Nutrients and Cancer: An Introduction to Cesium Therapy

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A brief overview on the relevance of dietary factors in both development and prevention of cancer is presented. The pharmacologic properties of various food ingredients are discussed. Establishing of a special diet for the cancer patient is suggested. In addition, avoidance of certain foods is recommended to counteract mucus production of cancer cells. Evaluation of the nutrient content of certain diets in regions with low incidence of cancer has advanced the use of certain alkali metals, i.e., rubidium and cesium, as chemotherapeutic agents. The rationale for this approach, termed the "high pH" therapy, resides in changing the acidic pH range of the cancer cell by cesium (or rubidium) towards weak alkalinity in which the survival of the cancer cell is endangered, and the formation of acidic and toxic materials, normally formed in cancer cells, is neutralized and eliminated.

TREATMENT MODALITIES OF CANCER

The cancer cell is known to produce large amounts of mucus and this, in turn, shields the cancer cells from the immune system and from being penetrated by chemotherapeutic agents. It even protects against radiation if the layer of mucus is thickened up. Later this mucus can become penetrated by chemotherapeutic agents. It even protects against radiation if the layer of mucus is thickened up. Later this mucus can be demonstrated in the blood, e.g., with the HLB blood test. Use of certain agents help to dissolve the blocking effect of the mucus. This includes the use of beta-carotene which decomposes blocking mucoid proteins mediated by electrical charges. It gets inactivated and decolorized by the blocking mucoid. The necessary dosage for optimal effect produces the so-called "carotenemia", an orange-yellow tinge to the patient's skin. Likewise, heparin inactivates the immune repelling and immune binding capacities of the mucoid proteins by electrical charges. Furthermore, compounds like bromelain, papain (in green papayas), Wobemugos®, or pancreatic enzymes will not only break down mucus but also destroy leukemic cells.

NUTRITION AND CANCER MUCUS

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**TABLE I**

<table>
<thead>
<tr>
<th>Substance/Compound*</th>
<th>Possible Mechanism of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cesium and Rubidium</td>
<td>Raise pH in cancer cells and gene repair.</td>
</tr>
<tr>
<td>Omega-3 Essential Fatty Acids</td>
<td>Repair cell membranes; enhance the immune system and prostaglandin synthesis.</td>
</tr>
<tr>
<td>Eicosapentaenoic acid and Docosahexaenoic acid</td>
<td>Decompose blocking mucus and enhance immune system.</td>
</tr>
<tr>
<td>Carotene and Vitamin A</td>
<td>Antioxidant and broadens electron donor capacity of cancer cell membrane.</td>
</tr>
<tr>
<td>Selenium</td>
<td>Enhance the immune system.</td>
</tr>
<tr>
<td>Vitamin C and Bioflavonoids</td>
<td>Oxygen carrier; interferon stimulator, and gene repair.</td>
</tr>
<tr>
<td>Germanium</td>
<td>Immune enhancer; precursor of tumosteron.</td>
</tr>
<tr>
<td>Vitamin D2 (Ergocalciferol)</td>
<td>Membrane stabilizer; part of xanthine oxidase which mobilizes iron from liver and of aldehyde oxidase necessary for fat oxidation.</td>
</tr>
<tr>
<td>Molybdenum</td>
<td>Electron donor; antioxidant, and immune stimulant.</td>
</tr>
<tr>
<td>Zink</td>
<td>Enzyme activator; gene and membrane stabilizer.</td>
</tr>
<tr>
<td>Magnesium</td>
<td>Natural cancer inhibitors.</td>
</tr>
<tr>
<td>Nitriles (amygdalin; mandelonitrile-+glucuronide or Laetrile®)</td>
<td>Broaden electron donor capacity of the cancer cell membrane; release of cancer growth inhibiting benzaldehyde and cyanide.</td>
</tr>
<tr>
<td>Alllicin and sinigrin</td>
<td>Desalting substance that lowers sodium in cancer cells.</td>
</tr>
<tr>
<td>Taurine</td>
<td>Precursor of dehydroepiandrosterone (DHEA) an anticancer, antiaging, and antiboity factor.</td>
</tr>
<tr>
<td>Squalane</td>
<td>Membrane and gene stabilizing.</td>
</tr>
<tr>
<td>Saponins</td>
<td>Decomposes blocking mucus surrounding the cancer cells (are found in KIRLIAN positive raw vegetables and fresh vegetable juices, especially carrot juice.)</td>
</tr>
<tr>
<td>Photonic Energy</td>
<td>Slows down mitosis and the multiplication of cancer cells.</td>
</tr>
<tr>
<td>Niacinamide</td>
<td>Reduces passage-time, decreasing the exposure of intestines, especially the colon, to carcinogens; binds toxic substances and carcinogens.</td>
</tr>
<tr>
<td>Food Fiber</td>
<td>Required for cell respiration (esp. B2) and catalysts for numerous enzymes.</td>
</tr>
<tr>
<td>B-complex vitamins, especially Riboflavin (Vitamin B2)</td>
<td>Membrane stabilizer; part of xanthine oxidase which mobilizes iron from liver and of aldehyde oxidase necessary for fat oxidation.</td>
</tr>
<tr>
<td>Vitamin E (mixed D-Tocopherols)</td>
<td>Membrane stabilizer; part of xanthine oxidase which mobilizes iron from liver and of aldehyde oxidase necessary for fat oxidation.</td>
</tr>
<tr>
<td>Pantothentic acid</td>
<td>Membrane and gene stabilizer.</td>
</tr>
<tr>
<td>Folic Acid</td>
<td>Electron donor; antioxidant, and immune stimulant.</td>
</tr>
<tr>
<td>Certain Amino Acids (Cystein, Arginine, Ornithine; also IGF 1, etc., from Comu parvum of the Chinese deer Cervus nippon/elaphus)</td>
<td>Required for cell respiration (esp. B2) and catalysts for numerous enzymes.</td>
</tr>
</tbody>
</table>

*See text for the presence of these compounds in various food ingredients.

Notes: 1 Rubidium is especially beneficial for pancreatic and liver cancers and other malignancies that present with clinical depression. Rubidium chloride (RbCl) is established as an antidepressant of low toxicity and acts similar to the equally nontoxic lithium orotate.

2 Amygdalin, found in bitter almonds, apricot and peach pits, is hydrolyzed to 1-mandelonitrile-+glucoside and oxidized with platinum black to mandelonitrile-6-glucuronide or Laetrile® which, in turn, is turned by a 6-glucuronidase into glucuronic acid, and the cancer-suppressive HCN and benzaldehyde.

It is recommended, therefore, to avoid all mucus producing foods, e.g., all dairy products, except goat milk derivatives, egg whites (whereas soft yolks are highly beneficial) for all blood types (with possible exception of Type Bs), and, more specifically, all grains, except brown rice, for Type Os (and Bs), all meat, especially for Type As (and Abs) which do well on whole grains and locally grown vegetables, all soy bean (Glycine max) derivatives for Type Os and Bs (e.g., soy milk, tofu, tempe, etc.), tomatoes, except for Type Os, most beans and nuts, except almonds and walnuts, for Type Bs and Os (pumpkin seeds: good). Foods with high contents of sugar, alcohol, citrus fruits, refined carbohydrates, salt, pork in every form, as well as the blood-type specific mucus-producing foods mentioned previously, may also increase the specific mucus production of cancer cells. The common (kosher) practice of not eating other items with milk is of significant bearing in this respect because (cow’s) milk produces a mucus that coats all the food, and thus prevents all the nutrients from being absorbed. We have also been using (CaNa₂-)EDTA for chelation therapy which has been shown to reduce incidences of cancer and heart diseases by 90% and 50%, respectively [1]. Likewise, increased cancer incidence may result from consumption of egg-whites and certain crustaceans, e.g., lobster, shrimp, and crayfish, due to their high content of nucleic acids which can be detrimental to the cancer patient. Therefore, the elimination of these items from diet may conceivably reduce the incidence of cancer.

