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ANTI NEOPLASTIC DRUGS

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Review Article

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Introduction:

Anticancer drugs are the drugs that prevent or inhibit the maturation and proliferation of neoplasms. These are not only used in different types of cancers but also in conjunction with radiotherapy and immunotherapy in the combined modality approach for many solid tumors, especially metastatic. Generally the term cancer is used for all malignant tumors.
Anti- neoplastic drugs is defined as the growth or mass of abnormal tissue formed due to excessive autonomous and uncoordinated cell proliferation.

Origin of cancer:

Generally in all the organs and tissues of human beings, a balance is maintained between cell and cell apoptosis (cell death program). There is a particular life span for all the mature old cells which die. And the new cells replace the old cells by proliferation and differenation mechanisms. This entire process is regulated such that the number of specific type of cells remains constant without altering the other process.
Occasionally, due to carcinogens (cancer causing agents) one of the cells gets mutated and does not respond to normal growth control mechanism. This mutated cell undergoes further mutation and transforms into tumor cell which starts proliferating vigorously.This interns results in a mass of abnormal cells called neoplasm or tumor.

Classification of Tumors Based on Origin and Composition:

Tumors are classified into many types based on their composition and origin cells. Basically tumor consists of parenchymal cells and stromal cells.

Parenchymal tumors:

Parenchyma is the function tumors. In which it alkylating agent. It is widely used to treat Hodgkin's andcontain only parenchymal cells are called medullary carcinomas.
a) Epithelial cells Eg: Squamous epithelial cells, hepatic cells, Glandular cells
b) mesenchymal cells Eg: Adipocytes, Fibrocytes, Bones, Skeletal muscles

Stroma:

It is a connective tissue of a tumour.it functions as the frame work for the parenchyma. It supplies blood
Eg: breast cell carcinoma

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Teratomous :

They are tumours of more than one germinal layer. They may be benign or malignant.
Eg: Embryonic cells
Ovaries
Testis

Causes of cancer:

Cancer is caused by changes (mutations) to the DNA within cells.

- Chemical carcinogens
- Age
- Lifestyle factors
- Radiation
- Infection
- Immune system
- Your genetic make-up

Classification of Antineoplastic Agents:

Classification of Antineoplastic Agents / Anticancer Drugs

Alkylating Agents

- Nitrogen mustards: Melphalan, Cyclophosphamide, Ifosfamide
- Nitrosoureas
- Alkylsulfonates
- Ethyleneimines
- Triazene
- Methyl Hydrazines
- Platinum Coordination complexes: Cisplatin, Carboplatin, Oxaliplatin

Alkylating agents are the covalent DNA binding drugs. These are the class 1 chemotherapy drugs .It help to stop the tumor growth by cross linking guanine nucleobases in DNA double- helix strands directly attacking DNA. It makes the strand unable to coil or separate. Alkylating agents are also mutagenic and Carcinogenic.

Nitrogen Mustard: Cyclophosphamide

Trade names: Cytoxan®, Neosar®

Cyclophosphamide is an Alkylating agent. Cyclophosphamide is used to treat Hodgkin's and non-Hodgkin's lymphoma. Because of wide variety of cancer it treats it also has a wide variety of administration options. Route of administration is given orally as well as intravenously with efficacy. The major site of alkylation within DNA is the N7 position of guanine. These interactions can occur on a single strand or on both strands of DNA through cross-linking. High doses of cyclophosphamide given as chemotherapy of cancer or as myeloablative therapy in combination of total body irradiation or busulfan in preparation for hematopoietic cell transplantation can induce sinusoidal obstruction syndrome.

Adverse effects:

- The side effects of cyclophosphomide depend on how much of the drug is given.

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• Low blood counts You're white and red blood cells and platelets may temporarily decrease. This can put you at increased risk for infection, anemia and bleeding.
Hair loss, nausea, vomiting, Discoloration of the skin or nails

Uses of Alkylating agent:

Alkylating agent are used in the treatment of a wide variety of hematologic and solid cancers, generally as part of a combination regimen

Antimetabolites

- Folate Antagonists: Methotrexate
- Purine antagonists
- Pyrimidine antagonists: 5-Fluorouracil, Cytarabibe

Antimetabolites drugs are the first effective chemotherapeutic agents. They are characterized by low molecular weights. Generally, antimetabolites induce cell death during the S phase of cell growth. Antimetabolites generally inhibit their synthesis or by competing with them in DNA or RNA synthesis.

