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**Review Article**

# A Review on Advances in Anticancer Pharmacology

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## ABSTRACT

Recent developments in anticancer pharmacology have significantly enhanced the efficacy and specificity of cancer treatments. Innovations in drug design, including targeted therapies and immunotherapies, have improved outcomes for various malignancies. Key advancements include the use of monoclonal antibodies, small molecule inhibitors, and checkpoint inhibitors that leverage the immune system to recognize and destroy cancer cells. Additionally, the integration of personalized medicine approaches, utilizing genomic profiling, allows for tailored therapies that maximize therapeutic benefit while minimizing toxicity. Novel delivery systems, such as nanoparticles and liposomes, enhance drug bioavailability and reduce side effects. Ongoing research continues to explore combination therapies and novel agents, promising to further improve survival rates and quality of life for cancer patients. These advances underscore the importance of a multidisciplinary approach in developing effective anticancer strategies. Ceramide drug use for cancer treatment they work in a irrevissible cell injury means apoptosis process. Ceramide are the classical anticancer drug.

## INTRODUCTION

In cancer research each cancer sample presents the researcher with an altered genome that contains a unique and unpredictable number of point mutations, indels, translocations, fusions, and other aberrations. Since many of these alterations might never have been observed before and might not necessarily reside in coding regions of the genome, whole-genome sequencing is increasingly seen as the only rigorous approach that can find all the variants in a cancer genome. Among all these

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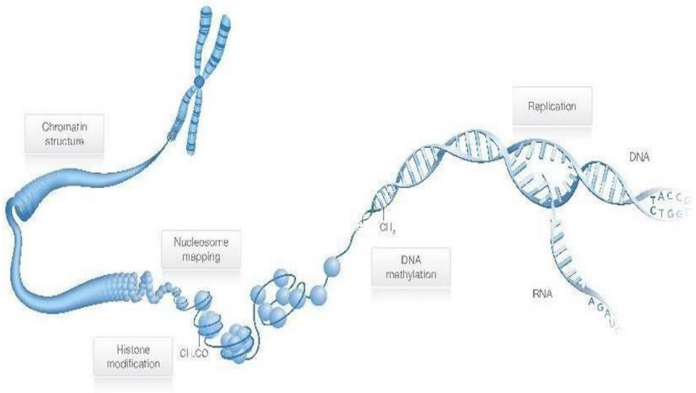
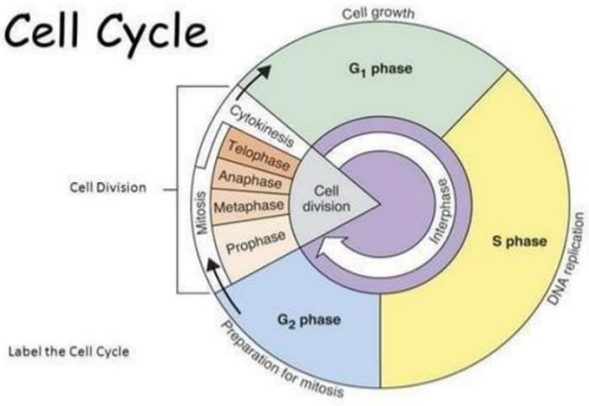



alterations are a select few that drive the progression of the disease. Based on the assumption that changes in gene expression levels impact disease progression, RNA- Seq is increasingly employed as a useful technique to determine if these genetic alterations impact disease progression. Genetic alterations have the potential to impact all cellular processes, including chromatin structure, DNA methylation, RNA splice variants, RNA editing, and microRNA (miRNA) to name but a few. Real progress in cancer research will come through the measurement and integrated analysis of all these interdependent processes. The key characteristic of next-generation sequencing technologies is that billions of independent sequence reads are generated in parallel, with each read derived from a single molecule of DNA. The resultant data approximate a random sample of DNA molecules which, in turn, represents the genomes of individual cells contained in the tumor sample.1 This provides us with a powerful toolbox to untangle the causes and mechanisms of cancer. (See Technical Considerations for additional information.) …(1-6)

**Anticancer Drugs**

Cancer is a disease of cells characterized by Progressive, Persistent, Purposeless and Uncontrolled Proliferation of tissues.