VITAMINS, ESSENTIAL FATTY ACIDS, AND FREE RADICALS

The Smithsonian Institute has done a study on the incidence of cancer in sharks. They examined 25,000 sharks and found only one individual case with cancer. This suggests that the shark (e.g., genus *Squalus*) is probably immune to cancer. The shark liver oil contains vitamins and other compounds with anticancer activity. This includes squalene which is also contained in cod liver and in olive oil. Squalene increases the...
polarization of the cell membrane and thereby may facilitate the action of immune system on the cancer cell. This compound is also a precursor for dehydroepiandrosterone (DHEA) which possesses anticancer activity. Moreover, squalene has been implicated in the mechanism of Na accumulation by the cancer cell. For example, the high uptake of Na⁺ by the cancer cell induces an electrical potential that defies the immune mechanisms. Thereafter, both, blood forming organs and the blood cells, show a high content of Na⁺ which can undergo desodification by squalene and certain sulphur-containing compounds also found in shark oil, such as taurine (oxidized sulfur-containing amine forming conjugates with cholic and deoxycholic acid in bile; also a CNS neurotransmitter or neuromodulator) and isethionic acid (H₂O₂, CH₃SO₃H). Lithium ortate counteracts Na⁺ retention as well and also effectively increases the monocyte and granulocyte counts. Certain vitamins, e.g., carotenes and Vitamin A, are associated with low incidence of lung cancer. The excess of other vitamins, i.e., over 5 g of vitamin C/day, may enhance tumor growth in leukemias, and over 10 g/day in lymphomas and similar cancers.

It appears that the foods with the most decisive effects on the reduction of cancer incidence in humans and in animals contain omega-3 unsaturated essential fatty acids (EFAs) which are also found in oils derived from linseed, chestnuts, beechnuts, soy beans, walnuts, and wheat germ. In addition, soybean and wheat germ oils contain high amounts of Vitamin E. Also rich in EFAs are cold climate legumes of which soybeans and their products, adzuki, black and pinto beans, and lentils are beneficial for blood type A, pinto and navy beans, lentils, and soybean products are beneficial for Type ABs, adzuki beans and black-eyed peas are beneficial for Type O's, and kidney and navy beans for Type Bs. Richest in •·3 EFAs are cold water fish of which cod, halibut, swordfish, sturgeon, and herring are beneficial for Type Os, cod, mackerels, salmon, and trout for Type As, cod, salmon, halibut, mackerel, sardines, sturgeon and caviar for Type Bs, and cod, halibut, herrings, mackerels, tuna, and sturgeon for Type ABs. Cold-water plankton and sea vegetables are other rich sources of •·3 EFAs. In cold climate animals, eicosapentaenoic acid (EPA) is immediate precursor of 3-series of prostaglandins which are involved in protection of cellular functions and prevent the formation of 2-series prostaglandins which are carcinogenic. EPA inhibits formation of thromboxane A₂ (TXA₂) an extremely potent platelet aggregator and vasoconstrictor and antagonist of prostacyclin that may contribute to cancer by local tissue hypoxia. A diet rich in EPA also decreases elevated levels of LDL cholesterol and triglycerides, and inhibits in vitro chemotactic and aggregating activities of neutrophils. Careful selection of oils should be considered which should be kept refrigerated and kept under a nitrogen seal until sold to prevent oxidation due to high contents of •·3 EFAs in cod liver oil and other unsaturated essential fatty acids (EFAs) which are also found in oils derived from linseed, chestnuts, beechnuts, soy beans, and wheat germ. Furthermore, in fish oils, the potential toxicity of Vitamins A and D should be considered. The use of margarine or hydrogenated oils, non-cold pressed polysaturated oils, shortenings, bacon, grease, non-dairy creamers, egg substitutes, commercial mayonnaise and salad dressings, as well as of tropical oils like coconut oil, palm oil or cottonseed oil should be avoided. Excess amounts of polysaturated compounds may be oxidized which damages the cell membrane and these oxidized EFAs cannot be transported anymore into the mitochondria by carnitine and thus circulate in the bloodstream. These oxidized EFAs promote the release of free radicals which are carcinogenic, may drive LDL cholesterol from the bloodstream into the liver and body cells, and destroy HDL cholesterol and interfere with the HDL-associated lecithin-cholesterol acyl transferase (LCAT). Note that terminal cancers consistently have HDL levels from 0.0 to 20.0 mg/dL and total cholesterol levels from 20.0 (l) to 120 mg/dL which seem to be the most reliable indicators of the life expectancy of the patient, and HDL levels under 30 mg/dL and total cholesterol under 150 mg/dL should prompt a workup to rule out a cancer. [For further details see Author's Note under (3) at the end of this article]

Oxidized EFAs also cause a significant elevation of uric acid (indicating destruction of cellular nucleoprotein), cause iron deficiency and anemia, liver disease, intestinal damage and obstruction, amyloidosis (abnormal waxy deposits in tissues), hypertension, gallstones, and increase the incidence of atherogenesis (formation of atherosclerosis). Free radical induction from overheating fats is also due to formation of the highly toxic decomposition of glycercin. Note that acrolein is one of the degradation products of cyclophosphamide, used in cancer chemotherapy, and is thought to be the cause of hemorrhagic cystitis and bladder cancer in patients treated with cyclophosphamide. [See also Appendix III: Dietary Carcinogens]

In subtropical or tropical climates the use of "southern oils" is recommended. These include light and dark sesame oil, sunflower and safflower (not for Type Os, Bs, and ABs), and especially, virgin olive oil (beneficial to all blood types, as is walnut oil) which contain small amounts of the anticancer shark factor squalene. The foregoing observations suggest the use of cod liver oil (except for Type O non-secretors) for several months to increase EPA intake, and to administer in addition 6000 U of Vitamin A and 600 U of Vitamin E. Other supplements will be wheat germ oil (not for Type Os) to provide energy from octacosanol and to improve physical fitness. Sesame oil may be advantageous against various bleedings, i.e., nose, gastrointestinal and gynecological bleedings. The supplement EPA can be given as concentrate or by consumption of EPA - rich foods which may also be beneficial in certain heart, blood vessel, bowel, and immune diseases, as well as cancer. In temperate climates, the caloric intake from EFAs should be approximately 2% to 3%. Furthermore, gamma linolenic acid from evening primrose oil has been found, via synthesis of the series-1 prostaglandins, most notably prostaglandin E1 (PGE₁), to decrease elevated LDL cholesterol levels, lower blood pressure by vasodilatation, inhibit thrombosis/platelet aggregation, and to normalize cancer cells.

OXYGENATION (see Appendix I)

It is generally assumed that healthy cells resist becoming cancerous if they are provided with adequate nutrients. Insufficient supply of a given critical nutrient may lead to or facilitate cancer induction. For example, the amount of oxygen reaching the cells since low cellular oxygen levels may result in anaerobic conditions which will further cancer development. The lack of oxygen has long been suspected in carcinogenesis because it leads to an anaerobic metabolism where, essentially, glucose is converted into lactic acid and the pH in the cancer cells becomes acidic. The acidic pH because of lack of O₂ may cause breakdown of RNA and DNA and damage the cellular control mechanisms involved. The development of acidic toxins usually will lead to the destruction of cell structures. Therefore, reversing this condition requires adequate oxygenation. There are certain elements, e.g., germanium, which may prove beneficial for cellular oxygenation. Germanium possesses 8 valences and therefore can carry 4 atoms of oxygen and may provide the oxygenation needed for the cancer cell to evoke anticancer effect. Ginseng normally grows only on germanium rich soil and should provide a good source for this phenomenon. However, the use of soil antibiotics in homegrown ginseng may interfere in the production of an effective ginseng due to its lack of soil derived geranium. This sensitivity of cancer cells to oxygenation explains, in part, the effectiveness of oxidative therapies, such as hydrogen peroxide and, particularly, ozone, in certain cancers. Ozone, especially directly I.V., in the author's experience, without any doubt, greatly enhances the effects of all cancer therapies including the one suggested herein.

