International Journal of Cell Science and Biotechnology
ISSN: 2320 –7574

Available at http://inpressco.com/category/ijcsb

Original Research Article

Molecular genetics in cancer research: current scenario and future prospective

Ashutosh Mangotra[Á], Ekta Singhmar[B] , Rajesh Kumar[Á] , Anil Khuttan[B] and Sanjeev Kumar Gautam[B*]

[Á]Department of Biotechnology Engineering, University Institute of Engineering and Technology, Kurukshetra University, Kurukshetra, Haryana, India.
[B]Department of Biotechnology, Kurukshetra University, Kurukshetra-136119, Haryana, India

Accepted 10 August 2013, Available online 12 December 2013, **Vol.2 (2013)**

Abstract

Cancer as we know that the uncontrolled growth of a cell, is now a day very common and a bigger problem to combat worldwide, accounting for 8.2 million deaths in 2012 according to WHO. Cancer is a generic term for a large group of diseases that can affect any part of the body. The main cause for this deadly disease is found to be in the heart of DNA. The principal targets of genetic alterations may be the four classes of normal regulatory genes-the growth-promoting proto-oncogene, growth-inhibiting tumor suppressor genes, genes that regulate programmed cell death(apoptosis), and genes involved in DNA repair. Now a days, the genetic code seems to be the progressive path to reach our target to cure this disease. Alternatively, the alkaloids of herbs may be the suitable solution. Which treatment is used depends upon the type, location and grade of the cancer as well as the person's health and wishes. As our understanding about the molecular pathways regulating cancer signatures has embossed an equally strong desire to abolish cancer before the resistance, or relapse that continue to worsen survival data of the disease.

Keywords*: Cancer, Genes, Genetic code, Alkaloids and pathways.*

1. Introduction

Cancer, medically known as a malignant neoplasm, is a group of different diseases, all involving unregulated cell growth. In Cancer, cells divide and grow uncontrollably, forming malignant tumors, and invade nearby parts of the body.

2. Description

The cancer may also spread to more distant parts of the body through the lymphatic system or bloodstream. Not all tumors are cancerous. The basic characteristics of cancer is the transmissible abnormality of cells that is manifested by reduced control over growth and function leading to serious adverse effects on the host through invasive growth and metastasis.

Cancer is the *Latin* word for crab. In ancient time people used the word to mean a malignancy, because of the crab –like tenacity a malignant tumor sometimes seems to show in grasping the tissues it invades. Cancer may also be called malignancy, a malignant tumor, or a neoplasm (literally, a new growth). It is a popular, generic term because the actual medical term for cancer is neoplasia which, from the *Greek*, means new formation. Cancers are new growths of the cells in our bodies. Malignant neoplasm refers to the fact that the new growth has virulent or adverse properties that it may display in the body. Through expression of these properties, it can cause destruction of major organs, and in some cases, life threatening disturbances in body function.

One out of every four deaths in the united states is from cancer. It is second most cause of death only to heart disease in united states. Annually, about 1.2 million Americans are diagnosed with cancer; more than 500,000 die of cancer annually.

Cancer can attack anyone during any stage of life but the occurrence of cancer increase as individual age, most the cases are seen in adults, middle-aged or older. Sixty percent of all cases are diagnosed in peoples who are older than 65 years of age. As we know well that our body is made up of cells and cells are the basic unit of life. Throughout people lives, the body cells grow, divide, and replacing themselves. The division process is controlled by our DNA, and in cancer cell DNA get mutated and lose its control over the cell division. The abnormal cell begins to divide uncontrollably and eventually forms a new growth known as a "tumor" or neoplasm. In a healthy individual, the immune system can recognize the malignant cells and destroy them before they start to divide. However, some mutant cells may escape immune detection and survive to become malignancy or neoplasia or cancers.

In a healthy individual, the immune system can recognize the malignant cells and destroy them before they start to divide. However, some mutant cells may escape immune detection and survive to become malignancy or neoplasia or cancers.

*Corresponding author: **Sanjeev Kumar Gautam**

The initial stage of caner does not determined because when cancer begins it invariably produce no symptoms with signs and symptoms only appearing as the mass continues to grow or ulcerate. The findings that result depends on the type and location of the cancer. Few symptoms are specific, with many of them frequently occurring in individuals who have other conditions. Cancer is the new "great imitator". Thus it is not uncommon for peoples diagnosed with cancer to have been treated for other diseases to which it was assumed their symptoms were due (Bast, R.C. *et. al.* 2005).

