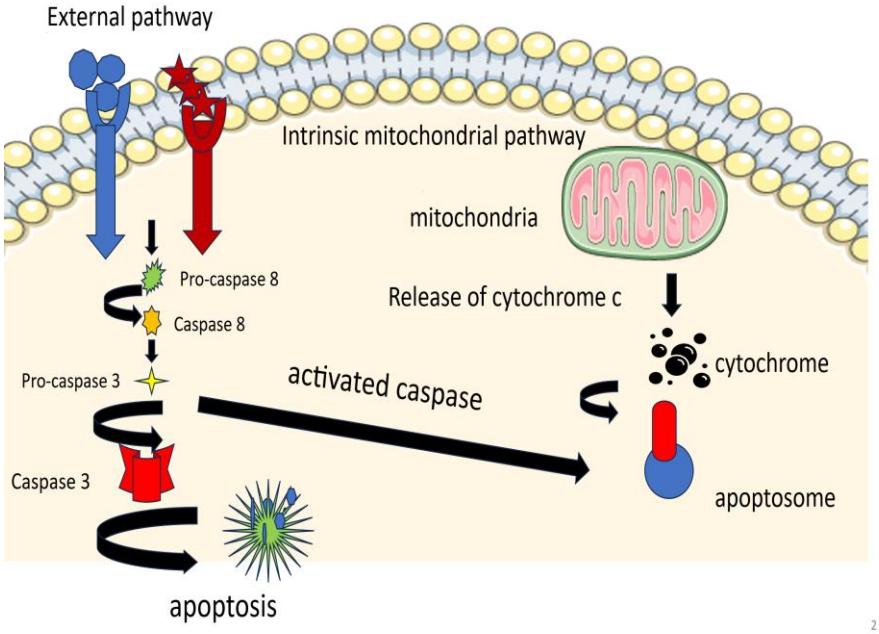

Figure 2: Apoptosis features a series of well-defined morphological changes, initially the volume of the cell diminish, a pyknotic nucleus is formed, and chromatin is condensed along the nuclear membrane. in two forma; intrinsic and extrinsic.