Certain sulfonated compounds are beneficial for Type As, cod, mackerels, salmon, and trout for Type As, cod, salmon, halibut, mackerel, sardines, sturgeon and caviar for Type Bs, and cod, halibut, herrings, mackerels, tuna, and sturgeon for Type ABs. Cold-water plankton and sea vegetables are other rich sources of •·3 EFAs. In cold climate animals, eicosapentaenoic acid (EPA) is immediate precursor of 3-series of prostaglandins which are involved in protection of cellular functions and prevent the formation of 2-series prostaglandins which are carcinogenic. EPA inhibits formation of thromboxane A₂ (TXA₂) an extremely potent platelet aggregator and vasoconstrictor and antagonist of prostacyclin that may contribute to cancer by local tissue hypoxia. A diet rich in EPA also decreases elevated levels of LDL cholesterol and triglycerides, and inhibits in vitro chemotactic and aggregating activities of neutrophils. Careful selection of oils should be considered which should be kept refrigerated and kept under a nitrogen seal until sold to prevent oxidation due to high contents of •·3 EFAs in cod liver oil and other unsaturated essential fatty acids (EFAs) which are also found in oils derived from linseed, chestnuts, beechnuts, soy beans, and wheat germ. Furthermore, in fish oils, the potential toxicity of Vitamins A and D should be considered. The use of margarine or hydrogenated oils, non-cold pressed polysaturated oils, shortenings, bacon, grease, non-dairy creamers, egg substitutes, commercial mayonnaise and salad dressings, as well as of tropical oils like coconut oil, palm oil or cottonseed oil should be avoided. Excess amounts of polysaturated compounds may be oxidized which damages the cell membrane and these oxidized EFAs cannot be transported anymore into the mitochondria by carnitine and thus circulate in the bloodstream. These oxidized EFAs promote the release of free radicals which are carcinogenic, may drive LDL cholesterol from the bloodstream into the liver and body cells, and destroy HDL cholesterol and interfere with the HDL-associated lecithin-cholesterol acyl transferase (LCAT). Note that terminal cancers consistently have HDL levels from 0.0 to 20.0 mg/dL and total cholesterol levels from 20.0 (l) to 120 mg/dL which seem to be the most reliable indicators of the life expectancy of the patient, and HDL levels under 30 mg/dL and total cholesterol under 150 mg/dL should prompt a workup to rule out a cancer. [For further details see Author's Note under (3) at the end of this article]
LOW–pH Therapy

In this therapy, glucose is injected into the blood stream. As a consequence the cancer cell pH drops to the 5.5 range. The patient is then placed in a chamber heated from 45 to 50°C (113 to 122°F) for 4 to 6 hrs [8]. Diathermy is also applied over the tumor area which, in the absence of a blood supply, will cause the temperature of the tumor mass to rise to over 55° (133°F). At these high temperatures, the life of cancer cells is observed to be very short. An apparent drawback to the therapy is that a case of severe toxemia may result from the disintegrating tumor mass. This approach has been referred to as “high pH” therapy. A combined effect of low pH and high body temperature has been also suggested in cancer treatment and termed the “low pH” therapy. Both pH therapies are briefly outlined below; the low pH therapy was devised by Von Ardenne [8] and the high pH therapy by Brewer [2]. Both have been shown to be effective therapeutic measures for the treatment of cancer in laboratory animals and humans.

HIGH–pH Therapy

The rapid uptake of cesium and rubidium observed for cancer cells is the theoretical approach of high pH therapy [2]. This therapy has been tested using CsCl or CsCO₃ in conjunction with the administration of ascorbic and retinoic acids, zinc and selenium salts. The weak acids which are absorbed by the tumor cells have been shown to enhance the negative potential gradient across the membrane. Zinc and selenium salts, when absorbed on the membrane surface, act as broad and moderately strong electron donors. These ions and salts have been shown in mice to drastically enhance the uptake of cesium and rubidium ions. For treatment of cancer patients the administration of 6 to 9 g of CsCl or CsCO₃ for several days is believed to be tolerable and sufficient to raise the pH in the tumor cells to a weak alkaline level of approximately pH 8 where the life cancer cell is shortened. In addition, the presence of cesium and rubidium salts in the body fluid is expected to neutralize the acidic and toxic material emanating from the disintegrating tumor mass.

Results from both animal experiments, mainly through Messiha [6], and these of ours in limited clinical trial in humans in our clinic [7], are indicative of a high success rate of Cs treatment in cancer therapy.

Thus, both dietary factors and selected elements and vitamins may play a more significant role in the pathogenesis and pathology of certain cancers than has been previously accounted for. Moreover, changes in dietary habits may have a lasting effect on protection against cancer development and progression.

To conclude, the author presents a list of cancer protective nutrients and their main functions and importance outlined in Table I. Appendix III lists “Naturally Occurring Dietary Carcinogens”.

REFERENCES


ACKNOWLEDGMENT

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Cesium Therapy in Cancer Patients

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SARTORI. H. E. Cesium Therapy in Cancer Patients. PHARMACOL BIOCHEM BEHAV 21: Suppl. I: 11-13. 1984. - The effect of cesium therapy on various cancers is reported. A total of 50 patients were treated over a 3 year period with CsCl. The majority of the patients has been unresponsive to previous maximal modalities of cancer treatment and was considered terminal cases. The Cs-treatment consisted of CsCl in addition to some vitamins, minerals, chelating agents, and salts of selenium, potassium and magnesium. In addition, a special diet was instituted. There was an impressive 5 year recovery of various cancers, including, cancers of unknown primary, breast, colon, prostate, pancreas, lung, and liver, lymphoma, Ewing sarcoma of the pelvis, and adenocarcinoma of the gallbladder by the Cs-therapy employed. There was a 26% and 24% death within the initial 2 weeks and 12 months of treatment, respectively. A consistent finding in these patients was the disappearance of pain within the initial 3 days of Cs-treatment. The small number of autopsies made showed the absence of cancer cells in most cases and the clinical impression indicates a remarkably successful outcome of treatment.

- Table I summarizes the results of the Cs-treatment of 50 cancer patients treated over a 3 year period. The initial death occurrences for the initial 2 weeks of treatment were in the same order and magnitude as those recorded for the 12 month period. The percent of survival of patients with breast, colon, prostate, and pancreas and cancers of unknown primary was 50%, while 3 out of 5 lung cancer patients, 2 out of 3 lymphoma patients, and one out 3 liver cancer patients treated in this series survived. An overall 50% recovery from cancer by the Cs-therapy was determined in the 50 patients treated. Data from the autopsies made indicated the absence of tumors in patients dying within 14 days of the Cs-treatment.

- Treatment was performed on 50 patients during the last three years at Life Science Universal Medical Center in Rockville, Maryland and in Washington, D.C. All patients were terminal subjects with generalized metastatic disease. Forty-seven of the 50 patients studied had received maximal modalities of conventional treatment, i.e., surgery, radiation, and various chemotherapies, before the metabolic Cs-treatment was initiated. Thirty-four of the patients were considered terminal due to previous treatment outcomes and cancer complications. The type of cancer of studied and the number of patients is detailed in Table I.

The Cs-treatment was given in conjunction with other supportive compounds. The patients were maintained on a cancer control diet. In addition, specific compounds and modalities were used to produce adequate circulation and oxygenation. According to individual cases, CsCl was given in daily dosages of 6 to 9 g, in three equally divided doses, in conjunction with vitamin A emulsion (100,000 to 300,000 I.U.), vitamin E (400 to 1000 I.U.), vitamin C (4 to 30 g), zinc (80 to 100 mg), selenium (600 to 1,200 mcg), and amygdalin (1,500 mg). Other supplementations were used according to the specific needs of the patient. The diet consisted mainly of brown rice, root and leafy vegetables, linolenic acid rich oils [linseed = flaxseed and walnut; soy (not for blood type O and B), wheat germ (not for Type O)], and other supplemental foods. To increase the efficiency of the treatment and to improve the circulation and oxygenation, the patients received the chelating agent (CaNa2) EDTA dimethylsulfoxide (DMSO), and also a combination of vitamins, and K and Mg salts.