**Cont…**




Both normal and cancerous cells must pass through the following phases of cell cycle

1. **G1 phase ( presynthetic phase):** Synthesis of enzymes and other cellular components needed for DNA synthesis.
2. **Synthetic phase (S phase):** DNA synthesis takes place.
3. **G2 phase (premitotic phase):** Synthesis of cellular components for mitosis (proteins and RNA synthesis).

4. **Mitotic phase (M phase):** Mitotic cell division takes place

**G0 phase (resting phase):** Cells stop dividing temporarily or permanently (7)

**Cell Cycle- Specific (Ccs) Or Phase- Specific Drug**

**Antimetabolites**: Methotrexate, 6-mercaptopurine (6-MP)

**Antibiotic**: Bleomycin

**Taxane**: Paclitaxel

**Epipodophyllotoxins**: Etoposide, teniposide

**Vinca alkaloids:** Vinblastine, vincristine

Ccs drugs act mainly on dividing cells

**Alkylating agents:** Cyclophosphamide, busulphan, mechlorethamine, melphalan

**Anticancer antibiotics**: Doxorubicin, daunorubicin, mitomycin, actinomycin D

**Metal complexes:** Cisplatin, carboplatin

**Novel mechanisms of action of classical chemotherapeutic agents on sphingolipid pathways:**

**Generation of ceramide:** Ceramide can be generated by two distinct sphingolipid

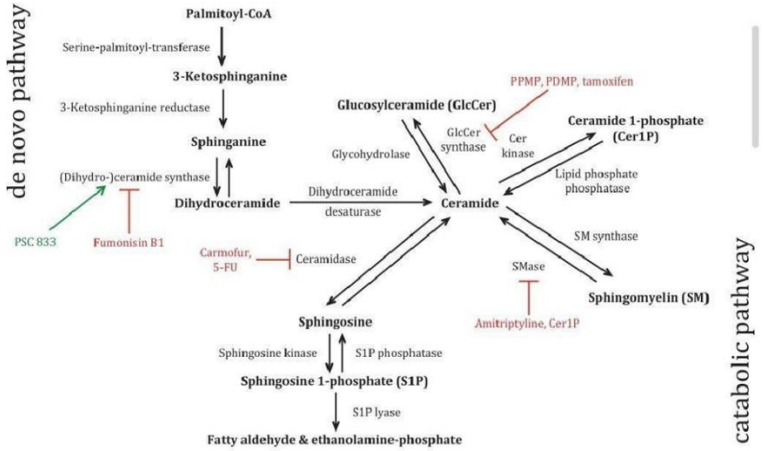
pathways. The first pathway is initiated by hydrolysis of the phospholipid sphingomyelin that is preferentially concentrated in the plasma membrane of mammalian cells. This hydrolysis occurs within seconds to minutes after exposure to cytokines (i.e., TNFα, Fas), hormones,radiation, or environmental stresses (Verheij et al., 1996).

Rapid catabolism of membrane-bound sphingomyelin to ceramide (Figure 1) is mediated by the action of the neutral or the acid sphingomyelinase (ASMase), which are sphingomyelin-specific forms of phospholipase C (Fukset al., 1995). Alternatively, the second pathway involves de novo synthesis of ceramide via the condensation of the sphingoid base sphinganine and fatty acyl-CoA catalyzed by the enzyme (dihydro-