2.3 The pathways and therapeutic targets in the management of metastatic colorectal cancer featuring BRAFv600E mutation

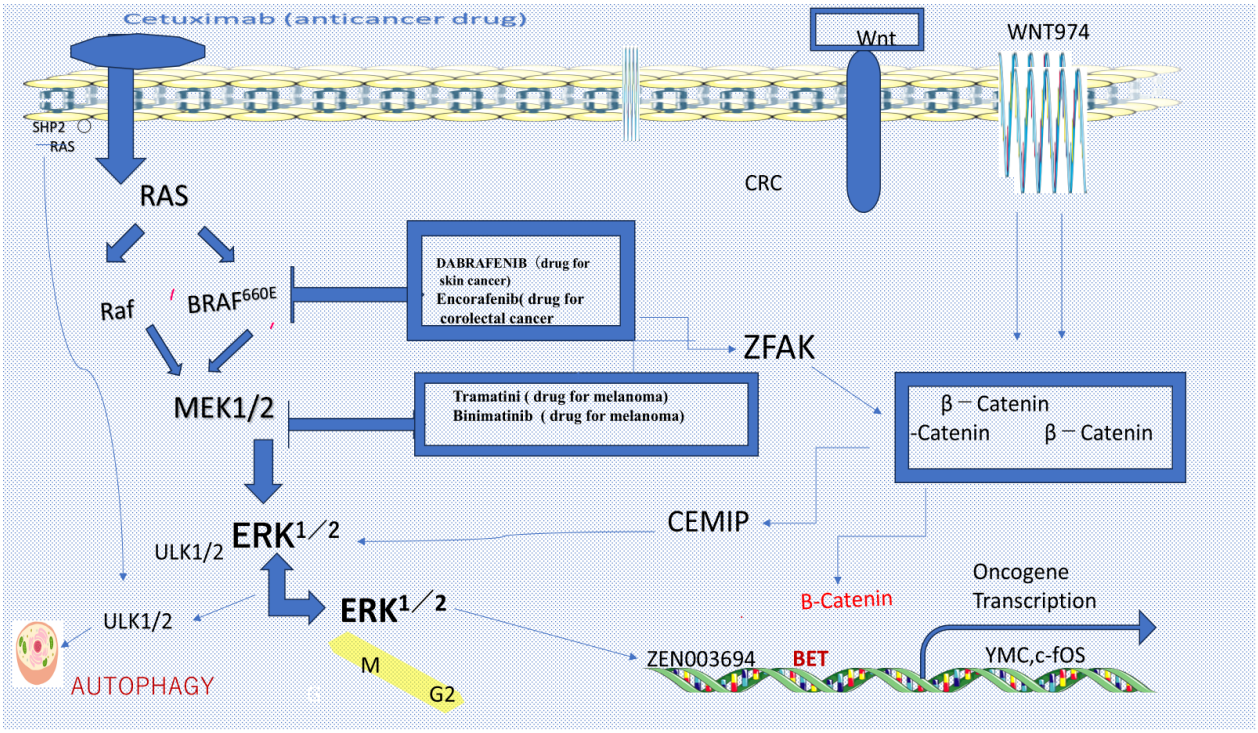

Figure 3: The pathways and therapeutic targets in the management of metastatic colorectal cancer featuring BRAFv600E mutation. RAS-RAF-ERK pathway is directory affected by the overactivated kinase activity of BRAFv600 Each pathway exibit exensive crosstalk each other.

2.4 Signaling pathway of the maintenance and survival of ovarian cancer stem cell

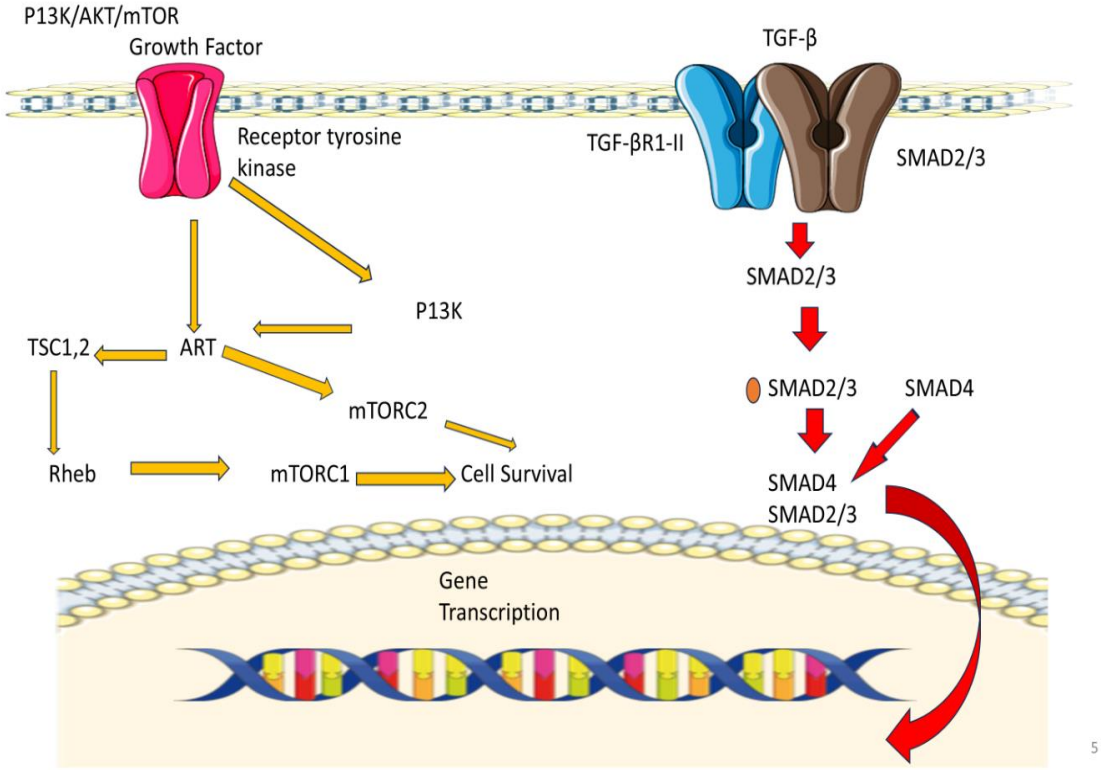

Figure 4: Signaling pathway of the maintenance and survival of ovarian cancer stem cell. The P13K/ARK/mOR pathway is activated upon growth factor binding to receptor tyrosine kinase. The left and right pathways are critically in the regulation of the ovarian cancer stem cell survival and therapy resistance.