3. Classification of Cancer

Depending upon the tumorigenecity cancer is of basically three types include; **Benign tumor, malignant** and **Metastatic tumor.** Benign tumor is actually not a cancer. It is the growth of the uncontrolled cells at a specific location. In this type of tumor cells does not spread from one location to another location. Cells remains at the confined area and grow in size at that area only. It does not the main cause of death of peoples suffering from cancer. Another type is metastatic type of cancer. Metastasis also called metastatic disease or mets, is the ability of cancerous cells to spill, leak or break away from their site of origin (pancrease, prostrate, lung or colon), or primary tumor, and enter the blood and lymphatic vessels. In metastatic tumor cells does not confined to specific area. These cancerous cells are deposited within healthy tissues of the body, where they multiply and grow-affecting vital organs. Most malignant cancers are capable of metastasizing. Malignant tumors are also spread by invasion the process in which cancerous cells invade the blood vessels.

3.1 Classification dependent upon the type of cell affected by cancer

Cancers are classified by the type of cell that the tumor cells resemble and is therefore presumed to be the origin of the tumor. These types include: ***Carcinoma*;** Cancers derived from the epithelial cells. This group include many of the most common cancers, particularly in the aged, and include nearly all those developing in the breast, prostrate, lung, pancrease, and colon cancer. ***Sarcoma:*** Cancers of the connective tissue (i.e. bone, cartilage, fat, nerve), each of which develope from cells originating in mesenchymal cells outside the bone marrow. ***Lymphomas and Leukemia:*** These two groups of cancers arise from hematopoietic (Blood forming) cells that leave the marrow and tend to mature in the lymph nodes and blood, respectively. ***Germ cell tumor:*** cancers of the pluripotent cells, most often presenting in the testicles or the ovary (**Seminoma** and **Dysgerminoma**, respectively). ***Blastoma:*** Cancers derived from immature "precursors" cells or embroyonic tissue. These are also most common in children. Cancers are usually named using -carcinoma, -sarcoma or –blastoma as suffix, with the *Latin* or *Greek* word for the organ or tissue of origin as the root. For example, cancer of the liver paranchyma arising from malignant epithelial cells is called a hepatoblastoma, while a malignancy arising from fat cells is called a liposarcoma. For some common cancers, the English organ name is used. For example, the most common type of breast cancer is called ductual carcinoma of the breast. Here, the adjective ductual refers to the appearance of the cancer under microscope, which suggest that it has originated in the milk ducts. Benign tumors (which are not cancers) are named using –oma as a suffix with the organ name as the root. For example, a benign tumor of the smooth muscle cells is called a leiomyoma (the common name of this frequently occuring benign tumor in the uterus is fibroid). Confusingly, some type of cancers also use the -oma suffix, example including melanoma and seminoma. Some types of cancers are named for the size and shape of the cells under a microscope, such as giant cell carcinoma, spindle cell carcinoma, and small cell carcinoma.

4. Molecular/Genetic basis of cancer

Cancer is the general name for over 100 medical conditions involving uncontrolled and dangerous cell growth. Some cancers are caused by genetic factors, while some forms are caused by environmental conditions. The process by which cancer arises is called oncogenesis. Tumors are tissues composed of cells that deviate from normal programming of cell division and differentiation (Cortés, J. *et. al.* 2013). There are three types of tumors: benign, malignant and metastasis tumors which we have discussed earlier and Four major causes:
1). Spontaneous gene or chromosomal mutation, 2). Exposure to mutagens, 3). Exposure to tumor viruses , 4). Genetically inherited mutation.
Tumor is basically arising from the disregulation of cell cycle or the disfunctioning of genes which control the cell cycle. Sometimes during the DNA replication, spontaneously some genes or chromosomes get mutated which may become the cause of cancer. We have found Four classes of normal regulatory genes-the growth-promoting proto-oncogene, growth-inhibiting tumor suppressor genes, genes that regulate programmed cell death (apoptosis), and genes involved in DNA repair are the principal targets of genetic damage. Mutant alleles of proto-oncogene are considered dominant because they transform cells despite the presence of a normal allele. In contrast, both normal alleles of the tumor suppressor genes must be damaged for transformation to occur, so this family of genes is sometimes referred to as recessive oncogenes. However, there are exceptions to this rule, and some tumor suppressor genes lose their suppressor activity when a single allele is lost or inactivated. This loss of function of a recessive gene caused by damage of a single allele is called haploinsufficiency. Genes that regulate apoptosis may be dominant, as are proto-oncogene, or they may behave as tumor suppressor genes.

