1. INTRODUCTION

1.1. Cancer

Cancer has a great impact on humans and proved to be a feared disease. It is a term for group of more than 100 diseases affecting different parts of the body; in which abnormal cells grows uncontrollably by not following the rule of normal cell division; invasion of adjoining tissues of the body and crawled to different body organs; the latter process is assigned as “metastasis” a serious phenomenon of death with cancer. The prevalence of cancer is rising day and day and is expected to increase by 50% in coming few years (Stewart, 2002). An uncontrolled cell growth results into cancer (neoplasm) and the name of the disease came from the crab, karkinos in Greek and cancer in Latin (http://www.creatingtechnology.org). In cancer, body cells stops obeying the normal cell signals.

Many vital requirements and properties of normal cells are still shared by abnormal cells but they show lack of controls which make our body function normal. The time taken in a process, in order to change a normal functioning cell into abnormal cell may be long and commonly influenced by outside factors (cancerquest@emory.edu.). The cancerous cell originates from the normal body cells, in which DNA is damaged and can be repaired in most of the cases or cells with damaged DNA gets died but unfortunately it is not done in case of cancer (Akulapalli Sudhakar, 2009). In cancer, cell approaches hayflick limit i.e. number of times a normal cell population will distribute before it cease to happen, presumably because the telomeres reach a length that is having potential to become disastrous (Hayflick, 1961; 1965).

Typical somatic (living) cells do not intimate telomerase, and replicative life-time of such cells is restricted to 30 cell divisions or in the neighborhood of it due to ongoing destruction of telomere repeats i.e. for somatic cells hayflick limit ~ 30 cell divisions (Braundwald, 15th edition). Tumor masses thus result from the clonal evolution of a single progenitor cell that has incurred genetic vandalism. The term "tumor" actually means "swelling", while on the other hand "benign
tumor” can be defined as abnormal tissue masses, non-cancerous and contain independently proliferating cells. Therefore, tumors can be categorized into two classes on the basis of their anatomical features as benign tumor and malignant tumor.

**Benign tumor**

- They remain stationary to their site of origin because they are surrounded by fibrous cap that prevents their ability to act in a malignant way.
- Examples of benign tumors are tumors of endocrine tissues, which results in excessive production of some certain hormones like thyroid adenomas, pituitary adenomas and adrenocortical adenomas).

**Malignant tumor (cancerous)**

- Metastasize i.e. cancer cells move from the originating tumor site to further parts of the body through bloodstream or lymphatic system to form secondary tumors.
- Common examples for malignant tumors include leukemia, renal cell carcinoma, rhabdomyosarcoma, hepatocellular carcinoma, and mesothelioma.

The term ‘malignant disease’ describes a diverse range of illness involving lung, breast and colorectal cancer which are common one among malignancies as well as rare one such as acute leukemia. Besides cardiovascular disease, another utmost cause of death is considered to be cancer. It has been established that mostly human neoplasms originate from mutations in DNA within a single population of precursor. The most commonly affected genes are those which play vital role in cell cycle check points, apoptosis, and differentiation and growth signaling (*Parveen Kumar, 7th edition*). The primary types of cancer which acts as a key to all embracing cancer related to loss of life are (*WHO Cancer* http://www.who.int/mediacentre/factsheets/fs297/en/):

- Lung (~1.3 million)
- Abdomen (~803,000)
- Colorectal (~639,000)
- Hepatic (~610,000)
- Mammary gland (~519,000)
- Pancreatic cancer (~417,780)
Different factors like lifestyle, genetic variation, infection due to many viruses and chronic inflammation are considered to be responsible for the occurrence of cancer (Wang, 2012). Simultaneously different treatments also came into existence for its control. The drugs used to treat cancer are grouped as cytotoxic (cell killing) and the second is cytostatic (cell stabilizing) drugs. Around 50 years ago the action of nitrogen mustard on hematopoietic cells initiated the discovery of anticancer agents. The major categories of anticancer drugs are polyfunctional alkylating agents (cisplatin, thiotepa etc), antimetabolites (5-fluorouracil, methotrexate), antitumor-antibiotics (daunorubicin, mitomycin-c etc), specific mitotic inhibitors (vincristine, vinblastine), Corticosteroids (prednisone, dexamethasone) & miscellaneous group of drugs (Chabner, 1996).

