INTRODUCTION:

Cancer is a group of diseases characterized by uncontrolled growth and spread of abnormal cells. Cancer is caused by both external factors (tobacco, infectious organisms, chemicals, and radiation) and internal factors (inherited mutations, hormones, immune conditions, and mutations that occur from metabolism). Ten or more years often pass between exposure to external factors and detectable cancer. Cancer is treated with surgery, radiation, chemotherapy, hormone therapy, biological therapy, and targeted therapy. The American Cancer Society estimates that in 2012 about 173,200 cancer deaths will be caused by tobacco use. Many of the more than 2 million skin cancers that are diagnosed annually. Cancer researchers use the word “risk” in different ways, most commonly expressing risk as lifetime risk or relative risk.

The National Cancer Institute estimates about 1,638,910 new cancer cases are expected to be diagnosed in 2012. In 2012, about 577,190 Americans are expected to die of cancer, more than 1,500 people a day. Cancer is the second most common cause of death in the US. By 2030, the global burden is expected to grow to 21.4 million new cancer cases and 13.2 million cancer deaths simply due to the growth and aging of the population, as well as reductions in childhood mortality and deaths from infectious diseases in developing countries.

<table>
<thead>
<tr>
<th>Table 1: Cancer facts and figures according to National cancer Institute (NCI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Estimated New Cancer Cases and Deaths by Sex, US, 2012</strong></td>
</tr>
<tr>
<td>Estimated New Cases</td>
</tr>
<tr>
<td>Both Sexes</td>
</tr>
<tr>
<td>Breast</td>
</tr>
<tr>
<td>Prostate</td>
</tr>
<tr>
<td>Lung &amp; bronchus</td>
</tr>
<tr>
<td>Colon</td>
</tr>
<tr>
<td>Ovary</td>
</tr>
<tr>
<td>Liver &amp; intrahepatic bile duct</td>
</tr>
<tr>
<td>Small intestine</td>
</tr>
<tr>
<td>Brain &amp; other nervous system</td>
</tr>
<tr>
<td>Skin (excluding basal &amp; squamous)</td>
</tr>
<tr>
<td>Melanoma-skin</td>
</tr>
<tr>
<td>Pancreas</td>
</tr>
<tr>
<td>Urinary bladder</td>
</tr>
<tr>
<td>Kidney &amp; renal pelvis</td>
</tr>
<tr>
<td>Genital system</td>
</tr>
<tr>
<td>Lymphoma</td>
</tr>
<tr>
<td>Myeloma</td>
</tr>
</tbody>
</table>
The primary modalities of cancer treatment are surgery, chemotherapy, and radiotherapy; these may be used alone or in combination. Surgical treatment often depends on the stage of cancer, the organ affected, and the patient condition. In surgically treated patients, pain relief can often be achieved and long-term neurologic stabilization tends to persist more often that it does with conservatively treated patients. Surgical intervention may be appropriate for patients with advanced disease for avoiding urgent life-threatening symptoms or serious functional disorders that annoys patients daily life, but surgical treatment for elderly cancer patients should be carefully considered after weighing the risk of post-operative mortality, morbidity, functional deterioration due to the surgery and speculated life surgery. Common surgical techniques are Laser surgery, Cryosurgery, Electrosurgery, Mohs surgery, Laparoscopic surgery, and Thorascopic surgery. Most often an adjuvant chemotherapy along with the surgical treatment of cancer is recommended. Surgical procedures for treatment of cancer is limited by Bleeding, Damage to internal organs and blood vessels, Reactions to drugs used (anesthesia) or other medicines, Problems with other organs, such as the lungs, heart, or kidneys, Infection at the site of the wound is another possible problem etc. Radiation therapy is often used for pain relief without curative intent.

Chemotherapy is a kind of treatment that uses drugs to attack cancer cells. Chemotherapy may be used to: Keep the cancer from spreading, Slow the cancer’s growth, Kill cancer cells that may have spread to other parts of the body, Relieve symptoms such as pain or blockages caused by cancer, Cure cancer. At present more than 50 anticancer drugs have been discovered. They are used in several ways: Monotherapy or only one drug, combined modality or chemotherapy along with other treatment such as surgery and radiotherapy, Combination chemotherapy or a group of drugs which work together. Major Side effects of chemotherapy are Nausea and vomiting, Hair loss, Bone marrow changes (Red blood cells, White blood cells, Platelets), Mouth and skin changes, Fertility problems, Memory changes, Emotional changes.