)ceramide synthase (CerS) to form dihydroceramide, which is then oxidized to ceramide by dihydroceramide desaturase (Figure 1). Ceramide is generated in the endoplasmic reticulum and transferred to the Golgi apparatus to be functionalized as the primary hydroxyl (Hirschberg et al., 1993; Shimeno et al., 1995; Spiegel et al., 1996). De novo sphingomyelin biosynthesis depends on non-vesicular ceramide trafficking by the CERamide Transfer (CERT) protein ( Hanadaetal., 2003). The ceramide-producing pathways are cell-type specific. In bovine aortic endothelial cells, it was shown that ionizing radiation, like TNFα, induces rapid sphingomyelin hydrolysis to ceramide (Haimovitz-Friedman et al., 1994). Conversely, in epithelial cells, the slower CerS pathway is predominant (Kolesnick and Fuks, 2003; Mesicek et al., 2010). Moreover, a selective tissue and subcellular distribution of six mammalian CerS isoforms was described combined with distinct fatty acyl chain length substrate preferences, implicating differential functions of specific ceramide species in cellular signaling (Mesiceket al., 2010). Interestingly, overexpression of CerS2 results in partial protection from irradiation-induced apoptosis, whereas overexpression of CerS5 increases apoptosis in HeLa cells (Mesicek et al., 2010). Generation of long chain ceramides C16-ceramide and C18-ceramide led to the inhibition of cell proliferation and induction of apoptosis, whereas very long chain ceramide, such as C24:0-ceramide and C24:1- ceramide, increased cell proliferation in MCF-7 (breast cancer) and HCT-116 (colon cancer) cells (Hartmann et al., 2012). Thus, in addition to the balance of ceramide producing and metabolizing pathways, the chain length of the resulting ceramide species generated seems to be important for inducing cell death or survival.

**Mechanisms of ceramide-induced apoptosis**

Ceramide acts as a second messenger in initiating apoptosis via the mitochondrial




system. Apoptotic cell death refers to an inducible preprogrammed pathway that

Figure 1: Pathways of ceramide can be synthesized de novo, released from sphingomyelin or generated from sphingosine, glucosylceramide or ceramide 1-phos generation. Ceramide phase. 5-FU, 5-fluoruracile; Cer, ceramide; Cer1P, ceramide 1- phosphate; GlcCer, glucosylceramide; PDMP, 1-phenyl-2-Decanoylamino-3-morpholino-1-propanol; PPMP, 1-phenyl-2-palmitoylamino-3-morpholino-1-propanol; S1P, sphingosine 1-phosphate; SM, sphingomyelin; SMase, sphingomyelinase.Involves sequential events that ultimately lead to activation of calcium- and magnesium- dependent endonucleases that fragment the nuclear chromatin at specific Internucleosomal linker sites. Elevation of ceramide with Exogenous ceramide analogs was shown to be enough For induction of apoptosis in bovine aortic endothelial Cells (Haimovitz-Friedman et al., 1994). Moreover, protein Kinase C (PKC) activation blocked both radiation-induced Sphingomyelin hydrolysis and apoptosis, and apoptosis Was reinstated by exogenously added ceramide analog (Haimovitz-Friedman et al., 1994).In addition to inducing apoptosis, ceramides are also Involved in autophagy by down- regulating nutrient transporters (Guenther et al., 2008). Autophagy refers to a survival pathway responsible for the breakdown of damaged Organelles, protein aggregates and long-lived proteins. This Process is initiated by the generation of double-membrane Vacuoles, the autophagosomes, which engulf these cellular Components. Subsequent fusion of the autophagosomes With the lysosomes results in the formation of single membrane autolysosomes in which the cellular contents are Degraded by hydrolytic lysosomal enzymes. Autophagy is Therefore a catabolic survival pathway that plays a role in Cancer suppression (Klionsky, 2007).It was shown that senescent fibroblasts are more Resistance to TNFα-induced apoptosis as the result of, At least in part, interrupted ceramide signaling, which Suggests that senescence may be another way to escape Apoptosis (Wright and Shay, 2001; DeJesus et al., 2002). In cancer cells, senescence can be induced by two distinct mechanisms: replicative senescence which involves Inhibition of telomerase, and senescent-like state induced Through the overexpression of cell-cycle-inhibitory proteins or DNA damage. Senescent cells present a barrier To the effective treatment of cancer because they might Be capable of subsequently re-entering active cell cycling Or could provide support to other cancer cells, including Stem cells (Modrak et al., 2009).Lastly, short-chain ceramide (C8- ceramide) was Shown to be capable of inducing both senescence and Apoptosis in a dose-dependent manner (Modrak et al., 2009). C8-ceramide-induced senescence

occurred at lower Concentrations, whereas apoptosis was observed at higher Concentrations.