Adverse effects:

Bone marrow suppression
Dermatologic
GI mucosa Natural Products

a. Plant Products

- Vinca Alkaloids: Vincristine, Vinblastine
- Taxanes: Paclitaxel, Docetaxel
- Epipodophyllotoxins: Etoposide
- Camptothecins: Irinotecan

The vinca alkaloids, vin cristine and vinblastine, disorganize the mitotic spindle to arrest cell division. While these are characteristic effects of the vinca alkaloids, they prob ably act by another mechanism, since vincristine differs from vinblastine pharmacologically and therapeutically. Vincristine is more effective in acute leukemia and vinblastine in Hodgkin's disease than the other plant alkaloids, colchicine and its derivatives and podophyllotoxin, which also produce metaphase arrest.

Adverse effects: The main adverse effects that can develop is the syndrome of inappropriate secretion of antidiuretic hormone (SIADH).
Microorganism Products

- Antibiotics: Doxorubicin, Bleomycin
- Enzymes: L-Asparaginase

L-asparaginase acts in a unique manner to hydrolyse asparagine to aspartic acid, and neoplastic cells unable to make this amino acid, die if the supply of L-asparagine inthe circulating blood, on which they are dependent,is destroyed by the enzyme. Normal cells synthesize L-asparagine for their needs, and thus appear to be unaffected by the L-asparagine deficiency in the blood stream.
Adverse effects: The main side effect of this agent is a hypersensitivity reaction manifested by fever, chills, nausea and vomiting. Severe cases can present with bronchospasm, respiratory failure, and hypotension.

Miscellaneous:

Miscellaneous drugs is an analog of urea whose mechanism of action involves the inhibition of DNA synthesis in the S phase by inhibiting the enzyme ribonucleotide reductase, resulting in depletion of deoxynucleoside triphosphate pools. Route of administration is mainly orally. It is

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mainly used for the treatment of chronic myelogenous leukemia and blast crisis of acute myeloid leukemia.

- Hydroxyurea
- Imatinib Mesylate
- Rituximab
- Epirubicin
- Bortezomib
- Zoledronic Acid
- Geftinib
- Leucovorin
- Pamidronate
- Gemcitabine

Hormones and Antagonists

The relationship between hormones and hormone-dependent tumors was initially demonstrated in 1896 when Beatson showed that oophorectomy produced improvement in women with advanced breast cancer. Corticosteroids have been useful in the treatment of acute leukemia, lymphoma, multiple myeloma, and other hematologic malignancies as well as in advanced breast cancer.

- Corticosteroids: Prednisone, Dexamethasone
- Estrogens: Ethinyloestradiol
- Antiestrogens: Tamoxifen
- Progesteron derivative: Megestrol Acetate
- Androgen: Testosterone propionate
- Antiandrogen: Flutamide , Bicalutamide
- Aromatase inhibitor: Letrozole , Anastrazole
- 5-alpha reductase inhibitor: Finasteride
- GnRH Analogue: Leuprolide, Buserelin
- Growth Hormone, glucagon and insulin inhibitor: Octreotide

Attempt to Cure or Palliate Cancer Employs 3 Principal Methods:

1) Operation
2) Radiotherapy
3) Chemotherapy

Differing from the radiotherapy and that emphasize on the treatment of local tissues, the chemotherapy is concerned with that of the whole body.

Operation: Cancer surgery or an operation is to repair or remove the part of the body to diagnose or treat cancer Surgery is the oldest type of cancer therapy and remains an effective treatment for many types of cancer today. It is often used to remove all the cancerous tissue. To diagnose cancer a surgeon may remove a small piece of muscle. This is called a biopsy. If the biopsy contains cancer cells, biopsy is a test where they will collect the tissue for the examination it show what type of cancer it is and how slowly or quickly it may grow. Mostly operations are done for the breast cancer, lung cancer, stomach cancer.
Breast cancer surgery is to remove the tumor and some of the surrounding healthy tissue.

Types of breast cancer surgery:

- **Lumpectomy:** This type of cancer surgery removes only a part of the breast. Lumpectomy, also known as breast-conserving surgery, Even though the lumpectomy is the least invasive breast surgery, it can still be very effective.

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• **Partial or segmental mastectomy or quadrantectomy** : Partial or segmental mastectomy or quadrantectomy cancer remove a larger portion of the breast than in the lumpectomy- perhaps a whole segment or quadrant of tissue- in order to eliminate the cancer.
• **Simple or total mastectomy**: Mastectomy is surgery to remove the entire breast.
• **Radical mastectomy:** A radical mastectomy removes the entire breast, underarm lymph nodes of the chest muscle.
• **Modified radical mastectomy**: Modified radical mastectomy is simple mastectomy and removal of axillary lymph nodes.