NATURAL DRUGS USED IN CANCER THERAPY:

Drugs obtained from natural origin contribute a major part in cancer treatment. An analysis of the number of chemotherapeutic agents and their sources indicates that over 60% of approved drugs are derived from natural compounds. Some recent developments in cancer chemotherapy from natural origin are summarized in table II.

Table 2: Summary of Drugs of Plant origin in different types of cancer

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Type of cancer</th>
<th>Mechanism of action</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>Camptothecin</td>
<td>Metastatic colon and rectal cancer</td>
<td>topoisomerase I</td>
<td>15-23</td>
</tr>
<tr>
<td>combretastatin A-4</td>
<td>Lung carcinoma and breast cancer</td>
<td>tubulin binding</td>
<td>24-29</td>
</tr>
<tr>
<td>Epipodophyllotoxin</td>
<td>small cell lung cancer, testicular carcinoma, lymphoma</td>
<td>topoisomerase I and II</td>
<td>30</td>
</tr>
<tr>
<td>Homoharrington</td>
<td>Lung carcinoma, colon adenocarcinoma</td>
<td>Protein synthesis inhibition</td>
<td>31-34</td>
</tr>
<tr>
<td>Ingenol</td>
<td>Skin cancer</td>
<td>protein kinase C activation</td>
<td>35-39</td>
</tr>
<tr>
<td>Daidzein</td>
<td>Breast and prostate cancer</td>
<td>NADH oxidase (tNOX) inhibition</td>
<td>40-43</td>
</tr>
<tr>
<td>Paclitaxel</td>
<td>Breast, ovarian, lung. bladder, prostate, melanoma</td>
<td>tubulin stabilization</td>
<td>44-52</td>
</tr>
<tr>
<td>Protopanaxadiol</td>
<td>stomach, lung, liver, pancreas, ovaries, &amp; colon</td>
<td>caspase 3, 8 and 9 stimulant</td>
<td>53-55</td>
</tr>
<tr>
<td>Triptolide</td>
<td>Prostate cancer</td>
<td>T-cell proliferation suppression, IL-2 expression and NFκ-B activation</td>
<td>56-62</td>
</tr>
<tr>
<td>Vinblastine</td>
<td>Hodgkin’s lymphoma, non-small cell lung cancer, breast, head and neck, &amp; testicular cancer</td>
<td>tubulin binding</td>
<td>63,64</td>
</tr>
<tr>
<td>Quercetin/Resveratrol</td>
<td>Colorectal cancer</td>
<td>induction of expression of caspase 3/8, causing DNA fragmentation, and arresting cells in G1 phase of the cell cycle</td>
<td>65</td>
</tr>
<tr>
<td>7,12-dimethylbenz(a) anthracene</td>
<td>Breast cancer</td>
<td>induced mammary carcinogenesis, and suppressed proliferation and lipogenesis in MCF-7 breast cancers</td>
<td>66,67</td>
</tr>
<tr>
<td>docosahexaenoic acid</td>
<td>Prostate cancer</td>
<td>increased lipid peroxidation and enhanced efficacy of anticancer drugs</td>
<td>68</td>
</tr>
<tr>
<td>glycyrrhizic acid and oleanolic acid</td>
<td>Skin cancer, Colon cancer and breast</td>
<td>Activate proapoptotic signaling cascades and suppression or nuclear translocation of various transcription factors including nuclear factor kappa B (NFκ-B)</td>
<td>69</td>
</tr>
<tr>
<td>Berberine hydrochloride</td>
<td>Lung cancer</td>
<td>Bind specifically to oligonucleotides and to stabilize DNA triplex or G-quadruplexes via telomerase and topoisomerase inhibition.</td>
<td>70</td>
</tr>
<tr>
<td>Curcumin</td>
<td>advanced pancreatic cancer</td>
<td>suppresses nuclear factor-nB (NF-nB) activation</td>
<td>71</td>
</tr>
</tbody>
</table>
COMBINATION CANCER THERAPY:
Chemotherapy drugs are most effective when given in combination (combination chemotherapy). The rationale for combination chemotherapy is to use drugs that work by different mechanisms of action, thereby decreasing the likelihood that resistant cancer cells will develop. When drugs with different effects are combined, each drug can be used at its optimal dose, without intolerable side effects.