Cancer can be treated by different processes like:
- Gene Therapy
- Immunotherapy
- Biomarkers in cancer detection
- Radiation Therapy
- Chemotherapy
- Bone Marrow Transplantation
- Hormone Replacement Therapy
- Surgery and Laparoscopy
- Molecular Targeted Therapies
- Natural Therapy and Acupuncture

Novel approaches to anticancer drug design include targeting DNA transcription / replication and cell-division at various stages (Zewail-foote, 2004). Prominent amongst the specific targets are telomerase or telomere maintenance apparatus, mitotic apparatus, histone deacetylases, cyclin dependent kinases, choline kinase, p-glycoprotein, glyoxalase-1, EGF receptor, farnesyl transferase, VEGF, and DNA – topoisomerases.

The development of new blood vessels from already existing microvasculature termed as angiogenesis is a complex process that normally occurs during wound healing, organ regeneration and the female reproductive cycle. The vascular endothelial growth factor (VEGF) is tangled in angiogenesis and is a key process for progression of cancer.
TNP470 (= AGM-1470) (Ingber et al., 1990; Corey et al., 1994) binds to and inhibits type 2 methionine aminopeptidase (MetAP2) (Griffith et al., 1997; Sin et al., 1997) and impedes with amino terminal action of methionine, which results into denaturation of enzymes that are vital for growth and movement of endothelial cells (Antoine et al., 1994; Turk et al., 1999) and hence act as anti-angiogenic drugs. Bevacizumab is anti-VEGF monoclonal antibody used in biological therapy to fight cancer. It inhibits VEGF-A is a chemical signal that stimulates the angiogenesis. Hence, bevacizumab has demonstrated activity in ovarian cancer (Konner et al., 2007) and in brain tumour (Cloughesy et al., 2010).

Phosphatidyl-inositol-3-kinases (PI3Ks) represents a lipid kinase, the PI3K/Akt pathway is a key regulator of survival throughout the time of cellular stress (Datta, 1999). Neoalbaconol, (I) obtained from Albatrellus confluens, modulate cell metabolism by attacking 3-phosphoinositide-dependent protein kinase 1 and restrain the growth of cancerous cells (Xiang jian Luo1, 2015). Plumbagin (2) from Plumbago zeylanica decreases the level of PI3K subunit p85 causing downstream AKT/mTOR pathway obstruction resulting into growth arrest and ultimately cell death (Kuo, 2006; Ahmad, 2008).

Telomeres are (TTAGGG) DNA–protein complexes at the ends of chromosomes which are essential for existence of cancerous cells. Partial replication of the lagging strand of DNA along with other processes leads to shortening of telomeres simultaneously with each cycle of cell division. This reduction has restrictive effect on cell division and can be conquered by the expression of telomerase (Gellert, 2005). Telomeric DNA consist of a fluctuating number of G-rich, non-coding, tandem repeats (at birth time in humans 10–15 kilobases (kb) long) of double stranded DNA sequence,
5′-(TTAGGG)n-3′, chased by a terminal 3′ G-rich single-stranded overhang (150–200 nucleotide long) which in turn helps telomeric DNA in constructing a large structure. It comprises 3′ single-stranded which invades the homologous double-stranded TTAGGG region, producing a telomeric loop (T-loop) that allocates 3′-end protection by seizing it from recognition by the DNA damage response machinery. Shelterin complex, are the proteins associated with telomere and are made up of three core shelterin subunits: TRF1, TRF2, and POT1. The first two directly recognize and bind to duplex TTAGGG repeats and third one identifies and bind to single-stranded TTAGGG overhangs (Doksani, 2013).