RESULTS

Table I summarizes the results of the Cs-treatment of 50 cancer patients studied for over 3 years. They had generalized metastatic disease, except for 3 patients. Initial death occurrences for the initial 2 weeks of treatment were in the same order and magnitude as those recorded for the 12 month period. The percent of survival of patients with breast, colon, prostate, and pancreas and cancers of unknown primary was 50%, while 3 out of 5 lung cancer patients, 2 out of 3 lymphoma patients, and one out 3 liver cancer patients treated in this series survived. An overall 50% recovery from cancer by the Cs-therapy was determined in the 50 patients treated. Data from the autopsies made indicated the absence of tumors in patients dying within 14 days of the Cs-treatment.

One of the most striking effects of the treatment was the disappearance of pain in all patients within one to three days after initiation of the treatment. The disappearance of pain may be explained by changing the acidic pH that triggers the pain in the pain receptors to slightly alkaline where all pain sensations disappear. This does not only apply to cancer pain but also to arthritic and neuralgic pain that also consistently disappear after application of Cs, Rb, K, M, and Ca, in descending order of effectiveness. Pain relief from alkalizing agents is primarily a function of a general shift of the body's acid-base balance towards the alkaline that takes place preferentially in acidic areas and is a principle that was known in medicine since ancient times (see, e.g., Huang Di Nei Jing, The Book of Internal Medicine of the Yellow Emperor).
The effect of CsCl treatment on various advanced types of cancer in man

Morbidity time post therapy

<table>
<thead>
<tr>
<th>Tumor</th>
<th>(n)</th>
<th>14 Days</th>
<th>12 Months</th>
<th>Number of Survivals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast**</td>
<td>(10)</td>
<td>3*</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Unknown Primary</td>
<td>(8)</td>
<td>2*</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Colon</td>
<td>(9)</td>
<td>2</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Prostate</td>
<td>(6)</td>
<td>1*</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Pancreas</td>
<td>(4)</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Lung</td>
<td>(5)</td>
<td>1*</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Liver</td>
<td>(3)</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Lymphoma</td>
<td>(3)</td>
<td>1*</td>
<td>—</td>
<td>2</td>
</tr>
<tr>
<td>Ewing Sarcoma Pelvis</td>
<td>(1)</td>
<td>—</td>
<td>1</td>
<td>—</td>
</tr>
<tr>
<td>Adenocarcinoma Gall bladder</td>
<td>(1)</td>
<td>1</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Total Cases</td>
<td>(50)</td>
<td>13</td>
<td>12</td>
<td>25</td>
</tr>
</tbody>
</table>

*An autopsy was performed on one patient of each group which did not indicate the presence of cancer
+One case of breast cancer died to traumatic fracture of the neck.

These studies were performed under my direction, initiated in April 1981. Forty-eight patients were initially treated with CsCl between April 1981 and October 1982. They were subjected to various conventional cancer therapies such as surgery, radiation, and chemotherapy, and were considered terminal cases with general metastatic disease, except for 3 patients who were not previously treated. Three patients were comatose at the time of the Cs treatment. Thirteen patients died within less than 2 weeks of treatment. Each patient showed a reduction in tumor mass by the Cs treatment. Of the breast cancer patients, the most impressive effect was seen in a 44 year old housewife who was comatose at the beginning of the Cs-treatment and was considered a terminal case with a life expectancy of at most of one week. The Cs-therapy, with the other ingredients used, was immediately instituted by the nasogastric route because there was no cooperation of the patient for regular oral administration possible. The daily CsCl dose amounted to 30 grams in three divided doses of 10 grams each. The patient regained consciousness within 24 hours after the initiation of the treatment and she was ready to leave her bed after five days on the therapy. However, when attempting to get out of her bed, a fall that resulted in a fracture of cervical vertebra 2 caused her unfortunate demise within another 48 hours. The autopsy revealed that cancer metastasis had essentially "eaten away" parts of the neck, head, and trochanter of her hip bone causing this tragic accident.

The next most frequent cancer treated was of unknown primary. Treatment of 8 cases showed a death rate of 2 within 14 days of treatment and an additional 2 deaths within 12 months while 4 of the patients are still living. In one case, an autopsy was made in a patient after one week of Cs treatment and showed a complete disappearance of the cancer. Thos patient's death was attributed to the fulminating cardiotoxicity from her treatment with doxorubicin HCl (Adriamycin®) that had resulted in an intractable congestive heart failure. There were 7 cases of colon cancer patients who were treated with CsCl. Two of these patients died within 14 days. One of these two patients had previous massive chemotherapy and too little time was available to restore her bone marrow depression and dysmetabolic condition. The previously existing infiltration of the abdominal wall had disappeared in both patients. However, no consent was given for an autopsy.

In one lymphoma case the patient displayed a maximally extended spherical abdomen which was hard and he weighed approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 112 kg (250 pounds). This massively enlarged abdomen progressively reduced in size with a loss of approximately 55 kg (121 lbs) of body weight after 3 months on the Cs therapy. Of this weight loss 30 kg (66 lbs) was attributed to ascites and 25 kg (55 lbs) to reduction of the tumor mass. The spleen which was originally massively enlarged and reaching into the pelvis, was reduced to almost normal. The liver position was down to about the level of umbilicus and was reduced to normal size in 3 months. The patient is still living 3 years after his discharge. Unfortunately there was no follow-up on this patient and he is maintained on chemotherapy.

Discussion

The results presented demonstrate the rate of efficacy of CsCl in cancer therapy. The total of 50 cancer cases shows an impressive 50% survival rate. This confirms the work of Messiha reported elsewhere in these proceedings showing that the higher the dose the more effective it seems. The autopsy obtained from the patient whose death was attributed to a traumatic fracture of the neck, indicates that the cancer has been initially further advanced resulting in bone destruction. However, the absence of cancer after the massive CsCl dose used in this case is demonstrable of the therapy. It appears that both dosage, i.e., as much as 30 grams/day, and route of drug administration, i.e., the nasogastric pathway, might have contributed to the patient's rapid recovery. It should be noted, however, that the oral CsCl dosage in humans should not exceed 20 to 40 grams/day to avoid side effects, notably nausea and diarrhea. The author's personal experience with CsCl after an acute dose of 40 g CsCl indicates that extended nausea and paresthesias around the mouth are the major side effects. The latter is probably due to K depletion. The usual doses used in the clinic ranges from 2 to 3 g given by mouth 3 times daily. At a latter time, at which time there is no indication of cancer presence, the CsCl dosage will be reduced to a preventative dose between 0.75 to 1.5 g a day. Less than 10 mg/kg or 0.6 g/60 kg of CsCl may enhance cancer growth.

The lymphoma case presented shows that CsCl efficiently reduced massive enlargements of spleen and liver as well as maximum ascites, causing an abdominal configuration of a tight, hard hemisphere to almost normalize after 3 months of therapy. This period of time was required to eliminate such a massive volume, resulting in the reduction of the body weight noted.
The clinical efficacy of CsCl high pH metabolic therapy is best demonstrated by a recent case of primary liver cancer (not included in the 50 cases reported in this study). The patient was a 39 year old female school teacher who was terminal. She was brought in the clinic on a stretcher on April 25, 1984 with a large liver tumor extending approximately 3 cm below the umbilical level with numerous large hard nodes ranging from 1.0 to over 5 cm (2/5 to > 2") in diameter. The treatment was then immediately instituted. This consisted of administration of CsCl, -carotene, vitamins A and C, Zn, Se, Mn, Cr and K salts by the oral route in addition to a concomitant massive I.V. doses of ascorbate, as well as K, Mg, Zn, Cu, Mn, and Cr salts, B-complex vitamins, folic acid, DMSO and heparin. After five consecutive treatment regimens, EDTA was introduced to the therapy and the minerals present in the IV solution were discontinued. On May 10, 1984, the patient was discharged, returned home, walking without assistance and displaying a pleasant smile on her face. The tumor-infiltrated liver had shrunk to 5 cm above the umbilicus or normal size and showed a smooth surface without any noticeable nodes. The determination of alphafetoprotein (AFP), a specific marker for liver cancers, and rare embryonic cancers and teratomas, decreased from an unusual high value of 39,000 units (120,000 µg/L), compared to normal levels of <13 units (<40 µg/L), seen before initiation of Cs-therapy, to 5,000 units (150,000 µg/L) obtained on the last day of treatment.