Combinations used in breast Cancer:
Breast cancer (malignant breast neoplasm) is a type of cancer originating from breast tissue, most commonly from the inner lining of milk ducts or the lobules that supply the ducts with milk. Worldwide, breast cancer comprises 22.9% of all cancers (excluding non-melanoma skin cancers) in women. In 2008, breast cancer caused 458,503 deaths worldwide (13.7% of cancer deaths in women). Breast cancer is more than 100 times more common in women than breast cancer in men, although males tend to have poorer outcomes due to delays in diagnosis. An estimated 226,870 new cases of invasive breast cancer are expected to occur among women in the US during 2012; about 2,190 new cases are expected in men.

Nabholtz JM suggested that the taxanes and anthracyclines have emerged as the most active agents for treating women with advanced breast cancer. Phase II trials of the docetaxel combinations with either doxorubicin or epirubicin showed high activity, with acceptable tolerability in patients with metastatic breast cancer. Consequently, three randomized trials have compared docetaxel-anthracycline-based regimens with standard anthracycline-based polychemotherapies as first-line therapy for women with advanced breast cancer. Therefore, docetaxel-anthracycline combinations represent a validated option in first-line treatment for women with advanced breast cancer, and are further evaluated as adjuvant treatment for early stage breast cancer. Joensuu H et al showed that adjuvant treatment with docetaxel, as compared with vinorelbine, improves recurrence-free survival in women with early breast cancer, when docetaxel compared with vinorelbine for the adjuvant treatment of early breast cancer, a short course of trastuzumab administered concomitantly with docetaxel or vinorelbine is effective in women with breast cancer who have an amplified HER2/neu gene. Pretreatment with dexamethasone increases antitumor activity of carboplatin and gemcitabine. To our knowledge, this is the first report that DEX significantly enhances the antitumor activity of carboplatin and gemcitabine and increases their accumulation in tumors. These results provide a basis for further evaluation of DEX as a chemosensitizer in patients. Eichhorn PJ et al identified the tumor suppressor PTEN as a modulator of lapatinib sensitivity in vitro and in vivo. Their data show that deregulation of the PI3K pathway, either through loss-of-function mutations in PTEN or dominant activating mutations in PIK3CA, leads to lapatinib resistance, which can be effectively reversed by NVP-BEZ235. A recent phase III trial demonstrated that the combination of capecitabine (Xeloda) and docetaxel (Taxotere) significantly improved objective tumor response rate, time to disease progression, and overall survival compared with single-agent docetaxel in anthracycline-pretreated patients with advanced breast cancer. Green tea polyphenol EGCG synergistically sensitized breast cancer cells to paclitaxel in vitro and in vivo. EGCG in combination with paclitaxel significantly induced 4T1 cells apoptosis compared with each single treatment. EGCG may be used as a sensitizer to enhance the cytotoxicity of paclitaxel.

Combinations used in Prostate Cancer:
Prostate cancer is the most common human visceral malignancy and the third most common cause of cancer-related deaths among men in the Westernized world. Autopsy studies show that men in the fourth decade of life have a one-third risk of harboring small carcinomas of the prostate. Death rates from prostate cancer vary across the globe, with Westernized nations having the highest risk of incidence and death and Asian nations having the lowest.