Telomerase activity is involved in most of the cancers and often harmonizes with the attainment of a more malignant phenotype. Telomere length is also typically shorter in tumor cells as compared to adjoining noncancerous cells. Epigallocatechin gallate (3), Apigenin (4) (Menichincherieta, 2004; Harley, 1991; Shay, 1991) and MST-199 (5) (Seimiya, 2002), are the compounds which have been found to show significant telomerase inhibitory activity.

EGFR represents a class of ErbB comprising of tyrosine kinase receptors modulating cell extension, existence, relocation, union and divergence (Markovic, 2012). This family is comprised of four related receptors: the epidermal growth factor receptor itself four receptors: ErbB-1 (EGFR), ErbB-2 (HER2 or Neu), ErbB-3, and ErbB-4 (Carraway, 1994; Van der Geer, 1994). A growth-inducing signal is emitted by EGFR to cells and they get accelerated by an EGFR ligand (TGFα and EGF) (Carpenter, 1990; Alroy, 1997).
Together EGFR and ErbB-2 have been chosen for the generation of inhibitors (Seymour, 2003) and research has reached a pinnacle of acceptance that gefitinib (6) and erlotinib (7) are used for the treatment of lung cancer. Lapatinib (8) (Xu G et al., 2008) having a strong effect as EGFR/ErbB-2 inhibitor is under Phase II and III trials for the treatment of solid tumors other than breast cancer instead it is best for the treatment of metastatic ErbB-2 positive breast cancer. Cetuximab, monoclonal antibody targeting EGFR, discovered to induce programmed cell death and helpful in colorectal cancers (Ciardiello, 1999; Erlichman, 2004; Liu, 2000).

The topological state of DNA is controlled by nuclear enzymes topoisomerases (Champous, 2001; Wang, 2002) by the mechanism of nicking and resealing (Vosberg, 1985) through a transient single (topo I) and double (topo II) strand break. Topoisomerase inhibitors stabilize the covalent linkage between an enzyme and DNA or by stopping the catalytical reactions. Consequence of their vital role in basic cellular functions, this group of enzymes has now been known as the molecular targets for numerous antibiotics and anticancer drugs. They are referred to as topoisomerases as they are capable of changing the topology of DNA molecules without causing any change in the underlying chemical framework of the DNA. These enzymes alter nucleic acid topology by provoking transient breaks in the sugar-phosphate backbone of DNA. To sustain the cohesion of the genetic matter during this process the covalent bonds formed
by topoisomerases with the newly inaugurated DNA terminal, which is a defining feature of all topoisomerases (Holden, 2001).

Topo-I and Topo-II are the chief forms of topoisomerase enzymes which occur in all the cells and are categorised on the basis of their physical and mechanical properties. Topoisomerase-I relax the positive helical tension and topo-II wrap the DNA around the protein histone. Camptothecin (9) (Garg, 1987) is a quinoline-based alkaloid obtained from the bark of _Camptotheca acuminata_ is an efficient topoisomerase-I inhibitor. Irinotecan (10) (CPT-11) and topotecan (11) are the derivatives of camptothecin which holds good cytotoxic effect (Pommier, 2006).

Topoisomerase-II inhibitors include etoposides and doxorubicin. Topo II poisons create lesions to DNA and also to proteins which are covalently bound to DNA, efficiently blocking transcription and replication, and thus having intense effects on cells, which subsequently undergo apoptosis.

Cyclin-dependent kinases [CDKs] regulate the transition of cell cycle and RNA transcription. CDK/cytokins interferes with phosphorylation of substrates present in DNA replication, chromatin condensation, assembly of the mitotic spindle and disassembly of the nuclear envelope. Palbociclib (12) (PD-0332991) is a potent and highly selective, reversible, oral inhibitor of cyclin-dependent kinases 4 and 6 which stops DNA synthesis
due to blockade of progression of G1- to S-phase of the cell cycle (Fry, 2004; Toogood, 2006; Saab, 2006).