2.5 The complexity between the collagen and the extracellular matrix

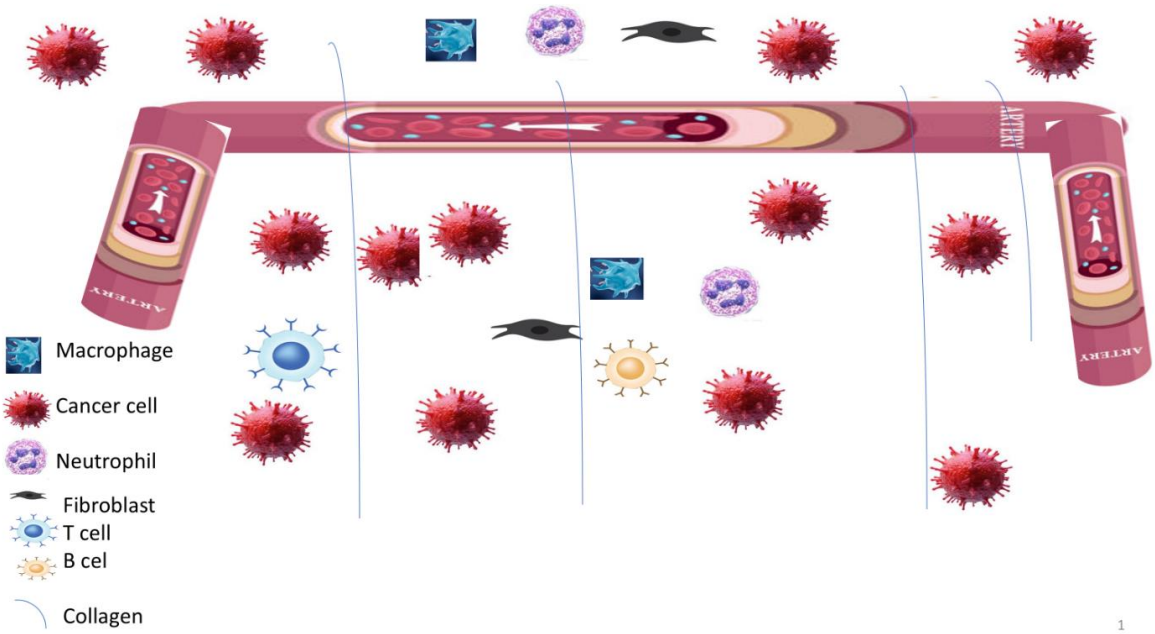

Figure 5: This diagram represents the complexity between the collagen and the extracellular matrix. The extracell matrix and multiple stromal cells play important roles in stimulating or inhibiting collagen function. Collagen-rich extracellular matrices bind to other molecules to form dense fibrosis. The activity of cancer cells is closely related to collagen.