Many tumors constitutively express high levels of the inducible form of proinflammatory enzyme, cyclooxygenase-2 (COX-2). Combination of Celecoxib and docetaxel inhibited COX-2 activity and associated alteration in cell death signaling, a potential clinical use of combined dosing of COX-2 inhibitors and cytotoxic drugs at lower, nontoxic dose than currently used to treat advanced prostate cancer. McCubrey JA et al isolated cell with the cancer initiating cell (CIC) phenotype from PC3 cells. Low doses of genistein can increase the sensitivity of prostate CICs to drugs such as docetaxel and cyclophosphamide, two drugs either used or under consideration for prostate cancer therapy. The polyamine analog PGI1047 potentiates the antitumor activity of cisplatin and bevacizumab in preclinical models of prostate cancer. The objective of the present study was to assess the antitumor effects of PGI1047 alone and in combination with approved anti-cancer agents. Another study showed that demonstrate the potential anticancer efficacy of genistein-topotecan combination in LNCaP
prostate cancer cells and the mechanism of the combination treatment. Treatments involving genistein-topotecan combination may prove to be an attractive alternative phytotherapy or adjuvant therapy for prostate cancer. An exploratory analysis of phase III trial participants found a substantial survival benefit to receiving docetaxel some months after sipuleucel-T. Sipuleucel-T is an active immunotherapy that triggers T-cell responses against prostate cancer. This trial highlights major unresolved questions concerning the optimum choice, dosing, and timing of chemotherapy relative to active immunotherapy. Stearns ME and Wang M have examined whether epigallocatechin-3-gallate (EGCG), and extract of green tea, in combination with taxane (i.e., paclitaxel and docetaxel), exerts a synergistic activity in blocking human prostate PC-3ML tumor cell growth in vitro and in vivo.

The overall chance of death from prostate cancer, even among Westernized nations that have not historically treated the disease for cure, is 3.5% to 4% from the large discordance between histologic incidence and death, there is great potential for overdetection and overtreatment. This is even more relevant as treatment-related morbidities associated with prostate cancer treatment can impact urinary function, sexual function, and quality of life. Disease prevention thus offers an attractive paradigm for addressing this important public health problem.

Combinations used in Colon Cancer:
An estimated 103,170 cases of colon and 40,290 cases of rectal cancer are expected to occur in 2012. Colon cancer, the fourth most common cancer in the world, is one of the leading causes of cancer death in both men and women in Western countries, including the USA. An expert panel assembled by the American Institute for Cancer Research/World Cancer Research Foundation came to a scientific consensus that there is evidence for a correlation between a high intake of saturated fats (and/or animal fat) and colon cancer risk.

Schroder CP, Maurer HR demonstrated in vitro, that pretreatment of human LS 174T colon cancer cells with nontoxic concentrations of tributyrin augments the sensitivity to spontaneous NK cell activity two-fold. However, when NK cells have been activated with an optimized combination of IL-2 and IL-12, the immunocytotoxicity increases up to five-fold (from 14% to 70%), versus a 3.8-fold increase against untreated cancer cells. These data suggested a synergistic link between induction of tumor cell differentiation and immunological defense mechanisms that may provide a rational basis for the improvement of clinical protocols, especially for colon cancer. In vitro, viable cell growth was determined by trypan blue exclusion assay and cell death was investigated by flow cytometry. ALA (50 µg/ml) and HESW (E1, EFD = 0.22 ml/mm², 1000 shots or E2, EFD = 0.88 ml/mm², 500 shots) showed a significant reduction of cancer cell proliferation at day 3 compared to cells exposed to ALA (p < 0.01) or HESW (p < 0.001) alone. In vivo, apoptosis detection was carried out by TUNEL assay, the pro-apoptotic gene Bad and Bcl-2 mRNA expression was evaluated by quantitative SYBR Green real time RT-PCR and cleavage of poly(ADP-ribose)-polymerase (PARP) was investigated by Western Blotting. The interaction between HESW and ALA is then effective in inducing apoptosis on a syngeneic colon cancer model. Antitumor activities of carboplatin and gencitabine with or without DEX pretreatment were determined in six marine-human cancer xenograft models, including cancers of colon (LS174T) and lung (LS174T). Although DEX alone showed minimal antitumor activity, DEX pretreatment significantly increased the efficacy of carboplatin, gencitabine, or a combination of both drugs by 2.4-fold in all xenograft models tested and increased their accumulation in tumors. Majumdar AP et al described that the combination of curcumin and resveratrol was found to be more effective in inhibiting growth of p53-positive (wt) and p53-negative colon cancer HCT-116 cells in vitro and in vivo in SCID xenografts of colon cancer HCT-116 (wt) cells versus either agent alone. In vitro studies have further demonstrated that the combinatorial treatment caused a greater inhibition of constitutive activation of EGFR and its family members as well as IGF-1R. Their current data suggest that the combination of curcumin and resveratrol could be an effective preventive/therapeutic strategy for colon cancer. In other review Felth J et al described that some cardiac glycosides (digitoxin, digoxin, Convallatoxin) have been reported to exhibit cytotoxic activity against several different cancer types like colorectal cancer etc. The combination of oxaliplatin and oxaliplatin exhibited synergism including the otherwise highly drug-resistant HT29 cell line. These findings demonstrate that such substances may exhibit significant activity against colorectal cancer cell lines.