**Immunotherapy and targeted therapies in cancer treatment:**

**Immunotherapy=**

- Immunotherapy (also called Biological therapy the "treatment of disease by inducing, enhancing, or suppressing an immune response".
- Immunotherapies designed to elicit or amplify an immune response are classified as Activation immunotherapies.
- Immunotherapies that reduce or suppress are classified as Suppression immunotherapies.
- In recent years, immunotherapy has become of great interest to researchers, clinicians and pharmaceutical companies particularly in its promise to treat various forms of Cancer.
- Immunotherapy is not yet as widely u as surgery, chemotherapy, and radiation therapy. However, immunotherapies have been approved to treat people with many types of cancer.
- Cell – based immunotherapies are effective for some cancers. Immune effector cells such as Lymphocytes, Macrophages, Dendritic cells, NK cell, Cytotoxic T – lymphocytes work together to defend the body against cancer by targeting abnormal antigens expressed on the surface of tumor cells.

**Cancer Immunotherapy**

- Cancer immunotherapy is the use of the immune system to treat Cancer.
- Immunotherapies can be categorized as Active, Passive or Hybrid (active and passive).
- Active immunotherapy directs the immune system to attack tumor cells by targeting Tumor associated antigens (TAAs).
- Passive immunotherapies enhance existing anti-tumor responses and include the use of Monoclonal antibodies, Lymphocytes and Cytokines.
- Cancer Immunotherapy may works in three principal They are

i) Stopping or slowing the growth of cancer cells.

ii) Stopping cancer from spreading to other parts of the body

Helping the immune system work better at destroying cancer cells.

- Many different types of immunotherapy are used to treat cancer. They include:

1. Monoclonal antibodies
2. Adoptive cell transfer
3. Cytokines,
4. Oncolytic virus therapy
5. Cancer vaccines
6. BCG vaccines.

**01. Monoclonal Antibodies**

- Monoclonal antibodies are antibodies that made by identical immune cells that are all clones of a unique parent cell. Monoclonal antibodies can have monovalent affinity, in that they bind to the same epitope. In contrast, polyclonal antibodies bind to multiple epitopes and are usually made by several different plasma cell lineages.
- Monoclonal antibodies, which are drugs that are designed to bind to specific targets in the body. They can cause an immune response that destroys cancer cells.
- Monoclonal antibodies can "mark" cancer cells

**02. Adoptive cell transfer**

- Adoptive cell transfer is a T-cell therapy.
- Adoptive cell transfer is a treatment that attempts to boost the natural ability of T cells to fight cancer.
- T cells are a type of WBC and part of the immune system.
- Researchers take T then isolate the T cells from the tumor. They cells that are most active against cancer, modify the genes in them to make them better able to find and destroy cancer cells.

Researchers then grow large batches of these T cells in the lab.

### 3. Cytokines

- "Cytokine" is derived from Greek words – "c meaning cell and "kinos" meaning movement.
- Cytokines are small proteins (5 to 20 kDa).

Cytokines are Cell signalling molecules that aid cell to cell communication in immune responses.

- Cytokines play important roles in the body's normal immune responses and also in the immune system's ability to respond to cancer.
- The two main types of cytokines used to treat cancer are called Interferons (Interferons help the immune system fight cancer and may slow the growth of cancer cells) and Interleukins

Anterleukins heln the immune system produce

### 4. Oncolytic Virus Therapy

- Oncolytic Virus Therapy Uses Genetic Modified viruses (Herpes simplex virus) to kill cancer cells.
- First, the doctor injects a virus into the tumor. The virus enters the cancer cells and makes copies of itself. As a result, the cells burst and die. As the cells die, they release specific substances called Antigens. This triggers the patient's immune system to target all the Cancer cells in the body that have those same antigens.
- The virus does not enter healthy cells.