Mutagen (literally *origin of change*) may be a physical, chemical or biological agent that changes the genetic material, usually DNA, of an organism and thus increases the frequency of mutations in that organism. Mutagen may be carcinogenic as many mutations cause cancer. There are a number of mutagens which may be carcinogenic by

damaging DNA. Lets we discuss some of mutagens which affect the DNA**.**

UV radiations known to acting as a pyrimidine dimer induces error prone repair. It causes mainly purine to purine or pyrimidine to pyrimidine i.e. G-C to A-T transitions, but it also causes other types of mutation including deletions, frameshifts, and rearrangements at somewhat lower frequency.

2-aminopurine (2-AP) is also known as a base analog which causes A-T to G-C and G-C to A-T transitions. Bromouracil is also acting as a base analog which causes G-C to A-T and A-T to G-C transitions.

Hydroxylamine (NH_2OH) is working as an alkylating agent, generates N^4-hudroxycytosine. It causes G-C to A-T transition when used in vitro. ***Ethylmethane sulfonate (EMS)*** are alkylating agents which generatetes O^6-methylguanine. It causes G-C to A-T transition.

Ethylethane sulfonate (DES) also functions as an alkylating agent which induces SOS response. . It causes G-C to A-T transition, other base substitution mutations.

Nitrous acid causes the oxidative deamination and causes G-C to A-T and A-T to G-C transitions, in this deletion produced at a lower frequency.

ICR-191 functions as an intercalating agent, alkylacridine derivative that stabilizes looped out bases by stacking between them and causes frameshift mutations, addition or deletions in runs of G or C.

Cancer causing viruses are called as **oncoviruses**. A number of viruses attack and cause infection in humans but not all the viruses cause cancer because evolution viruses and host occur parallarly (co-evolution). Virus causing cancer may have genome DNA or RNA. The WHO International Agency for Research on Cancer estimated that in 2002, 17.8% of human cancers were caused by infection, with 11.9% being caused by one of seven different oncoviruses. Oncoviruses were discovered by different scientists group at different time.

In **1964,** Anthony Epstein, Bert Achong and Yvonne Barr were the first who identified cancer in humans from Burkitt lymphoma cells (Epstein, M.A. *et. al.* 1964). A herpes virus which is formally known as human herpes virus but more commonly called Epstein-Barr Virus or EBV. In mid **1960s** Baruch Blumberg a Nobel leurate (1976) in Medicine or Physiology, first to isolate and characterized Hepatitis B while at NIH and later Fox Chase Laboratory. Although this agent was the clear cause of hepatitis and might contribute to liver cancer hepatocellular carcinoma. After in **1980** Human T-lymphotropic virus 1 (HTLV I), was the first retrovirus discovered at NIH by Bernard Poiesz and Robert Gallo. Later on Mistuaki Yoshida and coworkers discoverd the same in Japan. From **1984-1986,** Harald zur Hausen, together with Lutz Gissman discovered first HPV16 virus and then HPV18 which is considered responsible for approximately 70% of cervical cancers. In 2008, Harald zur Hausen won Nobel Prize for discovery of human papillomaviruses (HPV). Then in **1987,** Michael Houghton discovered Hepatitis C virus, or HCV, from cDNA library made from diseased tissues for foreign antigens recognized with patient sera at Chiron, a biotechnology company, and at CDC by D.W. Bradley . Controversy erupted with Chiron as work had been performed under contract with the CDC using Bradley's materials and ideas. Eventually, this was amicably settled. Further HCV was found to be a major contributor to liver cancer; worldwide. Patrick S. Moore and Yuan Chang (husband and wife) together with Frank Lee and Ethel Cesarman discovered Kaposi sarcoma-associated herpesvirus (KSHV or HHV8) in 1994. This agent was assumed to be a new virus. In **2008** Chang and Moore,(University of Pittsburgh Cancer Institute) isolate DNA fragments of Merkel cell polyomavirus from a Merkel cell carcinoma. This virus causes 70–80% of these cancers. A new method was also developed by them to identify cancer viruses based on computer subtraction of human sequences from a tumor transcriptome, called digital transcriptome subtraction (DTS). This is the first polyomavirus to be well-established as the cause for a human cancer.