Combinations used in lung cancer:
Lung cancer is the leading cause of cancer-related death in the world and can be broadly classified into small cell lung cancer (SCLC) and non-small-cell lung cancer (NSCLC). NSCLC accounts for approximately 85% of all lung cancers, and unlike SCLC, NSCLC is less sensitive to chemotherapeutic agents. Natural compounds in combination with chemotherapy agents enhance anticancer activities of drugs and reduce their toxicity. Milkarek M et al determined an effect of isothiocyanates and 5-fluorouracil used alone or in combination (in sequential or co-administrative treatments) on normal cell lines-V79. There was observed an antagonistic effect which was mainly dependent on the cell cycle distribution and their combination increased the cell number in the S phase. Another combination therapy using paclitaxel (PTX) as chemotherapeutic molecule and theophylline (TH) as differentiative agent in the prevention of metastasis in B16-F10 melanoma-bearing C57BL/6N mice. In vitro proliferation studies demonstrated that TH enhanced the antiproliferative effect of PTX. This study demonstrated that the simultaneous treatment of mice with TH and a low dose of PTX produced a similar anti-invasive effect than that caused by highly toxic PTX concentration. Antitumor activities of carboplatin and gencitabine with DEX pretreatment were determined in human cancer xenograft models, including lung (A549 and H1299) cancer. DEX pretreatment significantly increased tumor
carboplatin levels, including 200% increase in area under the curve, 100% increase in maximum concentration, and 160% decrease in clearance. DEX pretreatment similarly increased gemcitabine uptake in tumors. In other review PGI1047 is a polyamine analog currently in Phase I trials for advanced cancer in combination with a number of approved anti-cancer agents. The antitumor efficacy of PGI1047 as a single agent, and in combination with cisplatin and bevacizumab, was tested in models of lung (A549) cancer and the result that PGI1047 potentiated the antitumor effect of cisplatin. Green tea is now recognized as the most effective cancer preventive beverage. The synergistic enhancement of apoptosis and GADD153 gene expression in human non-small cell lung cancer cells by the combination of EGCG and celecoxib were mediated through the activation of the MAPK signaling pathway. This article reviews the synergistic enhancement of apoptosis, gene expression, and anticancer effects using various combinations of EGCG and anticancer drugs. Saha A et al studied the enhancing effects of EC on inductions of growth inhibition and apoptosis in human lung cancer cell lines PC-9 and A549 with curcumin. The combination similarly increased both apoptosis and expression of GADD153 and GADD45 genes, associated with their enhanced protein production. This report is the first report on the enhancing effects of EC on curcumin, and the data suggest that EC plays a significant role in the enhancement of the cancer-preventive activity of curcumin in the diet.

CONCLUSION:
Numerous experimental, clinical, and epidemiologic studies indicate that combination of drugs particularly natural drug combination show promise as anticancer drugs chemotherapy. The clinical application of these combination drugs is still limited by the lack of randomized evidence of their efficacy and safety. The combination therapy is definitely a promising area in chemotherapy to reduce the dose of anticancer drugs henceforth the adverse effects as well but more clinical studies are required.

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REFERENCES:

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56. Pharmagenesis: Further Information Available at http://www.pharmagenesis.net.