### *05* Cancer Vaccines

- Cancer vaccines belong to a class of substan known as Biological response modifiers.
- Biological response modifiers work by stimulating or restoring the immune system's

ability to fight infections and disease. There are two broad types of cancer vaccines:

1) Preventive (or prophylactic) vaccines, which are intended to prevent cancer from developing in healthy people. Example Cancer causing viruses (Hepatitis B virus and Human Papillomavirus).
2) Treatment (or therapeutic) vaccines, which are intended to treat an existing cancer by strengthening the body's natural immune

### C. BCG Vaccines

- BCG, which stands for Bacillus CalmeGuérin, is an immunotherapy that is used to treat Bladder cancer.

BCG is a weakened form of the bacteria that causes Tuberculosis (Mycobacterium bovis). When inserted directly into the Bladder with a catheter, BCG causes an immune response against Cancer cells.

### Administration Of Immunotherapy

- Intravenous (IV) The immunotherapy goes directly into a vein.
- Oral The immunotherapy comes in pills or capsules that you swallow.
- Topical – The immunotherapy comes in a cream that you rub onto your skin. This type of immunotherapy can be used for very early skin cancer.
- Intravesical The immunotherapy goes directly into the bladder.

### Side Effects Of Immunotherapy

- The most common side effects are skin reactions at the needle site. These side effects include: Pain, Swelling, Soreness, Redness, Itchiness and Rash.
- Flu- like symptoms, which include: Fever, Chills, Weakness, Dizziness, Nausea or vomiting, Muscle or joint aches, Fatigue, Headache, Trouble breathing and Low or high blood pressure.
- Other side effects might include: Swelling and weight gain from retaining fluid, Heart palpitations, Sinus congestion Diarrhea and Risk of infection.

### Introduction

➤Drugs or other substances that block the growth and spread of cancer by interfering with specific

molecules ("molecular targets") involved in progression, and spread of cancer. The growth,

➤"Molecularly targeted drugs," "molecularly targeted therapies," "precision medicines,"

➤The major modalities (pharmacotherapy) for cancer. Of medical treatment

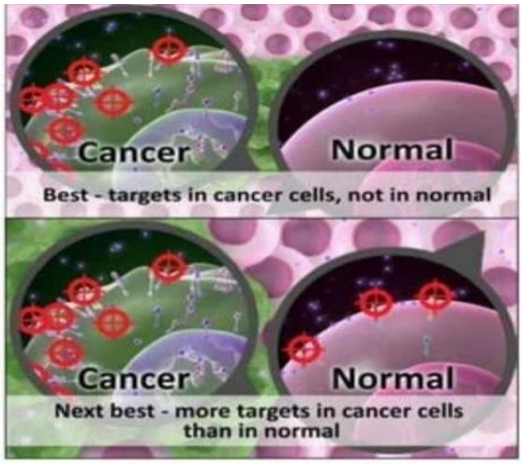
➤Interfere with specific targeted molecules needed for carcinogenesis and tumor growth rather than by simply interfering with all rapidly dividing cells (e.g. with traditional chemotherapy).

**Difference Between Targeted Therapy And Chemotherapy**

| Targeted Therapy | Chemotherapy |
|---|---|
| Act on specific molecular targets that are associated with cancer<br>Deliberately chosen or designed to interact with their target<br>Often cytostatic | Act on all rapidly dividing normal<br>and cancerous cells.<br>Identified because they kill cells.<br>Cytotoxic |

**Target Identification For Targeted Therapie**

Proteins present in cancer cells but not normal cells or more abundantin




cancer cells especially if they are known to be involved in cell growth or survival.

Example

- Human epidermal growth factor
- receptor 2 protein (HER-2) is expressed
- at high levels on the surface of some cancer cells.
- Neu growth factor receptor present at high number in breast cancer cells.

**Target identification cont'd….**

1. To determine whether cancer cells produce mutant (altered) proteins that drive cancer progression.

Example:

The cell growth signaling protein BRAF is present in an altered form (known as BRAF V600E) in many melanomas.

➤ Vemurafenib (Zelboraf®) targets this mutant form of the BRAF protein and is approved to treat patients with metastatic melanoma that contains this altered BRAF protein.