• Side effects for the breast cancer can cause short-term pain and tenderness in the treated area, surgery involving the lymph node can cause swelling in arm this type of condition is known as lymphedema.

Radiotherapy: Radiotherapy is the use of high energy x-rays to destroy cancer cells while reducing the impact on healthy cells. Radiotherapy is used to treat cancer in many sites of the body. Radiation therapy is synergistic with chemotherapy; it can also be used before or after the chemotherapy.
There are two types of radiation therapy
1. Internal radiation
2. External radiation

Side effects: skin problems, such as dryness, itching, blistering, or peeling.
Fatigue associated with cancer treatment is different from fatigue from lack of sleep

Chemotherapy:
Chemotherapy is a cancer treatment that uses drugs to stop the growth of cancer cells, either by killing the cells or by stopping them from dividing. The way the chemotherapy is given depends on the type and stage of the cancer being treated.

CONCLUSION

The main goal of anti-neoplastic drug is to eliminate the cancer cells without affecting normal tissues. Drug resistance is often associated with loss of p53 function, DNA mismatch repair system, and increased MDR1 gene expression.

REFERENCES

1. Klein C .Chemotherapeutics - Where to Now? Chemotherapy. 2012;1:e101.
2. Maiti AK. Why “Chemotherapy” Should be an Open Access Journal. Chemotherapy. 2012; 1:e102.
3. Wu J Predictive Biomarkers to Therapy, do they Exist in Hepatocellular Carcinoma? Chemotherapy. 2012;1:e104.
4. Becerra-Torres SL, Castillo-Hernández L .The Neurotoxic Effects of 2-Nitropropane on Nerve Conduction are Reversible, In Vitro. Chemotherapy. 2012;1:101.
5. Becerra-Torres SL, Castillo-Hernández L. Effect of 2-Nitropropane on Chemical Neurotransmission, Spontaneous and Evoked, in the Sartorius Muscle of the Frog. Chemotherapy. 2012; 1:102.
6. Maraveyas A, Muazzam IA .Thromboprophylaxis in Pancreatic Cancer: Why isn’t Prime Time Here Compared to Multiple Myeloma?. Pancreat Disord Ther.2015; 5: e137.
7. Pliquett U. Electrochemotherapy – A New Way for Enhancing Cancer Treatment. Chemotherapy. 2012;1:e106.
8. Figueiró Longo JP, Muehlmann LA , Velloso NV, Simioni AR, Lozzi SP, et al. Effects of Photodynamic Therapy Mediated by Liposomal Aluminum-phthalocyanine Chloride on Chemically Induced Tongue Tumors. Chemotherapy. 2012;1:103.