Cancer is not inherited at all but many families have risk of cancer from generation to generation and this type of cancer is called as inherited cancer. Cancer is caused by inheritance of mutated genes. Genes are the segments of DNA, contain instruction for making proteins the body needs to function and how to keep the cells in balance.

In cancer, two major functions are being performed at genetic level. First is of *oncogenes*, which cause cancer. Secondly the *tumor suppressor genes*, stop cancer from developing or growing.

Proto-oncogene is a normal gene which codes for proteins that help to regulate cell growth and differentiation . Proto-oncogenes usually involved in signal transduction and execution of mitogenic signals. When mutation occurs in proto-oncogenes (Weinstein & Joe. 2006), these become tumor inducing agent or oncogenes. Mutations are typically dominant in nature. Upon activation or mutation, oncogenes leads to increased cell division, decreased cell differentiation and inhibition of cell death, ultimately leading to cancer phenotypes.

Tumor suppressor genes are normal genes that often function to restrain or slow down cell growth and division, repair DNA mistakes by preventing the accumulation of mutations in cancer related genes, or tell cells when to die (a process known as *apoptosis* or programmed cell death) to keep cells in proper balance.

In this way, tumor suppressor genes act as "brakes" to stop cells in their tracks before they can take the road to cancer. Given this situation, loss of tumor suppressor gene function can be disastrous, and it often puts once-normal cells on the fast track to disease.

Loss of heterozygosity is a mutational change that lead to the inactivation of the second recessive allele of a tumor suppressor gene. such are mutatuion are loss of function mutations are often point mutation or small deletions that leads to inactive protein which loss its function that

encoded by gene. In this process, a heterozygous cell receives a second mutation in its remaining functional copy of the tumor suppressor gene, therefore it becomes homozygous for the mutated gene.

Normally a person born with healthy genes but some of them can become changed (mutated) over the life span. These changes are known as *sporadic* or *somatic mutations*, i.e. they did not transfered from parents to offsprings (non inheritable). Sporadic mutations are the common cause of most of the cancers. Our environment is the way for the exposures of different thing which can cause mutaions, including cigarette smoke, radiation, hormones, and diet (although in many cases there is no obvious cause). The rate of occurance of cancer is at peak level in the old age because of cell division and cell differntioation are less efficient. When mutated genes are inherited the next cell works with one mutation. This makes it all the easier (and quicker) for enough mutations to build up for a cell to become cancerous. That is why cancers that are inherited tend to occur earlier in life than cancers of the same type that are not inherited.

5. Current treatment available for cancer

5.1 Transitional /conventional ways of treatment of cancer

Actually there is no any permanent treatment for the cancer, although many management option for the cancer exist with the primary ones including: surgery, chemotherapy, radiation therapy, and palliative care (Fairchild, A. *et. al*. 2008). Which treatment is used depends upon the type, location and grade of the cancer as well as the person's health and wishes.

Surgery is the primary method of treatment of most isolated solid cancers and may play a role in palliation and prolongation of survival. It is typically an important part of making the definitive diagnosis and staging the tumor as biopsies are usually required. In localized cancer surgery typically, attempts to remove the entire mass along with, in certain cases, the lymph nodes in the area. For some types of cancers this is all that is needed for a good outcome (Bast, R.C. *et. al*. 2005).

Chemotherapy in addition to surgery has proven useful in a number of different cancer types including: breast cancer, colorectal cancer, pancreatic cancer, osteogenic sarcoma, testicular cancer and certain lung cancers. The effectiveness of chemotherapy is often limited by toxicity to other tissue in body (Mello, H.R. *et. al*. 2008).

Raditation therapy involves the use of ionizing radiation in an attempt to either cure or improve the symptoms of cancer. It is used in about half of all cases and the radiation can be from either internal sources in the form of brachytherapy or external sources. Radiation is typically used in addition to surgery and or chemotherapy but for certain types of cancer such as early head and neck cancer may be used alone. For painful bone metastasis it has been found to be effective in about 70% of people.