The mechanism of action of Cs in cancer has been little studied. That both Cs+ and Rb+ can specifically enter cancer cells and embryonic cells, but not normal adult cells, has been demonstrated by Brewer [2]. The cancer cells contain high amounts of hydrogen ions rendering them acidic and they also contain higher Na+ levels than found in normal cells. If Cs+ or Rb+ enter the cancer cells, their pH increases from as low as 5.5 to over pH 7.0. At a pH of 7.6 the cancer cell division will stop, and at a pH of 8.0 to 8.5 the life span of cancer cells is considerably shortened (to only hours). In one case, the author has observed the shrinkage of metastases of breast cancer one hour after Cs-therapy. Two days later wrinkles of the skin appeared where the tumor had been present. In another case of a colon cancer with massive metastasis, the massive infiltration of the abdominal wall, the liver and of other tissues seemed to have been reduced within 24 hours and was continuing rapidly until the demise of the patient on the 14th day of the Cs-therapy. The uric acid levels measured at the onset of treatment were approximately 3.5 mg/dL (normal: 2.0 to 8.0 mg/dL) which was increased to over 20 mg/dL suggesting massive breakdown of DNA, specifically, the purine nucleotides adenosine and guanosine which are catabolized to uric acid. Therefore, destruction of nuclear acids, as reflected by a significant rise in serum uric acid, may be used as predictive measurements for treatment outcome. The failure of serum uric acid elevation may be indicative of lack of destruction of cancer cells. This has proven to be a very consistent finding in our clinic. There are certain factors which may enhance the Cs-therapy. The Cs+ penetration into the cancer cell can be increased by the following three methods: The first approach resides in broadening the electron donor capacity of the cancer cell membrane by the application of cyanide (CN-), an electron donor radical found in nitriles (amgdalin, Laetrile® or mandelonitrile, prunasin, ficin, or cassivin), by selenium and zinc ions, which are electron donor radicals, or by the use of DMSO. The second approach enhances the potential gradient across the cancer cell membrane by the utilization of weak acids like ascorbic acid (Vitamin C) and retinoic acid (Vitamin A). The third method attempts to improve the circulation to the tumor and facilitate the destruction of cross linkages in the mucoid and fibrinous substances around the cancer cell. This can be achieved by chelation therapy, i.e., the use of EDTA, preferably CaNa2-EDTA directly I.V., as has been shown by Blumer [1], who reported on the reduction of cancer incidence by 90 % by chelating patients (an average of 15 chelations in 8 years). This approach also reduced cardiovascular disease by 50%. Other chelating agents can be also used. Moreover, the use of beta-carotene will tend to decomposition of blocking mucoid proteins which is mediated by electrical charges. Heparin which also acts through electrical charges will inactivate the immune repelling and immune binding capacities of the blocking mucoid proteins. These approaches will hinder cancer growth and they are virtually atoxic.

It should be noted that certain behavioral characteristics, "the cancer personality" of the cancer patient, may interfere in any projected treatment modality. This has been reported by Lawrence LeShan [4] in his book entitled "You Can Fight for Your Life." His studies suggested that cancer patients seeking treatment, e.g., chemotherapy, radiation or surgery are probably motivated by a covert desire for death. They fail to express statements such as, "rather than undergoing any of those treatments, I would rather die in peace," or "I would never undergo any of those treatments or let anyone of my family undergo them because the effectiveness is unproven, and the damage that is done with any of those treatments is higher than the effects", and willingly and in spite of the obvious failure of the treatments and the sometimes horrendous side effects of chemotherapy opt for repeated courses of it until their eventual demise. The most important psychological factors contributing to cancer are loss of a crucial relationship, both personal and professional, inability to express hostility or even show anger or aggression in self-defense, death of sibling or parent in early childhood causing tension, high degree of self-dislike or self-distrust, being programmed since early childhood that emotional relationships are dangerous and unsatisfying, being widowed, and, above all, being too controversial or not sufficiently proven to warrant inclusion. In this article. This slightly edited version restores the text of the original manuscript and on some places clarifies where all too terse language may have obscured the intended meaning.

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REFERENCES

APPENDIX I: COMMENTS ON OXYGENATION & OZONATION

EFFECTIVE CANCER TREATMENT & PREVENTION

The following methods are effective with virtually no adverse effects:
(a) Nutrition and lifestyle of longevity populations that consume foods rich in the nutrients that are the basis of the high pH therapy discussed in *Nature*. Also, low stress/high physical activity lifestyle is important in cancer prevention as is the ozone-rich air in the high altitudes in which they live. See "Cesura Therapy in Cancer Patients", supra, as well as the Chapter "Ozone and Cancer" in the author's book on "Ozone", and its forthcoming book "NO More Cancers".
(b) Herbs. Effective herbal antioxidants, as well as cancer treatments which were used worldwide since time immemorial including: *Cardiaceaea* spp. (cranberries, blueberries, etc.), *Astragalus membranacei* (Huáng Qí), *Fusarium gramineum* (gum), *Cordyceps militaris* (thymus), *Rheum palmatum* (root), etc., *PDR Cancer Prevention & Treatment 1000 to 4000 mg/day for a 60 kg patient. Except for a leukemias, and sarcomas such as chondro- and osteosarcomas. The numerous cancers and their metastases including cancers of the colon, lungs, prostate, breast, liver, kidney, brain tumors, lymphomas and leukemias, and sarcomas such as chondro- and osteosarcomas. The recommended dosage for prevention is 100 to 200 mg/day and for treatment 1000 to 4000 mg/day for a 60 kg patient. For a Herxheimer-type "healing crisis" reaction, no other adverse effects have been observed with this compound. If no effect is seen, the treatment should be discontinued after 60 days.

(d) Medical Ozone is the most powerful oxidative therapy, used in cancer treatment since the early 1920s, and where the DIRECT I.V. O₃ Application has the following advantages:
(1) EFFECTIVENESS of O₃ in the treatment of all cancers, including leukemia, lymphomas and sarcomas. Direct I.V. O₃ is particularly effective for lung cancers.
(2) Consistently better results compared with the LAHT. Direct I.V. application produces much faster results and requires fewer applications than LAHT. This is particularly apparent in patients with lung cancer & sarcoids, allergies, & AIDS.
(3) Prompt elimination of any allergic component contributing to cancer formation, e.g., by increasing the oxygen diffusion distance. This direct I.V. O₃ effect is much more consistent than with LAHT. The same applies to removal of viruses, bacteria, and fungi that may be adjunctive cancer factors. Direct I.V. O₃ consistently removes unwanted antibodies from the bloodstream, and seroconversions from HIV positive to HIV negative have been observed after only three to four consecutive administrations at the recommended dosage if patients are adequately supplemented, particularly with Zn, Se, vitamins A&E, & -carotene.

(c) Ozone is a powerful oxidizing agent. It is able to oxidize any substance with the highest electronegativity first. This is why ozone is a much more powerful oxidizing agent than other electron donors such as hydrogen peroxide and hydrogen sulfide. Ozone is a powerful electron donor. It is used to disinfect water, air, and food. In medicine, it is used to treat cancer patients. Ozone is a powerful inhibitor of cancer cell growth. It is effective against all types of cancer, including leukemia, lymphoma, and sarcoma.