9. Maiti AK. Reactive Oxygen Species Reduction is a Key Underlying Mechanism of Drug Resistance in Cancer Chemotherapy. Chemotherapy.2012;1:104.
10. Pongprasobchai S, Kongkam P, Rerknimitr R. Surveillance of Pancreatic Ductal Adenocarcinoma in Chronic Pancreatitis: An Ongoing Challenge. Pancreat Disord Ther 2015;5: 149.
11. Zang G, Thomas A, Liu Z, Chen D, Ling H, et al. Preventing Breast Cancer Growth by Cationic Cecropin B. Biol Syst.2013; 2: 112.
12. Konishi T. Complimentary Use of Antioxidant Dietary Factor is Promised in Cancer Treatment. Chemotherapy. 2012;1:e107.
13. Putz G. Dosing of Chemotherapeutic Drugs – Time to Leave the Stone Age. Chemotherapy. 2012;1:e108.
14. Johanning GL. Stapled Peptides as Anti-Apoptotic Drugs. Chemotherapy.2012;1:e109.
15. Wang J, Talmadge JE. Targeting Tumor-Related Immunosuppression using Metronomic Cyclophosphamide in Combination with Lenalidomide -New Mechanisms for Old Drugs. Chemotherapy.2012;1:e110.
16. Tu SM. Origin of Cancers: Gene-Centric Versus Cell-Centric. Chemotherapy.2012; 1:e111.
17. Muehlmann LA, de Azevedo RB .On How Could Light and Nanostructures Lead the Way to a Safer Anticancer Therapy. Chemotherapy. 2012; 1:e112.
18. Dobashi Y .Molecularly Targeted Therapy: Great Progress or Evil Cycle. Chemotherapy. 2012;1:e113.
19. Yan M. Cancer Chemotherapy and Cancer Personalized Medicine: An Old Car with a New Engine. Chemotherapy. 2012; 1:e114.
20. Dobashi Y, Goto A, Kimura M,Nakano T. Molecularly Targeted Therapy: Past, Present and Future. Chemotherapy. 2012; 1:105.
21. Loth K, Naik S, Kennedy L, Russell G, Levitan D, et al. Infectious Complications Associated with the Use of Antithymocyte Globulin in Reduced Intensity Allogeneic Transplants. Chemotherapy. 2012; 1:106.
22. Johanning GL. Chemotherapy: Too Much of a Good Thing? Chemotherapy. 2012; 1:e120.
23. Velloso NV , Muehlmann LA, Longo JPF, da Silva JR, Zancanela DC, et al. Aluminum-Phthalocyanine Chloride-Based Photodynamic Therapy Inhibits PI3K/Akt/Mtorpathway in Oral Squamous Cell Carcinoma Cells In Vitro. Chemotherapy. 2012;1:107.
24. Muehlmann LA, de Azevedo RB .There is Plenty of Room at the Bottom for Improving Chemotherapy: Exploiting the EPR Effect with Nanotechnology. Chemotherapy.2012: 1;e116.
25. Rudek MA. Do We Have the Right Dose? Dose Adjustments for Organ Dysfunction. Chemotherapy.2012: 1;e117.
26. Chen Y. NEK1 Protein Kinase as a Target for Anticancer Therapeutics. Chemotherapy.2012; 1:e118.
27. Maiti AK. Elevate the ROS Level to Kill Cancer Cells during Chemotherapy. Chemotherapy.2012; 1:e119.
28. Longo JPF, Muehlmann LA. No Nanoparticle is an Island - the Dynamic Interaction between Nanoparticles and Plasma proteins. Chemotherapy.2013; 2: e123.
29. Wagenlehner FME.New Treatment Strategies in Urinary Tract Infections. Chemotherapy.2013; 2: e124.
30. Berardi R, Santinelli A, Brunelli A, Morgese F, Onofri A, et al. Prognostic Factors in Early Stage Non-Small Cell Lung Cancer: The Importance of Number of Resected Lymph Nodes and Vascular Invasion. Chemotherapy.2013; 2: 120.
31. Hesselink JMK .Palmitoylethanolamide: A Useful Adjunct in Chemotherapy Providing Analgesia and Neuroprotection. Chemotherapy.2013; 2: 121.
32. Guo C, Yang Q, Sai K, Chen Z .Clinical Practice of Chemotherapy with Temozolomide in China. Chemotherapy. 2014; 3: 129.
33. Feng Y, Wang N, Hong M .Cancer Chemotherapy: Time for New Solution. Chemotherapy.2014; 3: 130.
34. Kung HN, Lu KS, Chau YP .The Chemotherapeutic Effects of Lapacho Tree Extract: β-Lapachone. Chemotherapy.2014; 3: 131.
35. Cui Y, Zhuang R, Li Q, Yu S, Yu Y, et al. β-Tubulin is a Predictive Marker of Docetaxel Combined with S-1 in Recurrent or Metastatic Gastric Cancer. Chemotherapy.2014; 3: 132.