**Target identification cont'd….**

Abnormalities in chromosomes present in cancer cells but not in normal cells. Sometimes these result in the creation of a fusion gene whose product, called a fusion protein, may drive cancer development. Such fusion proteins are potential targets for targeted cancer therapies.

**Development Of Targeted Therapy**

The main categories of targeted therapy are currently small molecules and monoclonal antibodies.

A. **Small-molecule compounds** are typically developed for targets that are located inside the cell because such agents are able to enter cells relatively easily.

**Monoclonal antibodies** are relatively large and generally cannot enter cells, so they are used only for targets that are outside cells or on the cell surface.


## A. Small Molecule Targeted Therapy

Identified in what are known as "high-throughput screens," in which the effects of thousands of test compounds on a specific target protein are examined. Compounds that affect the target (sometimes called "lead compounds") are then chemically modified to produce numerous closely related versions of the lead compound.

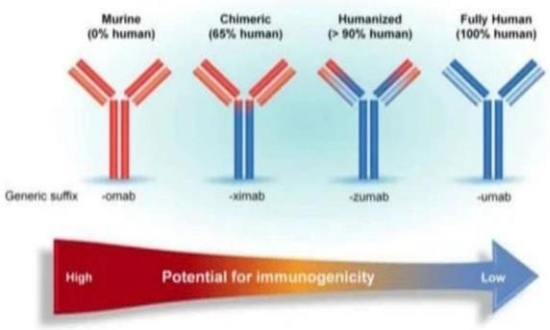
These related compounds are then tested to determine which are most effective and have the fewest effects on nontarget molecules.

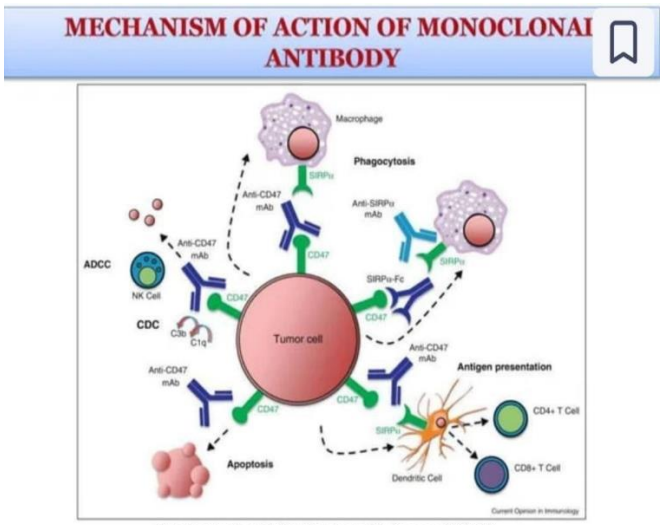
**Monoclonal Antibodies As Targeted Therap**

Developed by injecting animals (usually mice) with purified target proteins, causing the animals to make many different types of antibodies against the target.

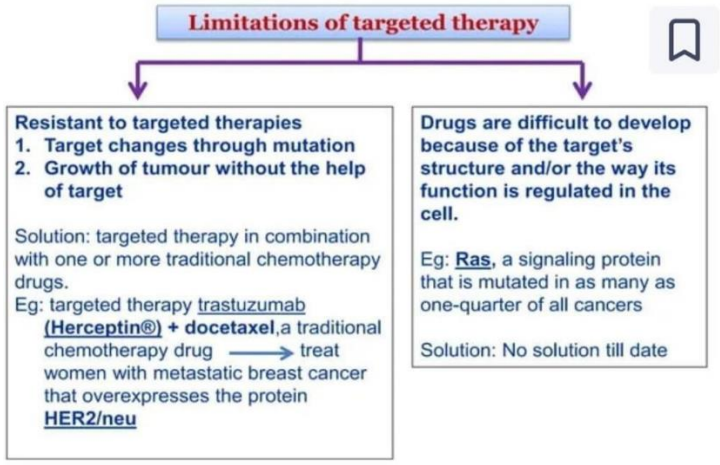
Antibodies are then tested to find the ones that bind best to the target without binding to non target proteins.