(4) Ozone is a powerful oxidizing agent. It is able to oxidize any substance with the highest electronegativity first. This is why ozone is a much more powerful oxidizing agent than other electron donors such as hydrogen peroxide and hydrogen sulfide. Ozone is a powerful electron donor. It is used to disinfect water, air, and food. In medicine, it is used to treat cancer patients. Ozone is a powerful inhibitor of cancer cell growth. It is effective against all types of cancer, including leukemia, lymphoma, and sarcoma.

(5) Precise dosage since there is no reaction with the vitamin C that most German researchers suggest be added to the large autohemotransfusion (LAHT).

(6) Homeopathic effect of direct I.V. ozone: The direct I.V. application of ozone (contrasted to the LAHT) in a dosage of at least 0.1 mg/kg has a unique homeopathic effect that is not observed (and/or has never been described) with LAHT. When ozone is injected into the bloodstream, it immediately starts to react with any available type of oxidizable substrate, most notably, the lipids of the cell membrane. Lipid peroxidation products of ozone include alcohols and peroxyl radicals, polyunsaturated fatty acid (PUFA) hydroperoxides, PUFA alcohols and peroxyl radicals, single oxygen, hydrogen peroxide ("peroxide burst"), the mechanism of killing viruses, bacteria and fungi of macrophages, microphages, and blood platelets, ozonides, and alkoxyl radicals. In all the different reactions products are taken into consideration, there may be perhaps 10,000 or more different oxidized species in minute ("homeopathic") amounts, formed under conditions of considerable turbulence (simulating homeopathic "succussion", or the vigorous shaking used in classic homeopathy to "potentize" the effect of the remedy). This brings about a "healing crisis" that simulates milder versions of any hidden but still significant disease processes, that have not been completely resolved, including allergies, old drug toxicities, environmental pollution, old viral, bacterial or fungal infections, even physical traumas, migraines, and other conditions. After the "healing crisis" is brought about by the ozone administration, these conditions are resolved (analogous to the mechanism of action of homeopathic remedies) and people reach a new level of wellbeing that was unattainable before. Particularly impressive is this effect in all chronic allergies, asthma, hay fever, "brain allergies", and in chronic fatigue syndrome, as well as in all types of substance abuse & addiction including nicotine, alcohol, drugs, cosmetics, etc.

(7) Direct IV Ozone oxidatively detoxifies any drugs or other toxic substances that have invariably accumulated in the bodies of patients with degenerative disease, especially cancer. This minimizes long-term detrimental effects and prevents late toxicity such as denervation failure and death from doxorubicin or daunorubicin, permanent sterility from alkylating agents such as cyclophosphamide, melphalan, ifosfamide, etc., pulmonary fibrosis seen after virtually all antineoplastic drugs, as well as carcinogenic environmental toxins such as dioxins, parathion, polycyclic aromatic hydrocarbons, & other (non-)halogenated aliphatic/aromatic/polycyclic hydrocarbons found in the air, water, or food taken in.

(8) Hyperthermia effect of direct IV ozone is observed in most patients, especially the ones with extensive tumors and/or significant (concomitant) infections. The effectiveness of hyperthermia or fever in reversing malignancies was known since antiquity. Note that externally induced hyperthermia includes immersion of the body in the hot healing waters of balneological cancer resorts, sitting in a sauna [especially, an infrared sauna] or steam bath, used since antiquity against cancers [-modern ways] and [Aguasizer baths with ultrapure ozonated water], or simply wrapping oneself in [space] blankets with a hot water bottle. The total body hyperthermia combined with potassium and glucose I.V. was developed in the 1940s by the late Professor Dr. med. Manfred von Ardenne in Dresden, Germany as his very successful low-pH therapy of cancer. This therapy was then superseded by the LSU (now ULS) ENHANCED HIGH-pH THERAPY OF CANCER since the mid-1970s.
APPENDIX II: SUMMARY OF ADVERSE EFFECTS OF CONVENTIONAL CANCER THERAPY

The "State of the Art" of Conventional Cancer Therapy

As stated previously in "Cesium and Cancer", (conventional) [treatment modalities of cancer include surgery, radiation and chemotherapy. In those cases where surgery was successful the body's own immune system was able to counteract the reduced amount of tumor tissue. Surgery is likely to produce metastasis without stimulation of the immune system. Radiation may cause damages to the organism, e.g., carcinogenicity of x-rays and other radiations, and their depression of the immune system is well known. Prolonged chemotherapy may cause severe to lethal side effect in some instances, and the use of hormones and interferon are useful only for very few cases of cancer. Conventional treatments of cancer have produced 5-year survival rates which do not reflect the inexorable gradual deterioration of the cancer patients, nor their suffering from various treatment-related adverse reactions, and where chemotherapy of, e.g., breast cancer may shorten the life by 18 to 36 months v. no treatments at all.'

Adverse Effects of Surgery may arise as anesthetic "accidents" and deaths, complications during surgery, & prolonged "aftereffects".

(1) Anesthesia: Over one half of all deaths attributed to anesthesia were caused by excessive blood loss during surgery; the remainder was from aspiration, overdose/misuse of anesthetics, & faulty technique. Brain damage from anesthesia was attributed in about 50% to faulty technique. Other longterm effects of anesthesia include lasting immunodepression.

(2) Surgery: Besides excessive blood loss and tissue trauma that is often unavoidable even for the most skilled surgeon, cancer surgery, in virtually all cases leads to dissemination of cancer cells and immunodepression. If there is enough immune function, the body may be able to recognize and eliminate the spread cancer cells. Failure of tumor recognition, most prevalent in blood type A & AB, may lead to extensive metastasis.

(3) Longterm Aftereffects: Lung surgery & massive abdominal resections may leave patients so crippled that they lose their will to live. The same applies to many patients with colostomies, paralysis, or brain damage, and children who suffer an amputation of a limb or other severe mutilations.

Few surgeons are aware of the fact that there is a clinically significant immunodepression after almost any, but especially after (ultra)radical cancer surgery [less prevalent in the U.S. since about 2000](while radiologists are much more aware of the similar one after radiation), and which is most pronounced in deficient tumor response to other antigens, cellular immunodeiciency with recurrent infections that, if severe, may include opportunistic pathogens, and phagocytic dysfunction (from operative stress) that interferes with removal of (pre)cancerous cells and contributes to bacterial/fungal infections.

Adverse Effects from Radiation include acute radiation syndrome, the hemopoietic, gastrointestinal, & CNS syndromes, & pneumonitis & pericarditis based on the location of the radiation, and late effects.

(1) Acute Radiation Syndrome is characterized by nausea, vomiting, fatigue, loss of appetite & general malaise that may last for 48 to 72 hours [less prevalent in the U.S., as is (4), since about 2000].

(2) Chest irradiation may cause granulocytopenia & thrombopenia from bone marrow depression, as well as radiation pneumonitis (insidious onset, cough, dyspnea, cyanosis, & fever 6-12 weeks after radiotherapy) & pericarditis.

(3) Abdominal irradiation may cause profuse (& later chronic) diarrhea (unresponsive to conventional therapy), nausea, vomiting, lymphocyte depression, severe gastrointestinal upset, after a latency of 1 to 2 weeks bloody diarrhea & hematuria, and fistulas as late complications.

(4) Cranial irradiation may cause after immediate nausea & vomiting, hypotension, apathy, ataxia, convulsions & coma, & delayed necrosis of the brain manifesting as headaches & a variety of neurological symptoms.

(5) Late Effects: Inductions of secondary cancers of almost all types, as well as of leukemias & lymphomas, and where the female breast and the thyroid appear to be especially radiosensitive, and immunodepression worse than after surgery [see supra], especially, in addition, chronic depletion of lymphocytes, pancytopenia (especially if the bone marrow had been irradiated), & severe phagocytic dysfunction.