36. Zhang XR, Wu D, Liu ZM, Liu XK, Li Q, et al. Influence of Pathologic Complete Response to Induction Chemotherapy on Long-Term Survival of Patients Advanced Squamous Cell Carcinoma of the Oral Cavity Tongue. Chemotherapy.2014; 3: 133.
37. Perkins B, Wu J. Targeting of HER Family Signaling Pathways in Gastric Cancer. Chemotherapy.2014; 3: e125.
38. Schmitt DC, Tan M .The Enemy Within: Regulation of Host Genes by Intronic microRNAs. Chemotherapy.2014; 3: e126.
39. Takeuchi N, Nomura Y, Maeda T, Tada H, Naba K, et al. Complete Response after Treatment with UFT/LV Regimen for Liver and Lung Metastases of Rectal Cancer: A Case Report. Chemotherapy.2014; 3: 122.
40. Fujiwara T, Kawai A, Nezu Y, Fujita Y, Kosaka N, et al. Circulating MicroRNAs in Sarcoma: Potential Biomarkers for Diagnosis and Targets for Therapy. Chemotherapy.2014; 3: 123.
41. Xu Y, Her C. DNA Double-Strand Break Repair in Tumorigenesis and Anticancer Treatment. Chemotherapy.2014; 3: 124.
42. Thomas ML, Coyle KM, Sultan M, Vaghar-Kashani A, Marcato P .Chemoresistance in Cancer Stem Cells and Strategies to Overcome Resistance. Chemotherapy.2014; 3: 125.
43. Liu Y, Sun S, Li J , Yu D .Targeting the PI3K/AKT Pathway for the Treatment of Gastric Cancer. Chemotherapy.2014; 3: 126.
44. Carpenter RL, Lo HW .Regulation of Apoptosis by HER2 in Breast Cancer.J Carcinog Mutagen.2013; S7: 003.
45. Yan B, Broek RV, Saleh AD, Mehta A, Waes CV, et al. Signaling Networks of Activated Oncogenic and Altered Tumor Suppressor Genes in Head and Neck Cancer. J Carcinog Mutagen.2013; S7: 00.
46. Matsuda S, Kitagishi Y. MAGI Scaffolding Molecules Involved in Cancer Cell Signaling.J Carcinog Mutagen.2013; S7: 005.
47. Coyle KM, Sultan M, Thomas ML, Vaghar-Kashani A, Marcato P .Retinoid Signaling in Cancer and Its Promise for Therapy.J Carcinog Mutagen .2013;S7: 006.
48. Volk AL, Johns L, Beck H, McCabe FL, Rafferty P, et al. Tissue Factor (TF) Expression and Angiogenesis in Tumor Progression and Inhibition of Tumor Growth by Anti-TF Antibodies in Human Tissue Factor Knock-In Mice.J Carcinogene Mutagene.2012; S7:001.
49. Gumireddy K, Li A, Cao L, Yan J, Liu L, et al. NOV-002, A Glutathione Disulfide Mimetic, Suppresses Tumor Cell Invasion and Metastasis.J Carcinogene Mutagene.2013; S7:002.
50. Vijaya Krishna V. Stem Cells-Cancer Research. J Carcinogene Mutagene.2011;S1:e001.
51. Quintana AM, Grosveld GC .Zebrafish as a Model to Characterize TEL2 Function During Development and Cancer.J Carcinogene Mutagene.2011; S1:001.
52. Mangum R, Nakano I .Glioma Stem Cells and their Therapy Resistance.J Carcinogene Mutagene.2011; S1:002.
53. Brescia P, Richichi C, Pelicci G .Identification of Glioma Stem Cells: What is Already Known and How Far do We Still Need to Go? The Biomarkers Dilemma.J Carcinogene Mutagene.2011; S1:003.
54. Sun L, Cabarcas SM,Farrar WL . Radioresistance and Cancer Stem Cells: Survival of the Fittest.J Carcinogene Mutagene:2011; S1:0.
55. Sethi S, Sarkar FH .Evolving Concept of Cancer Stem Cells: Role of Micro-RNAs and their Implications in Tumor Aggressiveness.J Carcinogene Mutagene.2011 ; S1:005.
56. Rabindra Kumar J, Sandeep SK, Trupti Rekha S .Cancer Stem Cell – Essence of Tumorigenesis. J Carcinogen Mutagen.2012; S1:006.
57. Jewett A, Nakamura H, Wang M, Teruel A, Paranjpe A,et al. Dedifferentiation of Epithelial Tumors Enhances Cytotoxicity, Survival and Expansion of Allogeneic CD8+ T Cells and Natural Killer Cells. J Carcinogen Mutagen.2012; S1:007.
58. Guo C, Nie D .Are Lipoxygenases Valid Targets of Cancer Prevention and Treatment? J Carcinogen Mutagen.2012; S1:008.
59. Mendes RA .Head and Neck Oncogenesis - An Evolving Conundrum with Molecular Shades.J Carcinog Mutagen.2013; S5: e001.