5.2 Immunotherapy

Immunotherapy is also an alternative way to treat different types of cancer. For the immunotherapy scientist try to make vaccine which contain may be the cancer antigen or cells that may when introduced in our body help our immune system to wake up or made more antibody against the cancer cells. There are two different types of cancer vaccines are under the research to make. **Specific cancer vaccine** which made against the specific type of cancer. It may cure the skin cancer or may treat the colon cancer or remedy for the breast cancer. A more appealing cancer vaccine would be one that could fight cancer cells regardless of cancer type. This type of vaccine is called a **universal cancer vaccine.**

5.3 Complementary and alternative therapies

Complementary and alternative cancer treatments are a diverse group of health care systems, practices and products that are not part of conventional medicine and substances used along with conventional medicine, while alternative medicine refers to compound used instead of conventional medicine. Most complementary and alternative medicines for cancer have not been rigorously studied or tested. Some alternative treatments have been investigated and shown to be ineffective but still continue to marketed and promoted.

In this context, uses of herbal products have been extensively used in various countries. More than hundred herbal products are available which have been used to treat cancer. In order to check their efficacy it is very much necessary that their mode of action on the tumor cells need to be studied. In India large no. of herbs are available to treat cancer like *tulsi*, *neem* etc. one of them is *podophyllum hexandrum*. This herb is extensively used to treat cancer but its mechanism is at cellular level is almost unknown.

With current research, scientists are finding the ways to use the genetic code of proteins produced in cells to aid the immune systems fight against cancer. Bits of DNA from the patient's cells are injected into the patient, which instructs the other cells to continuously produce certain antigens. This **DNA vaccine** increases production of antigens, which forces the immune system to respond by producing more T cells.

6. Current research on cancer

In light of the research in progress on the benefits of various phytochemicals in foods, it appears feasible that the chemical compounds from herbs also could be helpful in prevention or treatment of cancer and other diseases (Medard, M. *et. al.* 2009).

Current research occurs on the number of compounds which work as an anticancer agent. The active ingredient of spice turmeric, known as Curcumin, is a non-toxic, diferuloyl methane compound that has been shown to have a number of pharmacologic and therapeutic activities including antioxidant, antimicrobial, anti-inflammatory and anticarcinogenic properties (Kim, S.R. *et. al.* 2012).

Both in vivo and in vitro studies have demonstrated the ability of curcumin to effectively inhibit cancer growth. This potent anticancer property of curcumin is related to its ability to simultaneously modulate the functions of a number of different molecular pathways including MAPK, EGFR and NFkB pathways. In addition, curcumin also regulates the nuclear β-catenin/ T cell factor (TCF) transcriptional activity (Zong, H. *et. al.* 2011). However, the precise molecular mechanisms of curcumin mediated suppression of β-catenin transcriptional activity are not fully understood. Modulation of these cellular events by curcumin correlated with decreased cell proliferation, colony formation and cell motility and enhanced cell-cell aggregation in cancer cells (Wang, M.E. *et. al.* 2012). The potent anticancer effects of curcumin in vitro were also reflected in cancer xenograft mouse model. The in vivo inhibition of tumor growth also correlated with enhanced membrane localization of β-catenin.

In the world Tea is the most popular beverage, next to water. Among the polyphenols present in green tea (about 30% of the total weight of tea leaves), a major polyphenol, epigallocatechin-3-gallate (EGCG), has shown inhibitory effects on different stages of tumorigenesis based on *in vitro* experiments and *in vivo* studies using animal models of carcinogenesis (Wang, H. *et. al.* 2011). Anticancer activities attributed to exposure to EGCG include inhibition of cell proliferation and tumor growth, induction of apoptosis and cell cycle arrest, inhibition of invasion and metastasis, and suppression of angiogenesis. At the molecular level, EGCG markedly inhibits the binding of vascular endothelial growth factor (VEGF) with its receptor.

7. Future prospective of cancer treatment

Recently in 2013 there would be an estimated 22,240 new diagnoses and 14,030 deaths from single ovarian cancer in the United States. instead of worthier surgical approach and the novel active drugs that are available today in clinical practice, about 80% of women exhibiting late-stage disease have a 5-year survival rate of only 30%.

We are proposing a novel way in which to study and deal with cancer treatment based on the finding that differences among tumors within the same organ are larger than among tumors with the same molecular aberrations. This novel approach will lead to a change in the therapy of cancer and might accelerate success in the fight against this deadly disease.