Before monoclonal antibodies are used in humans, they are "humanized" by replacing as much of the mouse antibody molecule as possible with corresponding portions of human antibodies.




Ref: Drebin JA, Link VC, Weinberg RA, Greene MI (1986)




**Side Effects Of Targeted Cancer Therapy**

Common side effects: diarrhea and liver problems, such as hepatitis an elevated liver enzymes.

Others include:

- Skin problems
- Problems with blood clotting and wound healing
- High blood pressure
- Gastrointestinal perforation
- V. Immunosuppression and impaired sperm production
- Certain side effects of some targeted therapies have been linked to better patient
- outcomes…

Patients who develop acneiform rash while being treated erlotinib (Tarceva®) or gefitinib (Orissa®), have tended to respond better to these drugs than patients who do not develop the rash.

Patients who develop high blood pressure while being treated with the angiogenesis inhibitor bevacizumab generally have had better outcomes.

## CONCLUSION

Immunotherapy and targeted therapy represent transformative approaches in cancer treatment, offering personalized strategies that enhance the body's immune response or target specific cancer cell mechanisms. Ultimately, their combination can improve efficacy, reduce resistance, and lead to better patient outcomes. Continued research is essential to optimize these therapies and identify the best candidates for treatment. Would you like to delve deeper into a specific type of cancer or therapy Significant advances have been made in the chemotherapeutic management of cancer. Many new cytotoxic drugs that can potentially be used for the treatment of cancer, this life-threatening disease still causes near 7 million deaths every year worldwide and the number is growing.Cancer chemotherapy is usually accompanied by severe side effects and acquired drug resistance. We anxiously await the development of target therapy that will allow greater tumor specificity and less toxicity. So, targeted therapies using small molecules or monoclonal antibodies gaing importence due to its high specificity almost negligible effect on neighbouring cells Despite the promising clinical results from the agents that is highlighted, there is still significant limitation to the concept of "pathway-specific" targeted therapies as that most solid tumors are the result of numerous genetic mutations, and thus inhibiting a single cellular pathway may not result in a significant therapeutic outcome.

## REFERENCES


1. Garraway L. A. and Lander E. S. (2013) Lessons from the cancer genome. Cell 153: 17-37
2. Soon W. W., Hariharan M. and Snyder M. P. (2013) High-throughput sequencing for biology and medicine. Mol Syst Biol 9: 640
3. Nik-Zainal S., Alexandrov L. B., Wedge D. C., Van Loo P., Greenman C. D., et al. (2012) Mutational processe molding the genomes of 21 breast cancers. Cell 149: 979-993
4. Shendure J. and Lieberman Aiden E. (2012) The expanding scope of DNA sequencing. Nat Biotechnol 30: 1084-1094
5. Tuna M. and Amos C. I. (2012) Genomic sequencing in cancer. Cancer Lett
6. Yates L. R. and Campbell P. J. (2012) Evolution of the cancer genome. Nat Rev Genet 13: 795-806
7. Tara sambhag and smita shenoy pharmacology by 4th edition page no. 459
8. Verheij, M., Bose, R., Lin, X.H., Yao, B., Jarvis, W.D., Grant, S., Birrer, M.J., Szabo, E., Zon, L.I., Kyriakis, J.M., et al. (1996). Requirement for ceramide- initiated SAPK/JNK signalling in stressinduced apoptosis. Nature 380, 75– 79.
9. Fuks, Z., Haimovitz-Friedman, A., and Kolesnick, R.N. (1995). The Role of the sphingomyelin pathway and protein kinase C in Radiation-induced cell kill. Important Adv. Oncol. 19–31.
10. Hirschberg, K., Rodger, J., and Futerman, A.H. (1993). The long-chain Sphingoid base of sphingolipids is acylated at the cytosolic Surface of the endoplasmic reticulum in rat liver. Biochem. J. 290, 751–757.
11. Shimeno, H., Soeda, S., Yasukouchi, M., Okamura, N., and Nagamatsu, A. (1995). Fatty acyl-Co A: sphingosine acyltransferase in bovine brain mitochondria: its solubilization and Reconstitution onto the membrane lipid liposomes. Biol Pharm Bull 18, 1335–1339
12. Spiegel, S., Foster, D., and Kolesnick, R. (1996). Signal transduction through lipid