### Table 3: Summary of combinations of drugs of herbal origin in different types of cancer

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Mechanism of Action</th>
<th>Clinical Status</th>
<th>Type of Cancer</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isothiocyanates and 5-fluorouracil</td>
<td>Increased the cell number in the S phase</td>
<td>Lung cancer</td>
<td>114</td>
<td></td>
</tr>
<tr>
<td>Paclitaxel (PTX) and theophylline (TH)</td>
<td>TH enhanced the antiproliferative effect of PTX</td>
<td>Lung metastasis</td>
<td>115</td>
<td></td>
</tr>
<tr>
<td>Docetaxel and doxorubicin</td>
<td>Showed high activity, with acceptable tolerability</td>
<td>II Metastatic breast cancer</td>
<td>80,81</td>
<td></td>
</tr>
<tr>
<td>Trastuzumab and docetaxel/vinorelbine</td>
<td>Specific for HER-2-overexpressing tumor cells</td>
<td>II Breast cancer</td>
<td>82-88</td>
<td></td>
</tr>
<tr>
<td>Glucocorticoid and irinotecan</td>
<td>Decreased incidence and duration of diarrhea</td>
<td>Advanced colorectal cancer</td>
<td>118</td>
<td></td>
</tr>
<tr>
<td>Celecoxib and docetaxel</td>
<td>Inhibit COX-2 activity and associated alteration in cell death signaling</td>
<td>Advanced prostate cancer</td>
<td>96</td>
<td></td>
</tr>
<tr>
<td>Dexamethasone and carboplatin/gemcitabine</td>
<td>Enhanced the antitumor activity and increased their accumulation in tumors</td>
<td>Colon, Lung, and Breast cancers</td>
<td>89</td>
<td></td>
</tr>
<tr>
<td>Budesonide and loperamide</td>
<td>Decreased incidence and duration of diarrhea</td>
<td>Advanced colorectal cancer</td>
<td>118</td>
<td></td>
</tr>
<tr>
<td>Cisplatin and HemoHIM</td>
<td>Enhanced the antitumor efficacy and decreased the tumor size and weight</td>
<td>Melanoma</td>
<td>119</td>
<td></td>
</tr>
<tr>
<td>genistein and cyclopamine</td>
<td>BC cells are usually more rapidly proliferating than the CICs</td>
<td>Prostate cancer</td>
<td>97</td>
<td></td>
</tr>
<tr>
<td>Lapatinib and trastuzumab</td>
<td>Inhibited growth of HER2+ tumors resistant to anti-HER2 therapy</td>
<td>Advanced and metastatic breast cancer</td>
<td>90</td>
<td></td>
</tr>
<tr>
<td>capecitabine (Xeloda) and docetaxel (Taxotere)</td>
<td>Improved tumor response rate, time to disease progression, and overall survival</td>
<td>Advanced breast cancer</td>
<td>91</td>
<td></td>
</tr>
<tr>
<td>PG11047 and cisplatin and bevacizumab</td>
<td>Inhibit polyamine biosynthetic enzymes, induce the polyamine catabolic enzymes spermidine/spermine N(1)-acetyltransferase (SSAT) and spermine oxidase (SMO)</td>
<td>I Lung and Prostate cancer</td>
<td>98</td>
<td></td>
</tr>
<tr>
<td>genistein-topotecan</td>
<td>Triggers T-cell responses</td>
<td>Prostate cancer</td>
<td>99</td>
<td></td>
</tr>
<tr>
<td>EGCG and paclitaxel/cisplatin and docetaxel</td>
<td>Inhibits TNF-α-induced promoter activity of the chemokine IL-8 by an interference with the IκB/NFκB pathway/activation of the MAPK signaling pathway/inhibited P-glycoprotein (P-gp) efflux pump activity/blocking human prostate PC-3ML tumor cell growth in vitro and in vivo.</td>
<td>Breast carcinoma / lung cancer / liver cancer / Prostate cancer</td>
<td>92,101, 116,120, 121</td>
<td></td>
</tr>
<tr>
<td>Curcumin with (-)-epicatechin (EC)</td>
<td>Increased both apoptosis and expression of GADD153 and GADD45 genes</td>
<td>Lung cancer</td>
<td>117</td>
<td></td>
</tr>
<tr>
<td>Gemcitabine and Guggulsterone</td>
<td>Enhanced antitumor efficacy through apoptosis induction by suppressing Akt and nuclear factor κB activity</td>
<td>Pancreatic Cancer</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Curcumin and resveratrol</td>
<td>Inhibiting growth of p53-positive (wt) and p53-negative colon cancer HCT-116 cells in vitro and in vivo in SCID xenografts of colon cancer HCT-116 (wt) cells</td>
<td>Colorectal cancer</td>
<td>110</td>
<td></td>
</tr>
<tr>
<td>Convallatoxin and oxaliplatin</td>
<td>Exhibited synergism including the otherwise highly drug-resistant HT29 cell line</td>
<td>Colon cancer</td>
<td>111</td>
<td></td>
</tr>
</tbody>
</table>