Hence, new challenges are required to increase the success level of cancer treatment. In order to fulfill the task, techniques like microarray and proteomic profiling of CSCs will likely lead to identification of new markers, as well as potential therapeutic targets. CSC markers may have prognostic value by allowing assessment of the size of the CSC population within any selective tumor. The approach for patients with cancer may be distinctly upgraded by identifying disease-specific CSCs which are admissible to the development of each subtype of cancer.
The involvement of CSCs in chemoresistance and reappearance opens a new pathway to develop new CSC-specific drug-delivery conjugates in the form of aptamers, miRNA mimics, differentiating agents or targeting peptides/nucleotides. In addition, the application of personalized medicine in the form of a genetic material (DNA or RNA), even though not yet standardized and integrated into the health system for clinical attention, may help individual-specific drug and dose selection resulting in better cancer diagnosis and prognosis (Tomao, F. *et. al.* 2013).

One of the another alternative road to combat with cancer is whole genome sequencing (WGS). Just few years ago, it was cost prohibitive to sequence the full genome of a person, means to throw light upon all 3.1 billion base pairs of a person's DNA. More typically studies involved looking for a handful of specific genetic markers. Now that WGS is so much fast that just in two weeks and so much cheaper to just $4000. now it becomes possible to utilize WGS in cancer research and treatment. One example of a new study just launched using WGS is a study to be conducted at Memorial Sloan Kettering, UCSF, Vanderbilt and other institutions initially focusing on melanoma.

An alternative approach to combat with cancer in future may be targeted drug therapy. Targeted therapy drugs sometimes work when chemo drugs don't, and they often have different side effects (Widakowich, C. *et. al.* 2007).

Recently research has to be focused on the epidermal growth factor receptor (EGFR), a protein found on the surface of cells in most lung cancers.

EGFR is targeted by drugs developed for non-small cell lung cancer, but the success rate of these drugs is only 10%-15% in patients whose onco cells have mutations in the EGFR gene. These drugs can be very helpful for a time, but unfortunately they eventually stop working in most people. New directions are trying by the researchers to fight with this newly arrived problem.

The results for one such drug were presented in October at the International Conference on Molecular Targets and Cancer Therapeutics in Boston. In the trail of AZD9291 onto mice, the drug reduced lung tumors that were resistant to current EGFR-targeting drugs, as well as some that weren't. Now the trial of AZD9291 is under process in humans, and early results have been promising.

ALK is another drug discussed at the conference is also being tested against non-small cell lung cancer with a mutation in a gene. it is found that just 5% of people have this gene defect in non-small cell lung cancer. The drug named crizotinib (Xalkori), approved for use in 2011, is often helpful against these cancers, but eventually most of them start growing again.

Researchers are also working on lab tests to help predict which patients might be helped by which targeted drugs. Predicting who might benefit could save some people from trying treatments that are unlikely to work for them and would probably cause unneeded side effects.

8. Advancements in immune therapies

A new way should be developed to treat cancer by the development of drugs that help body immune system to fight with cancer. As we know, generally cancer stem cells

express a protein called PD-L1 that help cancer cells to escape from body immune system particularly from T cells. new drug that freeze the PD-L1 protein, on immune cells called T cells, can help the immune system to recognize the cancer cells and attack them.

Earlier, an anti-PD-1 drug named nivolumab (BMS-936558) reduce tumors in about 1 out of every 5 people with non-small cell lung cancer, while a drug targeting PD-L1 (BMS-936559) shrank tumors in about 1 out of 10 people. Extensive research work now being done upon these drugs.

At Amsterdam in September 2013 at the European Cancer Congress an data was presented on study of another anti-PD-L1 drug, MPDL3280A . in smokers with lung cancer this drug provide satisfactory results. Genetic mutations are higher in smokers than lung tumors in non smokers, hence immune system may respond better when PD-L1 is blocked.

Against different types of cancer a number of vaccines for boosting the body immune system are also under the clinical trial. Vaccines are designed in such a way so that they eradicate cancer not prevent. Most of the caner curing drugs have a lot of side effects and these treatments seem to have very limited side effects, so they might be useful in people who can't tolerate other treatments. At this time, vaccines are only available in clinical trials. One of the goals of ongoing studies is to determine whether any of the vaccines help people live longer (Simon, S. 2013).

The 76 suggestions implemented in 2013 in *Clinical Cancer Advances* report presenting curious developments in cancer research. It is what these advances represent collectively that gives tremendous hope to anyone who receives a cancer diagnosis in the future. Three areas of progress, in particular, provide important new insights into cancer care:

Genomics -This area of research demonstrated how to understand the genetic basis of cancer is already helping doctors make better, more singular treatment to particular type of cancer, as well as how it will guide future drug discovery and development.