second messengers. Curr. Opin. Cell Biol. 8, 159–167

13. Kolesnick, R. and Fuks, Z. (2003). Radiation and ceramide-induced Apoptosis. Oncogene 22, 5897–5906.
14. Mesicek, J., Lee, H., Feldman, T., Jiang, X., Skobeleva, A., Berdyshev, E.V., Haimovitz-Friedman, A., Fuks, Z., and Kolesnick, R. (2010). Ceramide synthases 2, 5, and 6 confer distinct roles In radiation-induced apoptosis in HeLa cells. Cell Signal. 22, 1300–1307
15. Hartmann, D., Lucks, J., Fuchs S., Schiffmann, S., Schreiber, Y., Ferreirós, N., Merkens, J., Marschalek, R., Geisslinger, G., and Grösch, S. (2012) Long chain ceramides and very long chain Ceramides have opposite effects on human breast and colon Cancer cell growth. Int. J. Biochem. Cell. Biol. 44, 620–628
16. Haimovitz-Friedman, A., Kan, C.C., Ehleiter, D., Persaud, R.S., McLoughlin, M., Fuks, Z., and Kolesnick, R.N. (1994). Ionizing Radiation acts on cellular membranes to generate ceramide and Initiate apoptosis. J. Exp. Med. 180, 525–535.
17. Guenther, G.G., Peralta, E.R., Rosales, K.R., Wong, S.Y., Siskind, L.J., And Edinger, A.L. (2008). Ceramide starves cells to death by Downregulating nutrient transporter proteins. Proc. Natl. Acad. Sci. USA 105, 17402–17407.
18. Klionsky, D.J. (2007). Autophagy: from phenomenology to molecular Understanding in less than a decade. Nat. Rev. Mol. Cell. Biol. 8, 931–937.
19. Wright, W.E. and Shay, J.W. (2001). Cellular senescence as a tumorprotection mechanism: the essential role of counting. CurrOpin. Genet. Dev. 11, 98–103
20. Modrak, D.E., Leon, E., Goldenberg, D.M., and Gold, D.V. (2009). Ceramide regulates gemcitabine-induced senescence and Apoptosis in human pancreatic cancer cell lines. Mol. Cancer Res. 7, 890–896
21. Press MF, Lenz HJ (2007). EGFR, HER2 and VEGF pathways: validated targets for Cancer treatment. Drugs 67: 2045-2075.
22. Drebin JA, Link VC, Weinberg RA, Greene MI (1986). Inhibition of tumor growth by A monoclonal antibody reactive with an oncogene-encoded tumor antigen. Proc. Natl. Acad. Sci. U.S.A. 83: 9129-9133.
23. Gibbs JB.(2000). Anticancer drug targets: growth factors and growth factor Signaling. J Clin Invest; 105:9-13. Druker, B. J, David A(2003). Karnofsky Award Lecture. Imatinib as a paradigm of targeted therapies. J. Clin. Oncol. 21, 239-245.
24. Alfaro. C,, Suarez N, Gonzalez A, Erro L, Solano S, Dubrot J (2009). Influence of bevacizumab, sunitinib and sorafenib as single agents or in Combination on the inhibitory effects of VEGF on human dendritic cell Differentiation from monocytes. Br. J. Cancer 100: 1111-1119.
25. Katzel JA, Fanucchi MP, Li Z (2009). Recent advances of novel targetedTherapy in non-small cell lung cancer. J Hematol Oncol 2: 2. 7.
26. Kim P. S, Perez-Gracia JL, Melero I, Gurpide A(2008). Antibody association With HER-2/neu-Targeted vaccine enhances CD8 T cell responses in mice through Fc-mediated Activation of DCs. J. Clin. Invest. 118: 1700-1711.

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