2.6 The therapeutic target and level of action of anticancer drugs

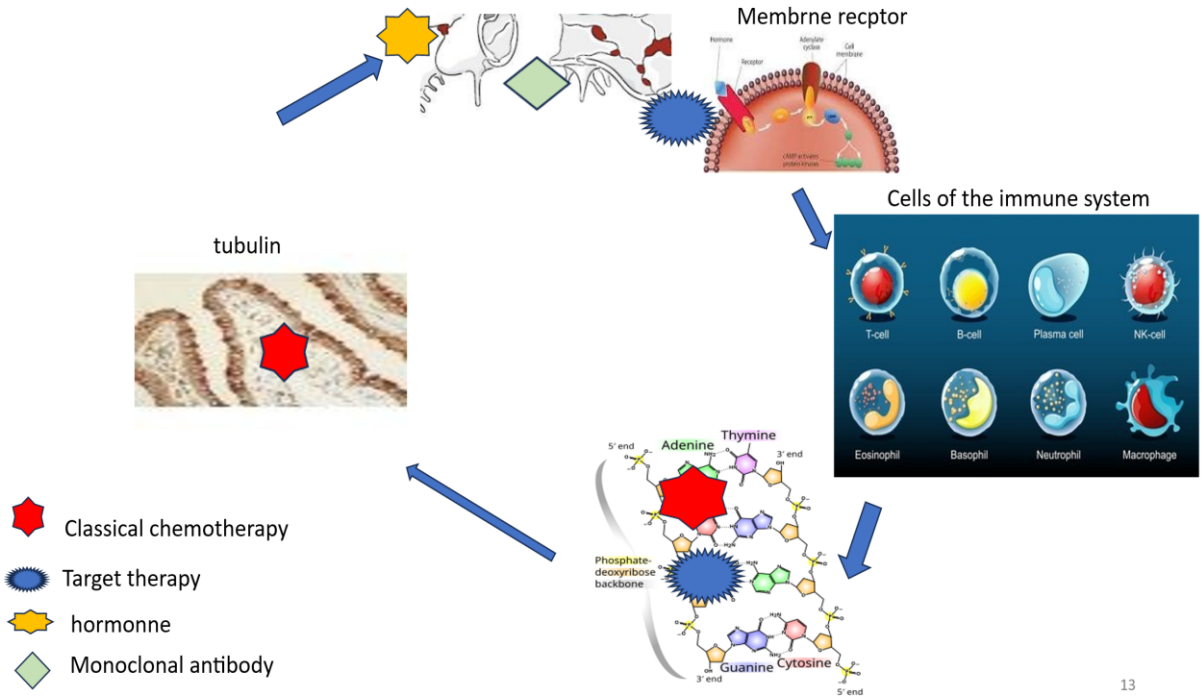

Figure 6: Anticancer drugs are able to categorized according to their therapeutic target and level of action. Most of them can interact with cancer cells, either in the nucleus, in the cytoplasm, or in the membrane. The mode of drug interaction will be either on tumor cells, on the tumor vasculature, on the immune system, or on the peripheral tissues.