Adverse Effects from Chemotherapy of the about 75 presently used "anti-cancer" drugs, NONE has been proven effective, singly or in combinations, in properly controlled, let alone, double blind, clinical trials. On the contrary, since about 1975 to the present [2004], careful analysis of the results of cancer chemotherapy by Arlin J. Brown proved that these anti-cancer drugs are the MAJOR CAUSE of DEATH of cancer patients. Adverse effects of these drugs not only caused a severe life quality deterioration but also, in most cases, actually SHORTENED the life expectancy as compared to matched untreated controls. The following gives some ideas about acute & delayed toxicity of these drugs, & where dose-limiting toxicity is listed in bold. Note that 35 pages, mostly tables, in "Ellenhorn's Medical Toxicology", 2d ed., Williams & Wilkins, Baltimore, 1997, are devoted to "Cancer Chemotherapeutic Agents/Cytotoxic Drugs".

(a) Acute Toxicity:

Nausea & vomiting is found with almost all, except, perhaps, with aminoglutethimide, cladribine, GaNO, gemcitabine, goserelin, the interferons, leuprolide, paclitaxel, tretinoin, & vincristine, & which is dose-limiting, e.g., with mechlorethamine (nitrogen mustard).

Anaphylaxis & other allergic reactions (A&A) may be encountered with many oft these drugs, e.g., anisocrine, asparaginase & pegaspargase, bleomycin, cisplatin, cytarabine (& acute respiratory distress), dacarbazine, dactinomycin, daunorubicin & doxorubicin, etoposide, fluorouracil (5-FU), methotrexate, paclitaxel, etc.

Aldesleukin (IL-2): in addition (+), causes fever; fluid retention; hypotension; respiratory distress; rash, anemia, thrombopenia; diarrhea; erythema nodosum; neutrophilic chemotactic defects, etc.

Daunorubicin & doxorubicin: (+) extravasation necrosis (ENV); acute cardiac toxicity (hours to days after) with EKG-changes including supraventricular arrhythmias, heart block, & ventricular tachycardia, major drop in ejection fraction, congestive heart failure (CHF), pericardial effusions (myocarditis-pericarditis syndrome), GI toxicity, etc.

Iofosamide: (+), cardiac toxicity: nephrotoxicity; confusion; metabolic acidosis & renal (de Toni-Fanconi syndrome (proximal renal tubule dysfunction with hyperaminoaciduria, glycosuria, hyperphosphaturia, bicitarone & water loss), etc.

Methotrexate: (+) hepatic necrosis; fever; diarrhea, etc.

Paclitaxel: A&A, dyspnea, hypotension, angioedema, urticaria, etc.

Asparaginase & pegaspargase: A&A, hypersensitivity; fever, chills; headache: abdominal pain; hyperglycemia leading to coma, etc.

Tretinoin: headache; xerosis: "retinoic acid syndrome" (i.e., fever, dyspnea, pulmonary infiltrates, pleural effusions, peripheral edema, hypotension); arthralgias, myalgias, etc.

(b) Delayed Toxicity: Almost all nonhormonal anticancer drugs show dose-limiting (1) bone marrow depression or (2) delayed leukopenia & thrombopenia (in the latter, especially in carbimustine, chlorozotocin, lomustine, et al.), as well as cutaneous reactions (sometimes severe), hyperpigmentation, & ocular toxicity, alopecia (which causes significant patient distress especially in women), & PERMANENT sterility after alkylating agents, e.g., cyclophosphamide, ifosfamide, & melphalan.

Other dose-limiting toxicities include:

(3) Pneumonitis & pulmonary fibrosis: bleomycin, busulfan, methotrexate, carmustine & lomustine, etc.

(4) Renal damage: cisplatin, streptozocin, etc.

(5) Stomatitis & oral ulcerations: dactinomycin, etc.; both oral & GI ulcerations: fluorouracil, fluorouracil (5-FU), methotrexate, etc.

(6) Cardiotoxicity (cumulative, dose-dependent cardiomyopathy/CHF; may be delayed for years): daunoo- & doxorubicin, etc.

(7) Hypophosphatemia: GaNO,

(8) Hemorrhagic cystitis: ifosfamide, less in cyclophosphamide, etc.

(9) Teratogenicity; chelitis: isotretinoin" & "tretinoin" & & where "2" cause rashes; pseudotumor cerebri; hypertiglyceridemia, & 1, (+), causes anorexia; xerostomia, xerophthalmia & conjunctivitis; bone & joint pain; & "5", (+), causes thrombophlebitis; & leukocytosis; whereas acute toxicity of vit. A (retinol & retinyl esters) can be almost completely avoided (up to 3 MU/day x 30 days) with concomitant vit. E (e.g., mixed tocopherols).

(10) CNS depression: mitotane

(11) Hemorrhagic diathesis: plicamycin

(12) Peripheral neuropathy: vincristine

(13) Mucositis: dauno- & doxorubicin, trimetrexate
APPENDIX III: DIETARY CARCINOGENS

A. NATURALLY OCCURRING DIETARY CARCINOGENS
Most naturally occurring dietary carcinogens are either "natural pesticides" (protecting plants against fungi, insects, & animal predators) or mycotoxins (metabolites produced by molds in food).

(a) Natural Pesticides have been found carcinogenic if given in high doses to animals though there is no evidence of carcinogenicity in humans; allyl isothiocyanate, in cabbage, cauliflower, Brussels sprouts, mustard, & horseradish is, in fact, clearly anti-carcinogenic. There is no evidence for human carcinogenicity of caffeic acid occurs in apples, pears, cherries, carrots, celery, lettuce, potatoes, endive, grapes, eggplant, thyme, basil, dill, caraway, rosemary, tarragon, & coffee beans; of safrole in nutmeg, mace, pepper, cinnamon, natural root beer; estragole in basil, fennel, & tarragon; carvacrol in marjoram; furocoumarins in lime, citrus oils, carrots, celery, parsley, & parsnips; nor of pyrrolizidines in comfrey tea. The hepatic carcinogenicity of hydrazine, in mushrooms is linked to the hypo-methylation of organ DNA that occurs only in folic acid deficiency. Note though that hydrazine overdose (if used as "anticancer agent") leads to ataxia, lateral nystagmus, loss of vibration sense, & seizures, & it is also hepatotoxic.

(b) Mycotoxins proven highly carcinogenic in humans are aflatoxin B1 (AFB1) & natural mixtures of aflatoxins (B1, B2, G1, M1, M2, etc., & which are also mutagenic, teratogenic, & immunodepressive), as well as sterigmatocystin, while griseofulvin (antifungal) is moderately CAgenic. Fumonisins and ochratoxin A are possible human carcinogens. Citrinin, patulin, penicillrinic acid, & zearealenon are carcinogenic for animals.

(1) Aflatoxins, produced by Aspergillus parasiticus & A. flavus, occur in peanuts, maize, cotton seed, rye, barley, food grains, etc. Fowl & other farm animals fed infected ground nut meal may die of aflatoxicosis. Aflatoxins cause liver & gallbladder cancers, & may increase lung cancer incidence via the AFB1-2,3-epoxide that forms the AFB1-N7-Gua adduct & may contribute to the suppression of cell-mediated immunity (i.e., resulting from activation of T- lymphocytes) seen in AIDS patients.

(2) Sterigmatocystin, from V. versicolor/Flavus/Ruber/Luteum in wheat, peanuts, and rice, is highly carcinogenic in animals.

(3) Fumonisins (esp. B1), in maize, are carcinogenic in rodents (liver & kidney cancers) & have been linked to esophageal cancer in humans.

(4) Ochratoxin, from A. ochraceus, P. veridatum/cyclopium in wheat, oats, rice, & green coffee beans, has been implicated in renal adenomas & Balkan nephropathy.

(5) Zearealenone, from Fusarium spp. in maize, wheat, sorghum, & feed grains, has been implicated in cervical cancer & premature thelarche.