Cyclin-dependent kinases play a crucial role in the succession of the cell cycle from the initial growth phase (G1) via DNA synthesis (S) phase, then to second growth phase (G2), and finally, mitosis, or (M) phase. In early G1, the phosphorylation of the retinoblastoma tumor suppressor protein (Rb), is imitated by activation of CDK4-cyclin D and CDK6-cyclin D protein complexes partially releasing the E2F family of transcription factors to allow the production of cyclin E in mid to late G1. CDK2 kinase activity increases by interacting with cyclin E, which directly hyper phosphorylates retinoblastoma, thereby further releasing the E2F transcription factors which starts the transcription of S phase genes (Geng, 2003; Su, 2004). Interaction of CDKs with cyclins (CDK-cyclin complex) is first step in initiating enzyme activity. Afterwards the CDK activation segment undergoes phosphorylation at a conserved threonine residue as catalyzed by CDK7 for the full expression of CDK-cyclin enzyme activity.

A number of cyclin-dependent kinases (CDKIs) with different mechanisms of action have been evaluated. CDK inhibitors can regulate uncontrolled cellular proliferation. INK4 and CIP/KIP are the two families of CDK-inhibitors (Harper, 1997). The later one is composed of proteins which are three in number and act to interact with other cyclin families (Dai, 2003). CDK inhibitor roscovitine (13) (ROSC) arrests human ER-alpha positive MCF-7 breast cancer cells in the G2-phase of the cell cycle (Wojciechowski, 2003). The INK4 carries p15, p16, p18, and p19 that inhibit CDK4 and CDK6, whereas the proteins p21, p27, and p57 of KIP family exhibit vast CDK inhibitory activity (Canepa, 2007).
Ras proteins are members of GTPases family and mainly four human Ras proteins H-Ras, 2 splice variants of K-Ras, and N-Ras are known. These proteins participate in protein synthesis and signal transduction (Boguski, 1993). An activation/deactivation cycle of exchange of GTP for GDP and following GTP hydrolysis results in regulation of Ras. Ras function is interrupted by the inhibition of farnesyl transferase. This inhibitory enzyme (FTIs) is involved in the coupling of a isoprenyl (15-carbon) group to Ras proteins. The signal transduction pathway accomplished with cessation of cell growth is blocked by Ras farnesylation inhibition. FTIs are good therapeutic agents in the treatment of cancer as it prevents the coupling of farnesyl to Ras protein. The membrane localization of Ras is prevented by inhibiting the Zn^{2+} metalloenzyme farnesyl transferase (FTase) and represents a valid target for the inception of novel cytostatic anticancer drug (Bell, 2004).

Thus, FTIs have been flourished as a class of extremely promising drugs for the treatment of cancer. The reason behind this is may be either the rational design for the structure of the CA1A2X carboxyl terminus of Ras or natural products (Ohkanda, 2002; Haluska, 2002). Farnesyl transferase inhibitors (FTIs) are a class of drugs initially generated to interfere with the farnesylation of oncogenic Ras, thereby retaining it in the cytosol and preventing its activity. The Ras proto-oncogenes plays essential role in cellular signal transduction pathways (Johnston and Kelland, 2001) and also provides a rational target for the treatment of malignances.

The development of several structurally different FTIs as anticancer agents was based on the initial observation that the phenotype of oncogenic Ras-transformed fibroblasts could be reversed by FTI treatment (Kohl, 1993). The farnesyl transferase
inhibitors are evaluated in phase II/III of clinical trials (Baum, 2003; Coponigro, 2003) are R-115777 (14) for colorectal cancer and BMS-214662 (15) for leukemia.