2.7 Neuro stimuli-dependent immune system in the tumor microenvironment

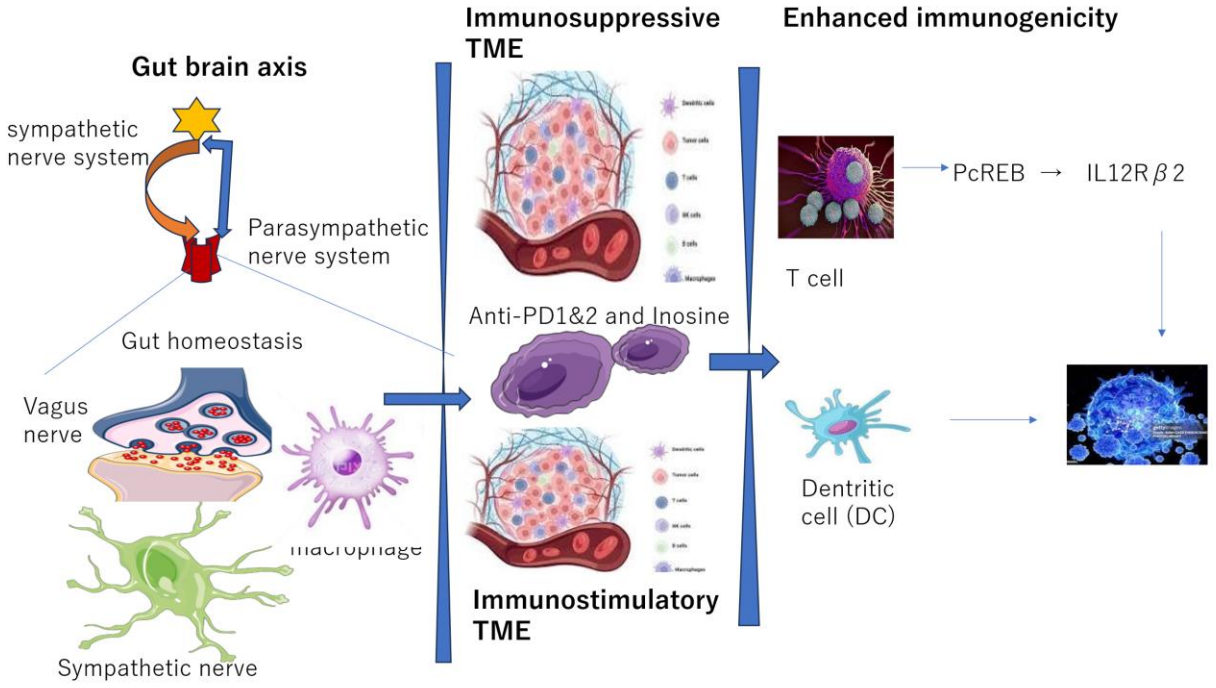

Figure 7: Neuro stimuli-dependent immune system in the tumor microenvironment. The gut and brain correspond through gut-brain axis (GBA). The immune and nervus systems cooperate to sustain inestinal homeostasis which is condusive to the development of probiotics. Through the inosine signaling cascate, inosine triggers the phosphorylation of cAMP response element-binding protein. This contribution of inosine in TME helps to alleviate enegetic contributon on CD8+ T cells leading to augment antitumor activity.（Note）TME：tumor-immune microenvironment.

2.8 The schematic representation of an atypical ERK3 in the regulation of breast cancer cell morphology and migration

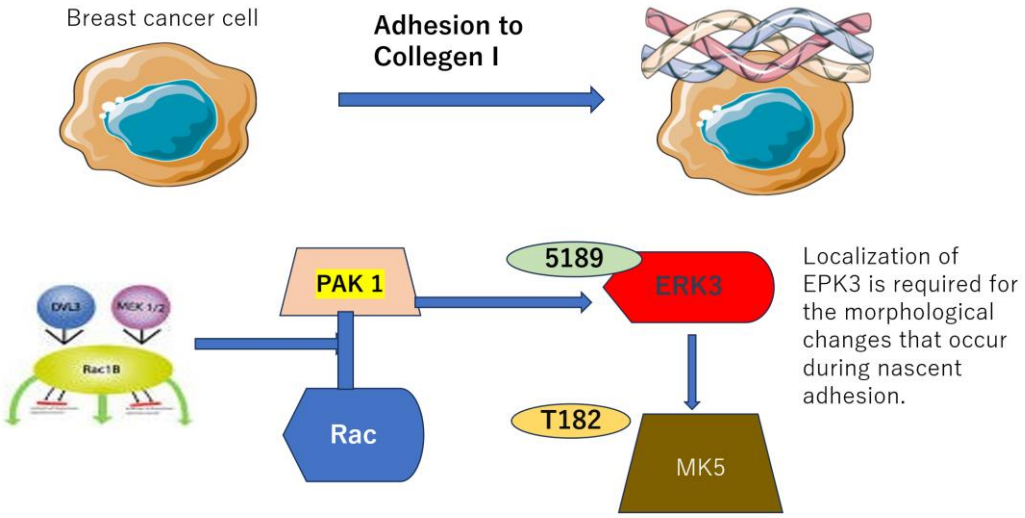

Figure 8: The schematic representation of an atypical ERK3 in the regulation of breast cancer cell morphology and migration.

Note）S189: Serine 189. T182: tyrosine 182. MK5: MAPK-activated protein kinase. MAPK: Mitogen-activated protein kinase. Rac: Regular Arrangement of Collecting venules.