Treatment-resistant cancers - First in the history the new research discovered first time effective therapy for cancer that has been resistant to drugs.

Targeted immunotherapy - in this we make therapies which can use own body immune system fight with that of cancer cells with help of T cells and provide better treatment idea for cancer. (Patel, J.D. *et. al.* 2013).

Acknowledgement

We are thankful to the Department of Biotechnology Engineering, UIET and Department of Biotechnology, Kurukshetra University, Kurukshetra for providing necessary facilities for carrying out research and writing this review.

References

Bast, R.C., Badgwell, D., Lu, Z., Marquez, R., Rosen, D., Liu, J., Baggerly, K.A., Atkinson, E.N., Skates, S., Zhang, Z., Lokshin, A., Menon, U., Jacobs, I., Lu, K. 2005. New tumor markers: CA125 and beyond. *Int J Gynecol Cancer*, Suppl 3:274-81.

Cortés, J., Calvo, E., Vivancos, A., Perez-Garcia, J., Recio, J.A., Seoane, J. 2013. New approach to cancer therapy based on a molecularly defined cancer classification. *American Cancer Society, Inc*, DOI: 10.3322/caac.21211.

Epstein, M.A., Achong, B.G., Barr, Y.M. 1964. Virus particles in cultured lymphoblasts from burkitt's lymphoma. *Lancet* , 1(7335):702-3.

Fairchild, A., Harris, K., Barnes, E. 2008. Palliative thoracic radiotherapy for lung cancer: a systematic review. *Journal of Clinical Oncology*, 26 (24): 4001–4011.

Kim, S.R., Park, H.J., Bae, Y.H., Ah, S.C., Wee, H.J., Yun, I., Jang, H.O., Ba, M.K., Ba, S.K. 2012. Curcumin down regulated visfatin expression and inhibits breast cancer cell invasion. *Endocrinology*. 153(2):554-563.

Mello, H.R. and Callagha, R. 2008. Resistance to chemotherapy in cancer: A complex and integrated cellular response. *Pharmacology*. 81: 275-300.

Medard, M., Petersen, M., Pra, N., Woolley, J.G. 2009. Plant cell factories as a source for anticancer lignans. *Phytochem Rev*. 1:27–35.

Patel, J.D., Krilov, L., Adams, S., Aghajanian, C., Basch, E., Brose, M.S., Carroll, W.L., de Lima, M., Gilbert, M.R., Kris, M.G., Marshall, J.L., Masters, G.A., O'Day, S.J., Polite, B., Schwartz, G.K., Sharma, S., Thompson, I., Vogelzang, N.J., Roth, B.J. 2013. Clinical Cancer Advances 2013: Annual Report on Progress Against Cancer From the American Society of Clinical Oncology. *Journal of Clinical Oncology*, DOI: 10.1200/JCO.2013.53.7076.

Simon, S. 2013. Researchers Closing in on the Future of Lung Cancer Drugs.

Tomao, F., Papa, A., Rossi, L., Strudel, M., Vici, P., Russo, G.L., Tomao, S. 2013. Emerging role of cancer stem cells in the biology and treatment of ovarian cancer: basic knowledge and therapeutic possibilities for an innovative approach. Journal of Experimental & Clinical Cancer Research, 32:48 doi:10.1186/1756-9966-32-48.

Weinstein, I.B., Joe, A.K. 2006. Mechanisms of disease: Oncogene addiction--a rationale for molecular targeting in cancer therapy. Nat Clin Pract Oncol., 3(8):448-57.

Wang, H., Bian, S., Yang, C.S. 2011. Green tea polyphenol EGCG suppresses lung cancer cell growth through upregulating miR-210 expression caused by stabilizing HIF-1. Carcinogenesis. 32(12):1881-1890.

Wang, M.E., Chen, Y.C., Chen, I.S., Hsieh, S.C., Chen, S.S., Chiu, C.H. 2012. Curcumin protects thioacetamide induced hepatic fibrosis by attenuating the inflammatory response.

Widakowich, C., Castro, G., Azambuja, E., Dink, P., Awada, A. 2007. Side effects of approved molecular targeted therapies in solid cancers. Oncology.12: 1443-1455.

Zong, H., Wang, F., Fan, Q.X., Wang, L.X. 2011. Curcumin inhibits metastatic progression of breast cancer cell through suppression of urokinase-type plasminogen activator by NF-kappa B signaling pathways. Mol Biol Rep.