Different target sites involved in cancer treatment

The greatest modalities of medical treatment (pharmacotherapy) for cancer is molecularly targeted therapy, which is possible by using the substances that can obstruct the propagation of cancer by impeding with molecular targets, which are vital for the cancer flourishment. So the main requirement is the recognition of targets. The molecules which are specifically essential for carcinogenesis and tumor growth are blocked by targeted therapy and hence the growth of cancer cells is prevented.

- Small molecule drugs
- Tyrosine-kinase inhibitors
- Therapeutic monoclonal antibodies
- Hormone therapies
- Implications of targeted therapy
- Targeted cancer therapy & health economics

These drugs are now a component of therapy for many common malignancies, including breast, lung, colorectal and pancreatic cancers, along with lymphoma, and multiple myeloma. The mechanisms of traditional cytotoxic chemotherapy are dissimilar to the action and toxicities of targeted therapies. The mechanism for traditional cytotoxic chemotherapy involves the inhibition of cell division which is contradictory to targeted therapy that works on the principle of interrupting specific molecules required for tumor development and growth (David, 2008) and then blocks the proliferation of cancer cells.
The various ways to identify and then validating new targets includes the method to compare the amount of protein in normal cell and in cancerous cell such as human epidermal growth factor receptor 2 protein HER-2 (trastuzumab).

Fig. 1.1: Genes involved in potential targets for drug discovery

The second approach involves the determination of cancer cells to produce altered (mutant) proteins that spread cancer at high rate such as BRAF. Simultaneously a number of oncogenes identified through high genome sequencing for mutations include EFGR (lung cancer), JAK 2 (myeloproliferative disorder), FGFR2 (endometrial cancer) etc. Various classes of a gene participates in malignancy and depict how to modulate various biochemical pathways by selecting different targets that are hijacked by cancer genes are shown in Figure 1.1.
1.2. Antioxidants

Free radicals are unstable, charged molecules in the cells. Free radical and reactive oxygen species are involved pathophysiology of diseases in humans due to an imbalance between formation and neutralization of pro-oxidants resulting in oxidative stress. They cause oxidative damage to lipids, proteins, and DNA, and in due course lead to multiple chronic diseases, like cancer, diabetes, aging, and other degenerative disorders of humans (Harman, 1995) along with lipid peroxidation. Oxidative stress is leading cause for most of the cancer cells that are linked with refine redox regulation of cellular signalling pathways and hence oxidative stress is considered to be involved in diverse types of cancers as it causes the oncogenic stimulation (Miranda-Vilelaa, 2011).

Free radicals especially reactive oxygen species are formed spontaneously in the body when an atom or a molecule either loses or gains an electron. Free radicals play a paramount role in many normal cellular processes (Setorki et al., 2013; Nasri, 2013). However, they can damage all major components of cells incorporating proteins, DNA, and cell membranes at high concentrations. These damages to cells, particularly the damage to DNA, can play a significant role in the expansion of cancer (Ardalan et al., 2014). Besides endogenous antioxidants (inside body), exogenous sources of antioxidants are required for the protection against ROS and free radicals. Exogenous antioxidants can prevent free radical induced damage associated with cancer development (Nasri et al., 2015; Rafieian-Kopaei 2013; Ardalan et al., 2013; Baradaran et al., 2014). An antioxidant is a molecule that discourages the oxidation of other molecules by donating electrons to free radicals, which neutralizes and stables them in the body by efficient antioxidant enzymes (superoxide dismutase, catalase, and glutathione peroxidase) and by the nutrient-derived antioxidant (vitamin E, vitamin C, carotenes, flavonoids, glutathione) and prevents them from causing harm. Thus, antioxidants act as line of defense against overwhelming assaults of free radicals on healthy cells. Antioxidants are constantly involved in the arrest of cellular damage, the leading pathway for variety of diseases (cancer, aging). Different classes of anti-oxidants from natural sources like vitamin C (16) and others have
been explored such as phenolics as cinnamic acid (17) hydrobenzoic acid (18), tetraterpenes and carotenoids like lycopene (19), β-carotene (20) have been shown to possess potent antioxidant activity within both *in vivo* and *in vitro* studies (*Palozza, 1992*). Vitamins like Vit. E (21), saponins and steroid viz. cortisone (22) and estradiol (23), is also implicated as antioxidant in several disorders.
1.3. Antimicrobials