2.9 CAR T cells are injected to the patient bloodstream

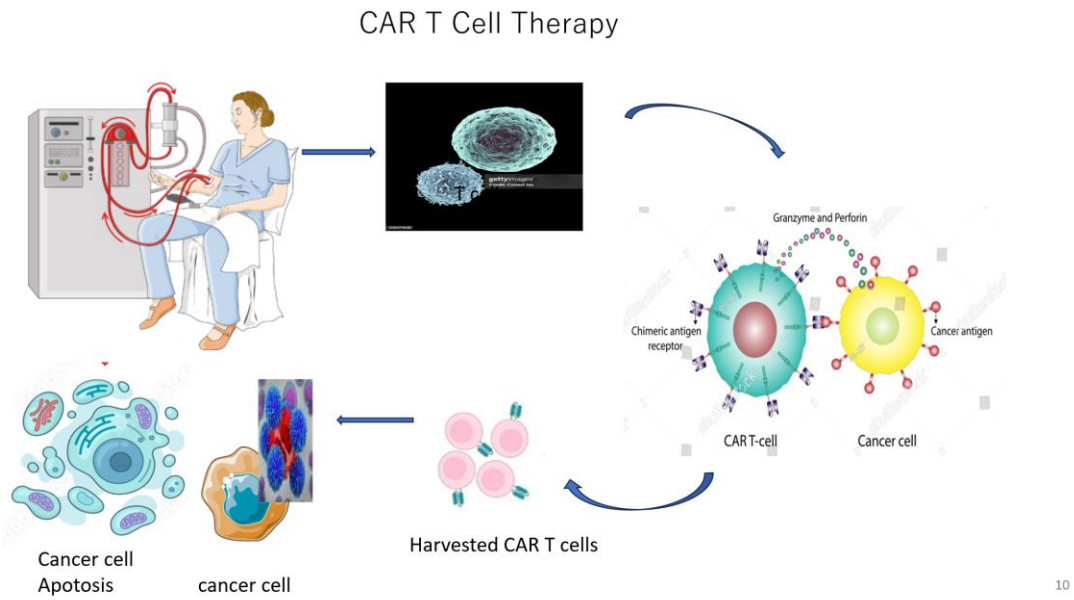

Figure 9: CAR T cells are injected to the patient bloodstream. The CAR T cells in the blood stream kill cancer cell. CAR T therapy is so-called immunotherapy in which the patient T cells are altered to introduce chimeric antigen receptor, enable them recognize and eliminate caner cells.

2.10 The schematic presentation of how mRNA chemotherapy takes place

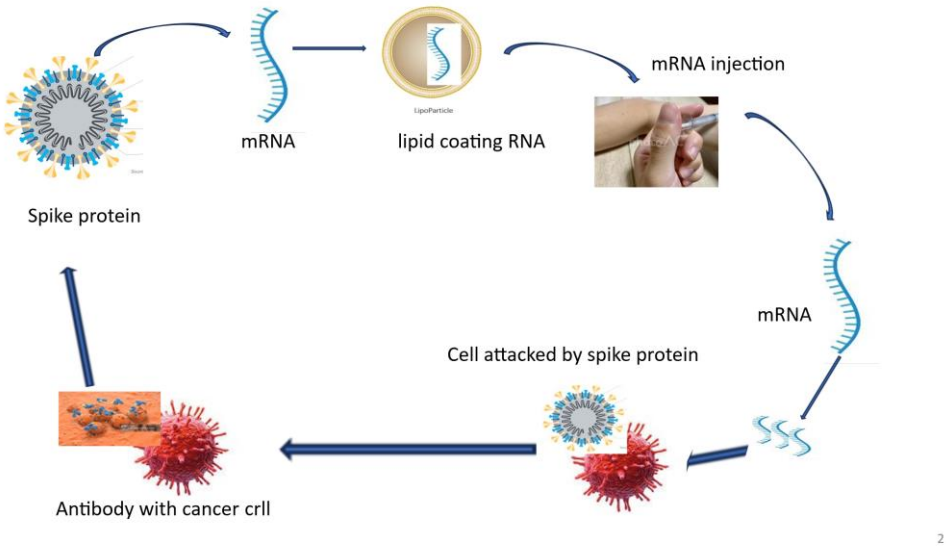

Figure 10: The schematic presentation of how mRNA chemotherapy takes place. The mRNA sequence codes for the virus spike protein and is generated in yellow-print so that the spike is swashed in lipid coating for delivery. Then protein fragments spur the immune system to produce antibodies which can protect the body to which virus enterdecos.