60. Martins MD, Castilho RM .Histones: Controlling Tumor Signaling Circuitry.J Carcinogene Mutagene.2013; S5: 001.
61. Ekanayaka RP, Tilakaratne WM .Oral Submucous Fibrosis: Review on Mechanisms of Pathogenesis and Malignant Transformation. J Carcinogene Mutagene.2013; S5: 002.
62. Bechet D, Frochot C, Vanderesse R, Barberi-Heyob M .Innovations of Photodynamic Therapy for Brain Tumors: Potential of Multifunctional Nanoparticles.J Carcinogene Mutagene. 2012; S8:00.
63. Grillier-Vuissoz I, Mazerbourg S, Boisbrun M, Kuntz S, Chapleur Y, et al. PPARγ-independent Activity of Thiazolidinediones: A Promising Mechanism of Action for New Anticancer Drugs? J Carcinogene Mutagene.2012; S8:002.
64. Amresh P, Kumar K, Islam A, Hassan I, Ahmad F .Receptor Chemoprint Derived Pharmacophore Model for Development of CAIX Inhibitors. J Carcinog Mutagen.2013; S8: 003.
65. Ali-Boina R, Cortier M, Decologne N, Racoeur-Godard C, Seignez C, et al. Activation of Akt by the Mammalian Target of Rapamycin Complex 2 Renders Colon Cancer Cells Sensitive to Apoptosis Induced by Nitric Oxide and Akt Inhibitor.J Carcinog Mutagen.2013;S8: 004.
66. Dinicola S, Cucina A, Antonacci D, Bizzarri M. Anticancer Effects of Grape Seed Extract on Human Cancers: A Review. J Carcinog Mutagen.2014;S8: 005.
67. Fifis T .Tumor Plasticity Driven by Spatial Micro-heterogeneity in the Tumor Microenvironment Contributes to Therapy Resistance. J Carcinog Mutagen.2014; S8: 006.
68. Kono T, Takeda H, Shimada M, Kase Y, Uezono Y.Novel Therapeutics for Adverse Effects of Antitumor Therapy: The Promise of Multicomponent, Traditional Japanese Herbal Remedies. Carcinog & Mutagen.2014; S8: 00.
69. Rossana B, Mariagrazia LD, Silvia P, Vittorio P, Francesca M, et al. Thymic Malignancies in the Targeted Therapies Era. J Carcinog & Mutagen.2014; S8: 008.
70. Alessandra R, Massimiliano B, Venera C, Laura V, Luca V, et al. Pharmacological Induction of Heme Oxygenase-1 Reduces KB Cell Viability: Role of Carbon Monoxide. J Carcinog & Mutagen.2014; S8: 00.
71. Rahbar A .Promotion of Tumorigenesis and Clinical Implications of Viral Infection in Breast Cancer. J Carcinog Mutagen.2015; 6: 2.
72. Kayar Y, Baysal B, Kayar NB, Kyio NH, Mahdi NM, et al. Hypereosinophilia Induced by Lung Adenocarcinoma: A Rare Case. J Carcinog Mutagen.2015; 6: 216.
73. Arif SH, Pandith AA, Bhat AR, Ramzan AU, Malik Nk,et al. EGFR and PTEN Gene Mutation Status in Glioblastoma Patients and their Prognostic Impact on Patient's Survival. J Carcinog Mutagen.2015; 6: 21.
74. Ahangari G, Pornour M, Aminzadeh S, Bakhtou H, Ahmadkhaniha. HR Significant Association between Catechol Amine O-Methyl Transferase (COMT) Gene Expression Changes and Breast Cancer Pathogenesis. J Carcinog Mutagen.2015; 6: 21.
75. Kuhn E, Tisato V, Rimondi E, Secchiero P .Current Preclinical Models of Ovarian Cancer. J Carcinog Mutagen.2015; 6: 2.
76. Mohanty S, Sahu SK, Chattopadhyay NR, Kumar A, Choudhuri T, et al. TAp63alpha Induced Apoptosis Inhibited by Kaposi's Sarcoma Herpesvirus Latency Nuclear Antigen. J Carcinog Mutagen.2015; 6: 22.
77. Chao T, Xiaowen W, Zhiqi L, Qi Y, Zixiao Y, et al. A Systemic Review of Clinical Trials on Dendritic-Cells Based Vaccine Against Malignant Glioma.J Carcinogene Mutagene.2015; 6: 222.
78. Sennerstam RB,StrombergJO. Genomic Instability or One-Gene Theory for Tumor Progression: A Breast Cancer Study.J Carcinogene Mutagene.2015; 6: 223.
79. Mazilu L, Suceveanu AI, Parepa IR, Tofolean DE .Colorectal Cancer Screening: Is there a Role for Stool DNA Testing? .J Carcinog & Mutagen.2014; S10: 006.
80. Attaallah W, Turkoz K, Yegen C .A Fibrosarcomatous ("High-Grade") Variant of Dermatofibrosarcoma Protuberans (DFSP). J Carcinog Mutagen.2014; S4: 007.