Infectious ailments are another major reason for death among population globally. Most of the infections came into existence by microorganisms that are multi-resistant resulting in difficulty in treating diseases and, accordingly, significant rise in the economy of healthcares. The easily access to various antimicrobial agents and an immense service in various sectors have strongly contributed in progression of the antimicrobial resistance.

Another driving factor for the renewed attention towards antimicrobials from plant origin is due to the extinction of plant species at rapid rate (Lewis, 1995). The microbiologists along with natural-products chemists feel that number of potentially functional phyto-chemical constituents which could be synthesized chemically is at higher risk to be lost forever (Borris, 1996). Ethnopharmacology, is a branch of science, utilizes group of information gathered by peoples about the indigenous plant and animal products and can be used to maintain their health conditions (Georges, 1949; Rojas, 1992; Silva, 1996; Vanden Berghe, 1986).

Recent advancements in natural products research have established a significant growth in the breakthrough invention of novel scaffolds which have significant antimicrobial activity. In fact, nature is a kind of treasurer from where number of molecules has been obtained to treat diseases, including infectious diseases. Some of the resources are like medicinal plants, marine and terrestrial organisms, including fungi and bacteria. Even there is still a diverse fauna and flora which waited to get explored, could supply antimicrobial leads and new drugs. Further investigations in this field is required to explore thousands of still untouched natural substances which can act as lead compound for discovery of new compounds in the area of antimicrobial agents and other.

The plants have an immense power to synthesize diverse aromatic/heterocyclic molecules bearing phenolic or derived from the substitution at oxygen atom (Geissman, 1963). Most of the secondary metabolites (~12,000) have been isolated which is estimated to be less than 10 % of the total constituents (Schultes, 1978). The natural products or constituents also have plant protection mechanisms in themselves against the attack by microorganisms, insects and
herbivores. For example, some terpenoids extend out odors to plants; pigmentation to plant is provided by the quinones and tannins. Other compounds are accountable for plant flavor (capsaicin from chili peppers), and few of the similar herbs and spices used by humans to garnish food yield useful medicinal compounds. The efficacious antimicrobial phytoconstituents can be grouped into several categories, mentioned and summarized in table 1.1.

**Table 1.1. Antimicrobial phytoconstituents with their mechanism**

<table>
<thead>
<tr>
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<th>Sub class</th>
<th>Example</th>
<th>Mechanism</th>
<th>References</th>
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<tbody>
<tr>
<td>Phenolics</td>
<td>Simple phenols</td>
<td>Catechol</td>
<td>Substrate deprivation</td>
<td>Peres <em>et al.</em>, 1997</td>
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<td>Epicatechin</td>
<td>Membrane disruption</td>
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<td>Phenolic acids</td>
<td>Cinnamic acid</td>
<td>Bind to adhesins</td>
<td>Toda, 1992</td>
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<td>Quinones</td>
<td>Hypericin</td>
<td>Complex with cell wall</td>
<td>Duke, 1985;</td>
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<td></td>
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<td></td>
<td>Inactivate enzymes</td>
<td>King, 1994</td>
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<td>Flavonoids</td>
<td>Chrysin</td>
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<td>Perrett <em>et al.</em>,</td>
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<td>Abyssinone</td>
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<td>Ono <em>et al.</em>, 1989</td>
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<td>Complex with cell wall</td>
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<td>Complex with cell wall</td>
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<td>Keating, 1997; Hoult, 1996</td>
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