(6) T-2 toxin, in barley, maize, safflower seeds, & cereals, may contribute to pellagra (primarily caused by failure to convert tryptophan to niacin or niacin deficiency & characterized by dermatitis, mucositis, diarrhea, & psychic disturbances including depression, irritability, anxiety, confusion, disorientation, delusions, & hallucinations).

B. PRODUCTS OF FOOD PREPARATION & PROCESSING
Food preparation & preservation are major sources of dietary carcinogens, including heterocyclic aromatic amines (HAAs), formed during frying, broiling, & grilling of high-protein foods and more prevalent in well-done meats; polycyclic aromatic hydrocarbons (PAHs), formed during broiling & smoking food, particularly ground beef and steaks, less in pork & chicken; & N-nitroso compounds (NOCs), formed in smoked, salted, & pickled foods cured with nitrate or nitrite. Note that NOCs are also formed in the stomach from nitrates & amines in the diet. Acrolein, a decomposition product of glycerin from overheating fats, has been discussed in "Nutrients in Cancer", supra [p. 9].

(a) Heterocyclic Aromatic Amines (HAAs) are potent mutagens & animal carcinogens causing cancers of the liver, colon, mammary gland, skin, prostate, lymphoid tissue, oral cavity, lung, etc. Of 20-some HAAs, 4 are possible human carcinogens, including 2 amino 3 methylimidazo[4,5-f] quinoline (IQ); 2 amino 3,4 dimethylimidazo[4,5-f]quinoxaline (MeIQ); 2-amino-3,8-dimethylimidazo[4,5-f] quinoxaline (8-MeIQx); and 2 amino 1 methyl 6 phenylimidazo[4,5-b] pyridine (PhIP). HAA-DNA form major adducts with C8, & IQ & MeIQx form minor adducts at N2 of guanine, and where the metabolic activation appears to occur via N-oxidation & O-acetylation to form N-acetoxy arylamines that bind to DNA, & where rapid oxidizers & acetylators may be at greater cancer risk.

(b) Polycyclic Aromatic Hydrocarbons (PAHs), particularly benzo[a]-pyrene, less benzo[a]anthracene, dibenzo [a,h]pyrene, and chrysene, are animal carcinogens. There seems to be a correlation between PAH-DNA adducts & ras oncogene mutations & that the hGSTP1 gene is an important factor in susceptibility to PAH-related cancers.

(c) N-Nitroso Compounds (NOCs) are carcinogenic in animals, including primates, after oral ingestion. They may be a significant risk factor for human cancers of the stomach, esophagus, colon & rectum, nasopharynx, urinary tract, & liver. Note that ascorbic acid, tocopherols, retinoids,phenolic & sulphydryl compounds, orange peel, & other nutrients & specific foods mentioned in "Nutrients and Cancer", supra, may inhibit the formation of endogenous NOCs.


(a) Direct/Intentional Synthetic Additives include i.e., antioxidants, colorants, flavor ingredients, artificial sweeteners, solvents, & humectants.

(b) Indirect/Unintentional Synthetic Additives include pesticides, solvents, and packaging-derived chemicals. These chemicals, which number more than 2000, enter the food supply during production, processing, packaging, and storage from a variety of sources. Of these, PESTICIDES are of major concern since most, if not all, pesticides have human toxicity potential, including carcinogenicity. Of these, arsenicals, benzal chloride, & fluoride insecticides are highly carcinogenic, while amitrole, benzonitrilechloride, carbon tetrachloride, chlorophenols, p-di-chlorobenzene, ethylene dibromide & thiourea, formaldehyde, phenoxy acids, 2,3,7,8-tetrachlorodibenzo-p-dioxin, & 2,4,6-trichlorophenol are moderately CAgenic in humans, and where, because of bioaccumulation, the cancer & other longterm health risks from pesticides increase with repeated exposure over many years.[50 pages in "Ellenhorn's Medical Toxicology",2d ed., 1997, deal with "Pesticides"]. Note that the most effective insecticide, DDT (p,p'-dichlorodiphenyldichloroethane, now: chlorophenate), is anticarcinogenic & (properly used) virtually non-toxic to humans. Its ban in 1972, since it almost eradicated Anopheles spp., caused excess deaths from malaria alone of >90 million people by 2004. Of packaging materials, vinyl chloride, is a human carcinogen, and several phthalate ester plasticizers (especially DEHP & its metabolite MEHP), are potential carcinogens. Note that extensive use of DEHP-plasticized PVC blood tubing caused necrotizing dermatitis & plasticizer-induced hepatitis, & testicular atrophy with sterility in dialysis patients, & inhalation of DEHP & MEHP causes bronchial asthma; NBBS (in plastic resins & fungicides) may pose risk of anytrophic lateral sclerosis.

(c) Chemical Contamination of Water with carcinogens, principally, trihalomethanes (CHCl3, CHCl2Br, etc.), which are disinfection by-products in public water supplies which do not occur with ozonation, industrial solvents trichloroethylene & -ethylene, tetra- & 1,2 dichloro-ethlene, as well as arsenic & asbestos, and pesticides in wellwater are major public health concerns. Fluoride is a carcinogen, is THE aging factor, >1 ppm in drinking water increases risk of osteoporotic hip fracture, & ingestion of >10 mg/kg of F (in toothpaste) may be lethal.
APPENDIX IV: LIST OF CESIUM & RUBIDIUM ARTICLES


1: Brewer AK.
The high pH therapy for cancer tests on mice and humans.
Pharmacol Biochem Behav. 1984; 21 Suppl 1: 1-5.

2: Sartori HE.
Nutrients and cancer: an introduction to cesium therapy.

3: Sartori HE.
Cesium therapy in cancer patients.

4: Neulieb R.
Effect of oral intake of cesium chloride: a single case report.

5: Pinsky C, Bose R.
Pharmacological and toxicological investigations of cesium.

6: Tuft MJ, Tuft FW, Brewer AK.
The response of colon carcinoma in mice to cesium, zinc and vitamin A.

7: Messiha FS.
Biochemical aspects of cesium administration in tumor-bearing mice.

8: Messiha FS, Stocco DM.
Effect of cesium and potassium salts on survival of rats bearing Novikoff hepatoma.

9: Messiha FS.
Effect of cesium and ethanol on tumor bearing rats.
Pharmacol Biochem Behav. 1984; 21 Suppl 1: 35-40

10: Yung CY.
A synopsis on metals in medicine and psychiatry.

11: Malek-Ahmadi P, Williams JA.
Rubidium in psychiatry: research implications.

12: Yung CY.
A review of clinical trials of lithium in medicine.

13: Yung CY.
A review of clinical trials of lithium in neurology.
Pharmacol Biochem Behav. 1984; 21 Suppl 1: 57-64. Review.

14: Paragas MG.
Lithium adverse reactions in psychiatric patients.

15: Yung CY.
Neuropsychiatric manifestations of alkali metal deficiency and excess.
Pharmacol Biochem Behav. 1984; 21 Suppl 1: 71-5. Review.

16: Pannell KH, La Neave CK, Rico E, Arkles B.
Concerning the relative non-toxicity of silicicron ionophores.
Pharmacol Biochem Behav. 1984; 21 Suppl 1: 77-80.

17: Roberts LA.
Chronotropic effect of alkali metals on spontaneously beating right atria.

18: Messiha FS.
Lithium, rubidium and cesium: cerebral pharmacokinetics and alcohol interactions.

19: Messiha FS, McGrath J.
Modulation of nitrogen dioxide toxicity by lithium.

20: McGrath JJ.
The effects of carbon monoxide on the heart: an in vitro study.

21: McGrath JJ, Leviseur C.
Cardiorespiratory responses to intestinal injection of carbon monoxide.

22: Baudhuin MG, Jefferson JW, Greist JH.
The Lithium Information Center: an efficient information service.

23: Messiha FS.
Cesium: a bibliography update.