3. PROSPECTIVE PARADIGM AND COMMISSION

I have presented various aspects of cancer from a global standpoint. I elucidated the following issues.

a) DNA shouldn't be incapacitated from their original commission
b) Many types of cancers are caused by the oncogenes such as signal gene induced by proliferation associated protein 1 (SIPA1), a mitogen-inducible gene1, aberrant Sorcin expression and/or gene, chimeric fusion genes and so on. Strategies to eliminating oncogene will be effective to block relate cancers.
c) Cancer-causing factors will be pointed out to be

smoking, alcohol, obesity, radical exercise. Therefore we should avoid those cancer-causing factors which can be attributed not only in obligation occupations but also in the personality activities.

d) Aberrant Sorcin expression and/or gene amplification were found related to epithelial-mesenchymal-transition, tumor, immune microenvironment, and drug resistance. One way to block these obstacles, is the virtual screening to search molecule, which can block these obstacles will be beneficial.

e) A tyrosine kinase is an enzyme that can transfer a phosphate group to the tyrosine residues of specific proteins inside a cell from ATP. Tyrosine kinase inhibitors (TKIs) work by blocking tyrosine kinase enzymes. TKI enzymes help manage how cells work, including cell signaling and growth. Some tyrosine kinase inhibitors are used to treat cancer. TKIs work by blocking enzymes and keeping cancer cells from growing. In our body, there are so many types of enzymes, and we may be able to find pharmaceutical agents which block or inhibit harmful enzyme just like tyrosine kinase inhibitor to treat cancers.[86-97]

f) One of the difficulty in cancer therapy rises in the fact that we have to deal with so many types of cancers. As far as chemotherapeutic drugs are concerned, at least 17 drugs exist. Yet, we need more additional, beneficial agents. One of the clever approach is "thalidomide approach".[85] I propose an efficient way to find the second usage from the original usage which was approved by the government. This strategy can accomplished by docking and/or virtual screening.[88-92]

g) Cancers exist as so many types and forms. It will be medically ideal if we can find a homogenous cancer therapy including synchronized chemotherapy.[93]

CONCLUSION

Lycorine, decorin, cinamaldehide, ursolic acid, yigong san, forsythoside A, polysaccharide, protopanaxadiolginsenoside, Claudin 8, and resperatorol are worth mentioning substances for the cancer treatment. The exposure to pesticides may be contributed to increase risks to Hematopoietic cancer. Risks of lip cancer and melanoma may be caosed by sun exposure. The reduced alcohol and smoking are attributed to the decreased risk of cancers such as for lung cancer, liver cancer, prostate cancer, and larynx cancer.

As far as the statistically elevated cancer risk for men worker is concerned, waiters, beverage workers, cooks, stewards, and hairdressers show higher SIR ( ). Relationship between personality and cancer is concerned. weak positive association between object-dependence/nappiness and CRC() exist. Negative association shows hysterical personality and CRC. More severe depression symptoms were associated with reduced likelihood of breast cancer. Increased neuroticism is significantly associated with breast cancer screening. Increased conscientiousness was related with breast, cervical, and prostate cancer. The highest extraversion score had higher odds of gastric cancer screening attendance.

Xnoestrogens affects breast cancer cell morphology and motility and at the same time affects breast cancer celll morphology as well. Resistance exercise dose effects on muscle morphology, muscle function and quality of life in advanced‐stage ovarian cancer survivors. Cell morphology and migration capabilities were influenced by PAI1 overexpression or silencing. Resistance exercise dose effects on muscle morphology: MCTS (multicellular tumor Spheroid) morphology could be used in basic oncology research and as a clinical prognostic tool. Short-term exposure to foodborne xenoestrogens affects breast cancer cell morphology. Cachexia compromises CNS(central nervous system) morphology mostly causing change in the GM in the chachetic patients. A new quantitative method of single cell analysis from DHM (digital holographic microscopy), facilitated evaluation of K19–substrate interactive effects on cell morphology.

The pathways and therapeutic targets in the management of metastatic colorectal cancer features BRAFv600E mutation. Each pathway exhibit extensive crosstalk each other. The P13K/ARK/mOR pathway is activated upon growth factor binding to receptor tyrosine kinase.

The immune and nervus systems cooperate to sustain intestinal homeostasis which is conducive to the development of probiotics. Through the inosine signaling cascades, inosine triggers the phosphorylation of cAMP response of element-binding protein. Leading to augment antitumor activity.

The CAR T cells in the blood stream kill cancer cell. CAR T therapy is so-called immunotherapy in which the patient T cells are altered to introduce chimeric antigen receptor, enable them recognize and eliminate caner cells. As far as how mRNA chemotherapy takes place, the mRNA sequence, which codes for the virus spike protein, is generated.

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